THE PREVALENCE OF PERIODONTITIS IN OBESE ADULTS IN THE KINGDOM OF BAHRAIN

Leena Abdulla Al Salihi
BDS, King Saud University Riyadh, 1993
MGDS, Royal College of Surgeons Edinburgh, 2009

Presented to the Hamdan Bin Mohammed College of Dental Medicine of
The Mohammed Bin Rashid University of Medicine and Health Sciences
in Partial Fulfilment of the Requirements for the Degree of
Master of Science in Periodontology
2018

ABSTRACT

The Prevalence of Periodontitis in obese adults in the Kingdom of Bahrain

Supervisors: Professor Crawford Bain and Professor Alexander Milosevic

Background: Adult obesity in Bahrain is an increasing problem and evidence suggests obesity could be a novel risk factor for periodontitis. This study aimed to assess prevalence of periodontitis in overweight/obese adults attending Ministry of Health (MOH) clinics in Bahrain and to determine which measure of obesity, body mass index (BMI) or waist circumference (WC), is associated with periodontitis.

Materials and Methods: This was a cross-sectional study of a convenience sample of overweight subjects attending MOH Nutrition Clinics at primary health centers. Ethical approval and patient consent were obtained prior to the study. A range of demographic and anthropometric data, including BMI and WC using WHO thresholds for severity of obesity, were recorded. Dental assessments of periodontal status were based on CPI probing depths at six sites per tooth and the extent of periodontal disease was categorized according to the number of sextants with CPI codes 3 and 4.

Results: A total of 372 participated with a mean age 44.0 (±10.5) years for males, and 42.5 (±11.2) years for females. Periodontitis was present in 361 (97%) of participants. Hypertension and diabetes were the most prevalent co-morbidities at 23.4% and 16% respectively. Mean WC was significantly greater in males at 114cm (±15.6) compared to females 109.5cm (±12.5) ($p<0.001$). BMI was not associated with severity or extent of periodontitis but WC was weakly correlated in males but not in females (Spearman rho=+0.2, $p<0.05$). In the logistic regression model using

overall WC to predict the severity of periodontitis, the adjusted OR was 1.02 (95%CI 1.00-1.04) and for age it was 1.05 (95%CI 1.00-1.07).

Conclusion: The prevalence of periodontitis was high in this sample of overweight Bahrainis. BMI was not correlated with periodontitis but WC had a weak positive correlation. Implementation of a periodontal health screening as a routine part of a Nutrition Clinic program is required as a preventive approach.

DEDICATION

This thesis is dedicated to my beloved Husband Dr. Khaled AlJasem

For being a wonderful source of support and encouragement, and being patient in allowing me to pursue my ambition to be a Periodontist

DECLARATION

I, **Leena Abdulla Al Salihi**, declare that this dissertation is my own original work, and that it has not been presented and will not be presented to any other University for a similar or any other degree award.

Signature…… Date…….

ACKNOWLEDGMENTS

As well as my own efforts, the success of this study depended largely on the encouragement of many others. I would like to take this opportunity to express my gratitude to those people who have contributed to the successful completion of this study.

I would like to express the deepest appreciation to my thesis supervisor, Professor Crawford Bain. I express my heartfelt gratitude, in the most special way, for being my 'father' in the university. His continuous guidance and support persisted even throughout his recent illness. In Professor Bain, I believe that I have learned from the best periodontist in the world.

I am highly grateful to Professor Alexander Milosevic for taking over the responsibility to continue my supervision at such short notice. I am particularly grateful that he could share his valuable time and experience to provide advice, suggestions and help to allow me to complete my thesis on time.

Special thanks to Dr. Amar Hassan, Associate Professor of Biostatistics for his never-ending support, including his valued statistical data handling and analysis in this thesis.

I am also grateful to Dr. Abeer Janahi for her keen interest and co-operation in collecting the data.

I would like to thank the Directorate of Training in the Ministry of Health in Bahrain for their sponsorship that provided financial support for my Master's degree.

Particular thanks and gratitude must be recorded to Nutrition section in the Ministry of Health in Bahrain. I am indebted to all of the dietician specialists who helped in organising my visits to the nutrition clinics and made the data collection process so easy.

The highest appreciation goes to my parents and sisters for their continuous prayers and encouragement; to my beloved children, who served as my inspiration, allowing me to pursue this undertaking; to my colleagues, Asmaa, Sireen and Taha for their moral support and bonding that I will treasure forever; to my tutors Dr. Maanas Shah, Dr. Haifa Hannawi, Dr. Geoffrey Sharpe, Dr. Adrian Salpanha and Dr. Mehnaz Kamali for sharing their knowledge and clinical experience with us; to our dental assistants Nazeene, Girlie and Harmond and to the Dubai Dental Clinic Director and staff who were available when I needed them.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

BMI: Body Mass Index

BOP: Bleeding On Probing

CAL: Clinical Attachment Loss

CP: Chronic Periodontitis

CPI: Community Periodontal Index

CPITN: Community Periodontal Index of Treatment Needs

CRP: C-Reactive Proteins

CVD: Cardiovascular Disease

DB: DistoBuccal

DL: DistoLingual

E-Probe: Epidemiological probe

FBS: Fasting Blood Sugar

GCC: Gulf Cooperation Council

Hb: Hemoglobin

HBMCDM: Hamdan Bin Mohammed College of Dental Medicine

HDL: High Density Lipoprotein

IL: Interleukin

LA: Loss of Attachment

LDL: Low Density Lipoprotein

MB: MesioBuccal

MBRU: Mohammed Bin Rashid University

Mid B: MidBuccal

Mid L: MidLingual

ML: MesioLingual

MOH: Ministry of Health

NCD: Non-Communicable Diseases

NHANES III: National Health and Nutrition Survey

OR: Odd Ratio

PPD: Periodontal Probing Depth

RR: Relative Ratio

S. Arabia: Saudi Arabia

S. Creatinine: Serum Creatinine

T. Forsythia: Tannerella Forsythia

TNF-α: Tumor Necrosis Factor Alpha

UAE: United Arab Emirates

UK: United Kingdom

USA: United State of America

VFA: Visceral Fat Area

WC: Waist Circumference

WHO: World Health Organization

WHR: Waist Hip Ratio

CHAPTER ONE

1. Introduction

Obesity is defined as abnormal or excessive fat accumulation that may impair health. Globally the fundamental cause of obesity and overweight is an energy imbalance between calories consumed and calories expended. The worldwide prevalence of obesity nearly tripled between 1975 and 2016. Overall, about 13% of the world's adult population (11% of men and 15% of women) were obese in 2016 [(1)].

Unlike other major global health risks such as tobacco, smoking and childhood malnutrition, obesity is not decreasing worldwide. Rates of obesity seem to be increasing in both developed and developing countries where almost two in three of the world's obese people live. Obesity is already a major public health challenge in many middle-income countries [(2)].

WHO in 2017 [(1)] reported the adverse effects of obesity on chronic health outcomes, such as diabetes, cardiovascular diseases (mainly heart disease and stroke), which were the leading cause of death in 2012, musculoskeletal disorders (especially osteoarthritis - a highly disabling degenerative disease of the joints) and some cancers (endometrial, breast, and colon).

Among the most common oral infections worldwide [(3)], periodontitis is a destructive disease of the gingiva and of the supporting structures of the teeth, which develops through inflammatory processes induced by a microbial biofilm [(4)].

Extensive epidemiology and experimental evidence exists for the role of several risk factors in the initiation, progression, and severity of periodontal diseases. Obesity is specifically, one systemic risk factors for susceptibility to periodontal disease that is relatively common in the population and likely to have a substantial population-attributable risk [(5)]. Furthermore, Suvan et al. in 2011

suggested, by several lines of evidence that obesity could be a novel risk factor for periodontitis [6].

Many systematic reviews and meta-analysis which explore the association between obesity/ overweight and periodontitis have corroborated the positive association between those conditions [6],[7] , although they have largely been based upon cross-sectional epidemiological data collection. Suvan et al. in 2015, in a case -control study, ascertained that the odds of having periodontitis are more likely if an individual is overweight or obese compared to normal weight individuals [8]. Moreover, the most recent systematic review and meta- analysis of prospective longitudinal studies by Nascimento et al. in 2015, demonstrated a relationship between weight gain and incidence of periodontal disease [9].

Several mechanisms were proposed by which obesity may lead to an increase in periodontal diseases. Obesity is considered as a chronic low-grade inflammatory disease because of changes in adipose tissue [10]. Adipocytes secrete dozens of biologically active molecules, these include leptin, resistin, tumor necrosis factor α (TNF-α), interleukins:IL-1, IL-6, IL-8 and IL-10, growth factors, complement components, angiotensinogen, plasma plasminogen, activator-1(PAI-1), and a number of other substances. Therefore, obesity may alter the response of the host to antigens derived from bacterial plaque, and thus cause disturbances in the inflammatory response in the course of the periodontal disease [10],[11],[12],[13],[14],[15] .Periodontitis itself is an inflammatory disease, which induces its own set of cytokines both locally and systemically in response to bacterial pathogens and endotoxins and thus adds to the levels of circulating pro-inflammatory mediators [16]. However, if an association between obesity and periodontitis exists then that association most likely lies in the commonality of their inflammatory pathway [17].

The worldwide prevalence of obesity nearly tripled between 1975 and 2016 [1]. Available statistics indicate that an alarming proportion of people in most Arab countries suffer from obesity [18] . However, there is a scarcity of studies on obesity as a risk factor for periodontitis in the Arab population particularly in the Gulf region. Only three published studies investigated the association between obesity and periodontal disease in the Arab population; Khader et al. in 2008 in Jordan [19] El-Sayed Amin in 2010 in Egypt [20], and Awad et al. in 2015 in UAE [21]. The majority of available studies reported the relationship of obesity with periodontitis in subjects from European, American, Japanese and Korean population [8],[22],[23],[24],[25],[26],[27],[28],[29],[30],[31],[32],[33],[34] .

Furthermore, studies have revealed that the prevalence of overweight and obesity among adults in Eastern Mediterranean region ranged from 50 to 80%, with higher prevalence among women than men [35]. The analysis of the latest national data from the Middle East revealed worryingly high obesity rates in many countries. In particular, the results for Kuwait, Oman, Saudi Arabia, Lebanon, Turkey, Bahrain and Jordan showed that the problem of obesity is growing for men and women of all ages in each country [36]. A recently published national nutrition survey from an adult sample of 2037 found that the overall prevalence of obesity was 36.3% and was higher in females than males (40.3% and 32.3%, respectively) [37].

BMI has traditionally been the chosen indicator by which to measure body size and composition, and to diagnose underweight and overweight. The majority of studies used BMI as an indicator for obesity, however, alternative measures that reflect abdominal adiposity, such as waist circumference, waist–hip ratio and waist–height ratio, have been suggested as being superior to BMI in predicting cardiovascular disease risk (CVD). This is based largely on the rationale that increased visceral adipose tissue is associated with a range of metabolic abnormalities, including

decreased glucose tolerance, reduced insulin sensitivity and adverse lipid profiles, which are risk factors for type 2 diabetes and CVD [38].

Prevalence of adult obesity in Bahrain is increasing at an alarming rate, with associated morbidity and mortality. Most adults in primary care are overweight or obese; two thirds of patients with weight problems have other obesity-related conditions [39]. Therefore, Nutrition Clinics for the treatment of obesity in adults, 19 years and above, were established and funded by the Nutrition Section, Ministry of Health, Bahrain in 5 main health centers since 2009, to combine the use of mainly dietary and physical activity in a mid to long term intervention program. The program was administered by a team of nutritionists, nurses and family physicians [40].

The aim of this study was to investigate the prevalence of periodontitis in obese adults in the Kingdom of Bahrain, and assess which measure of obesity (BMI, WC) is a better predictor of a periodontitis.

1.1 Literature review

1.1.1. Review of the literature on periodontal disease

1.1.1.1 Definition/ Etiology of periodontal disease

Among the most common oral infection worldwide [(3)], periodontitis is a destructive disease of the gingiva and of the supporting structures of the teeth, which develops through inflammatory processes induced by a microbial biofilm [(4)].

Gingivitis refers to the inflammation of gingiva caused by bacteria accumulating along the gingival margin. Periodontitis can be developed from a pre-existing gingivitis. However, not every case of gingivitis develops into a more advanced periodontal disease [(41)].

Over the years various plaque hypotheses have been formulated for the role of bacteria in initiating periodontitis. Starting from 1870 when Koch postulated that bacteria isolate from diseased tissue, must cause the disease in animals. In 1977 Socransky modified Koch's postulates for periodontitis, and stated that putative pathogen must be found in a large number in diseased sites and must be able to demonstrate immune response to putative pathogens, elimination of these organism should result in clinical improvement [(42)]. Three phases of plaque hypotheses can be broadly identified; the non -specific theory in microbial etiology of periodontal disease that was discussed by Theilade in 1966; in the classical version of the non-specific theory; periodontal disease develops due to bacterial proliferation exceeding the threshold of host resistance and all dental plaque microorganism are actually capable of causing periodontal pathology if present in high numbers [(43)]. Then Loesche in 1980 discussed the specific plaque hypothesis; a single pathogenic species causes periodontal disease, periodontal disease related more to quality of plaque composition rather than merely plaque quantity [(44)]. This was followed by the ecological plaque hypothesis which was introduced by Slots in 1986; the modern version of specific theory, where the single

pathogen is dropped in favor of disease initiated by a number of pathogens. So certain dental plaque microorganisms are preferably selected and increase as a result of changes in local environment which adversely alters normal protective microbial homeostatic mechanisms [45].

The current concept suggests that pathogens are necessary, but not sufficient for disease activity to occur. Factors which influence activity include susceptibility of the individual host and the presence of interacting bacterial species which facilitate or impede disease progression. Recent studies have attempted to distinguish virulent and a virulent clonai types of suspected pathogenic species and seek transmission of genetic elements needed for pathogenic species to cause disease. In addition, the local environment of the periodontal pocket may be important in the regulation of expression of virulence factors by pathogenic species. Thus, in order that disease results from a pathogen, 1) it must be a virulent clonal type; 2) it must possess the chromosomal and extra-chromosomal genetic factors to initiate disease; 3) the host must be susceptible to this pathogen; 4) the pathogen must be in numbers sufficient to exceed the threshold for that host; 5) it must be located at the right place; 6) other bacterial species must foster, or at least not inhibit, the process; and 7) the local environment must be one which is conducive to the expression of the species' virulence properties [46].

There are different clinical manifestations of periodontal disease, and it may be acute or chronic. Clinical signs of the disease include deepening of periodontal pockets and loss of attachment, progressively leading to loosening of teeth and ultimately to tooth loss. Periodontal destruction may be caused by local factors, such as dental biofilm, or it may reflect an inadequate immune response. Gingivitis and periodontitis can also be manifestations of certain systemic conditions or diseases, such as mucocutaneous, allergic, hematological, and genetic disorders [47].

1.1.1.2 Risk factors for periodontal disease

Extensive epidemiologic and experimental evidence exists for the role of several risk factors in the initiation, progression, and severity of periodontal disease. From the 1990s to the present time, a series of epidemiologic studies have been carried out that measured a large battery of potential risk factors in large populations and identified those associated with periodontal disease [(5)]. In most of these studies, the results were statistically adjusted for confounding factors or correlations and isolated those that are likely to be true risk factors. Common confounding factors for periodontal disease include age, gender, and race/ethnic origin, with older, male, and nonwhite people having more periodontitis [(48)].

Genco and Borgnakke in 2013 [(5)] reviewed over 700 publications focusing specifically on systemic risk factors for susceptibility for periodontal disease that are relatively common in the population, and are likely to have a substantial population –attributable risk. They critically analyzed the past and present evidence supporting the management of risk factors as an essential component in the prevention and the treatment of periodontal disease in susceptible individuals.

In brief, the individual risk factors that were reviewed as follow;

- Gender, smoking, and alcohol (lifestyle).
- Diabetes.
- Osteoporosis, dietary calcium, and vitamin deficiency.
- Stress.
- Genetic factors.
- Obesity and metabolic syndrome.

- Gender as a risk factor for periodontal disease:

The most prevalent risk factor for periodontal disease is being male. It has been recognized that men of all ages, race/ethnic groups, and geographic locations have significantly more periodontal disease than women, assessed by prevalence, extent, and severity. From the data collected and analyzed by the National Health and Nutrition Examination Survey (NHANES III) 2009–2010, men had about 50% higher prevalence of periodontitis than women, with over half the men affected (56.4%): 10.0% had mild, 33.8% moderate, and 12.6% severe periodontitis [(5)].

- Smoking as a risk factor for periodontal disease

Cigarette smoking has long been associated with periodontal disease, and suggested as probable major risk factor for subsequent tooth loss. When adjustments for confounding and correlations were made, smoking was one of the major risk factors, with odds ratios for moderate smoking of 4–5, both for periodontal attachment loss as well as for alveolar crest bone loss. Tonetti in 1998 reviewed studies relating smoking to periodontal disease, which showed that smokers have both increased prevalence and more severe extent of periodontal disease, as well as higher prevalence of tooth loss and edentulism, compared to non-smokers. It was found that the greater severity of periodontal destruction may be partly accounted for by the reported increases in the rate of periodontal disease progression. It was concluded that smoking is an independent risk factor and the effect appeared to be dose dependent, with heavy smokers (> 10 cigarettes / day) presenting with higher levels of disease progression [(49)].

- Alcohol consumption as a risk factor for periodontal disease

Analyzing Data of NHANES III 2009–2010 from 13,198 employed adults 20 years and older, showed that there was a significant, linear relationship between number of alcoholic drinks per week and log clinical attachment loss (P = 0.0001). Odds ratios (95% confidence interval) for the

risk of attachment loss, using 5, 10, 15, and 20 alcoholic drinks per week as cut-off points, were 1.22 (1.02–1.47), 1.39 (1.13–1.71), 1.54 (1.22–1.93), and 1.67 (1.25–2.23), respectively. Further studies are necessary to understand more fully the association between alcohol consumption and periodontal disease [(5)].

- Diabetes mellitus as a risk factor for periodontal disease

Considerable evidence has been presented to support the concept that diabetes mellitus (types 1 and 2, and gestational) is a risk factor for periodontitis by increasing the prevalence, severity, extent, progression, possibly the initiation (incidence) of periodontal disease. There is also evidence that worsened glycemic control is associated with more periodontal disease, and that diabetic patients with good glycemic control may suffer from little or no periodontal disease. It appears that this is a bidirectional relationship (i.e., patients with periodontal disease and diabetes mellitus suffer from worsened glycemic control as evidenced by elevated glycated hemoglobin) and that patients with severe periodontitis and diabetes mellitus suffer from more cardiorenal mortality and microalbuminuria than do patients with diabetes who have little or no periodontal disease [(5)].

- Osteoporosis as a risk factors for periodontal disease

Martinez-Maestre and coworkers in 2010 published a systematic review of the relationship of osteoporosis to periodontitis, tooth loss, and mandibular atrophy. They report that 35 studies were considered suitable for selection, and most studies showed that systemic osteoporosis was associated with mandibular osteoporosis, and that systemic osteoporosis was associated also with increased tooth loss. Only 15 studies assessed the association of osteoporosis with periodontitis, using measurements of alveolar crest height or alveolar bone height, and others assessed

periodontitis on the basis of clinical attachment loss. Approximately half of the studies that assessed periodontitis on the basis of alveolar crest height or alveolar bone height found a positive association with systemic bone loss, while the other half found no correlation. Similarly, of those studies that used clinical attachment loss to assess periodontitis, about half showed a positive association with systemic bone loss. These studies vary in size, periodontal measurement method, and also in the systemic bone measurement. Hence, the variability in these studies could explain this lack of consensus. In spite of this lack of consensus, it was concluded that there is suggestive evidence of an association of osteoporosis and periodontal disease [(50)].

- Depression of dietary calcium and vitamin D as risk factors for periodontal disease

The role of dietary calcium in periodontal disease has also been studied and an inverse relationship was found. Nishida et al. in 2000, reported from the NHANES III data that association of lower dietary calcium intake with periodontal disease was found for young males and females (20 to 39 years of age), and for older males (40 to 59 years of age). The relationship between low dietary calcium intake and increased levels of periodontal disease showed an estimated odds ratio (OR) of 1.84 (95% CI: 1.36 to 2.48) for young males, 1.99 (95% CI: 1.34 to 2.97) for young females, and 1.90 (95% CI: 1.41 to 2.55) for the older group of males. These odds ratios were adjusted for gingival bleeding and tobacco consumption. These results suggest that low dietary intake of calcium results in more severe periodontal disease. Further studies will be needed to better define the role of calcium in periodontal disease [(51)].

- Stress, distress, and coping skills as risk factors for periodontal disease

Many controlled cross-sectional studies and randomized controlled trials have addressed the role of psychological stress, distress and coping as they affect the more common adult chronic

periodontal disease and suggested a correlation between chronic periodontal disease and the psychosocial stress status of patients. These studies have been summarized in two systematic reviews; one was reported by Peruzzo et al. in 2007; they reviewed 58 articles and only fourteen articles (seven case-control studies, six cross-sectional studies, and one prospective clinical trial) were included in the analysis. 57.1% found a positive outcome between psychosocial factors/stress and periodontal disease, 28.5% observed a positive outcome for some characteristics and a negative outcome for others, and 14.2% found a negative outcome [(52)]. In another systematic review by McCracken in 2009, one prospective clinical trial, seven case-control studies and six cross-sectional studies were included. Eight studies found a positive outcome between psychosocial factors or stress and periodontal disease; four studies observed a positive outcome for some characteristics; whereas two studies found a negative outcome between psychosocial factors or stress and periodontal disease. Both authors concluded that within the limitations of their review, the majority of studies showed a positive relationship between stress or psychological factors and periodontal disease. In the future, however, well-designed and more representative studies should be considered to confirm these factors as a risk for periodontal disease [(53)].

- <u>Genetic factors as risk factors for periodontal disease</u>

Evidence for the role of a genetic component in chronic (adult) periodontitis has been conducted from twin and family studies. The twin model is probably the most powerful method to study genetic aspects of any disease, including periodontal disease. Michalowicz et al. in 2000 evaluated the periodontal conditions (attachment loss, pocket depth, gingival index, and plaque index) of 110 adult twins with a mean age of 40 years ranging from 16 to 70 years. The results indicate that between 38% and 82% of the population variance for these measures may be attributed to genetic factors [(54)].

It has been hypothesized that some genes may also modify periodontal disease. It also is clear that other genetic factors, such as gene-gene interactions and gene-environmental interactions (epigenetic factors) may be important in the periodontal development of periodontal disease. Some studies suggested familial aggregation of disease. Meng and co-workers in 2011 reviewed genetic studies of families affected with aggressive periodontitis. Their review of the literature showed that the familial aggregation of aggressive periodontitis is often very high among certain families, with the percentage of affected siblings and affected pedigree members reaching 40–50%, suggesting that genetic factors may be important in susceptibility to aggressive periodontitis. They also found that family history is a valid representation of shared genetic and also shared environmental factors that contribute to an individual's periodontal status. Their analysis, however, could not distinguish between the genetic and the environmental factors. They reported that the basis for the heritability of periodontitis appears to be biological and not behavioral in nature [(55)].

An extensive review of gene polymorphisms in chronic periodontitis was carried out by Laine et al. in 2010. They present a comprehensive literature search up to 2009 and have analyzed studies looking at polymorphisms in the following: the interleukin-1 gene cluster; the tumor necrosis factor-a gene cluster; the interleukin-4 and interleukin-4RA genes; the interleukin-6 and interleukin-6R genes; the interleukin- 10 genes; the FcR genes; the vitamin D receptor genes; pattern recognition receptor genes such as CD-14; and a series of miscellaneous genes. Often the associations found are restricted to specific racial or ethnic groups. Overall, the evidence points to polymorphisms in the interleukin-1, interleukin-6, interleukin- 10, vitamin D receptor, and CD-14 genes as playing a role in chronic periodontitis, but most find that these associations are restricted to certain populations. They conclude that there is as yet no gene polymorphism that has

definitively been shown to be a risk factor for chronic periodontitis susceptibility in a broad representation of the population [56].

- Obesity as a risk factor for periodontal disease

This risk factor will be discussed in detail in the subsequent part of this chapter.

1.1.1.3 Epidemiology of periodontal disease

Epidemiology is the study of the distribution of disease in human populations and the factors that influence this distribution; heredity, biology, physical environment, social environment, and personal behavior [57].

Epidemiology research in periodontology must (1) fulfill the task of providing data on the prevalence of periodontal diseases in different populations (the frequency of their occurrence) , as well as on the severity of such conditions, i.e. the level of occurring pathologic changes; (2) elucidate aspects related to the etiology and the determinants of development of these diseases (causative and risk factors); and (3) provide documentation concerning the effectiveness of preventive and therapeutic measures aimed against these diseases on a population basis [58].

In the field of periodontal epidemiology there remains a need for well-designed, carefully analyzed, and correctly interpreted observational studies, including case-control studies, in order to adequately understand both the causes of periodontal disease as well as the associations between periodontal disease and systemic disease in populations [59].

The focus of the global periodontal epidemiology during the last half century has been on identifying populations who have periodontal disease and on disparities in disease prevalence between groups. Unlike dental public health activities directed toward dental caries, less effort has been made in periodontal epidemiology with regard to surveying or monitoring groups who may be more at risk for moderate or severe disease and in evaluating public health initiatives aimed at mitigating risk or reducing disease prevalence [60].

The prevalence and severity of chronic periodontal disease have been measured in population surveys undertaken in countries with the use of a variety of case definitions, a wide range of objectives, designs and measurement criteria [61].

The CPITN was developed in the late 1970s to facilitate performance of population-based surveys under a variety of conditions [62]. The CPITN was endorsed by the World Health Organization in 1997, who later renamed it the Community Periodontal Index (CPI). In UK and another parts of the world it is also known as the Basic Periodontal Examination (BPE), and Periodontal Screening and Recording (PSR).

WHO recommends the use of the Community Periodontal Index (CPI) for prevalence studies so that the results can be compared among different populations. It can be used as a tool with which countries may produce profiles of their periodontal health status and plan intervention programs for effective control of periodontal disease [47]. In addition, the CPI population data may be helpful in oral health surveillance at country and intercountry levels. While this index has certain shortcomings as a stand-alone means of assessing the extent and severity of periodontal disease, it has been widely used for descriptive periodontal epidemiological studies and for needs assessment

in both developed and developing countries. The major advantages of the CPI are simplicity, speed, reproducibility and international uniformity [63]. In 1997, the WHO suggested including information on loss of periodontal attachment in oral health surveys. However, data on loss of attachment are scarce as, to date, only a few countries have carried out such systematic surveys [63]. According to the WHO experience, the recording of loss of attachment is often considered difficult to carry out in the field and time-consuming [64].

Certain indicator age groups have been chosen by the WHO for intercountry comparisons of oral health status and oral health surveillance. The essential age-groups relevant to periodontal health are 15–19, 35–44 and 65–74 years. Over the past decades several countries have provided CPI data for warehousing in the WHO Global Oral Health Data Bank [65]. The standard parameters for presentation of CPI data are percentage of persons by their maximal CPI score (prevalence rate) and the mean number of sextants (severity) with a partial recording system CPI scores are; Score 0 = healthy periodontal conditions; Score 1 = gingival bleeding; Score 2 = gingival bleeding and calculus; Score 3 = shallow periodontal pockets (4–5 mm); Score 4 = deep periodontal pockets (≥ 6 mm); Score 9 = excluded; and Score X = not recorded or not visible. The extent of loss of attachment (LA) is recorded for sextants using the following codes: Score 0 = LA 0–3 mm; Score 1 = LA 4–5 mm Score 2 = LA 6–8 mm; Score 3 = LA 9–11 mm; Score 4 = LA ≥ 12 mm; Score X = excluded; and Score 9 = not recorded [64].

The most severe score or sign of periodontal disease (CPI score 4) varies worldwide from 10% to 15% in adult populations; however, the most prevalent score in all regions is CPI score 2 (gingival bleeding and calculus), which primarily reflects poor oral hygiene [66].

Overall, it appears that there is not enough current information to refute or support earlier estimates of severe periodontitis, which ranged from 5–15% of the adult global population [60].

In most epidemiologic studies carried out globally, significant relationships between socioeconomic i.e., low income or low education contribute to poor periodontal disease status [58, 61]. The distribution of periodontal disease within countries also differs according to race or ethnic groups regarding prevalence and severity [66].

The CPI databank is updated continuously and the population data is available in the WHO Global Oral Health Data Bank. These are displayed through a component of the so called WHO Country ⁄ Area Profile Programme (http://www.dent.niigata-u.ac.jp/prevent/perio/contents.html).

In Summary, Dye Bruce in 2012 reported that the periodontal status of populations globally in the first decade of the 21st century is not known. Fewer than 10 national or regional oral health surveys have included periodontal assessments, with the majority being performed in the developed world. Among these, only five countries reported findings using comparable methodologies that permit assessments of general trends. Among adults under 55 years of age, pocket depths ≥4 mm have decreased in Australia, the UK and the USA, but has increased in Germany and Hungary. Methodologies for practicing periodontal epidemiology are in flux again. Large epidemiological studies are shifting away from focusing on pocket formation (CPI methodology) to assessing loss of attachment. Both smaller epidemiological studies and large national surveys are now more likely to include a combination of pocket depth and attachment loss to describe periodontitis, and these efforts are now promoting the creation and use of standardized case definitions for population-based studies [60].

1.1.2 Review of the literature on obesity

1.1.2.1 Definition and classification of obesity; WHO

Recognition of obesity as a disease was in theory established in 1948 by the WHO introducing it on the International Classification of Disease as ICD-10-CM E66.9, but the early highlighting of the potential public health problem in the United States and in the United Kingdom 35 years ago was considered irrelevant elsewhere. The medical profession disregarded obesity as important despite the new evidence, and WHO data set out in the 1980s. Only in 1995 did WHO find greater problems of overweight than underweight in many developing countries, but it required the first special obesity consultation in 1997 and particularly the Millennium burden of disease analyses to suddenly highlight its crucial role in the current unmanageable and escalating medical costs globally [(67)].

Obesity is defined as abnormal or excessive fat accumulation that may impair health. Globally the fundamental cause of obesity and overweight is an energy imbalance between calories consumed and calories expended. There has been an increased intake of energy-dense foods that are high in fat; and an increase in physical inactivity due to the increasingly sedentary nature of many forms of work, changing modes of transportation, and increasing urbanization [(1)].

In addition, a WHO report in 2017, demonstrated the adverse effect of obesity on chronic health outcomes among adults. Obesity is considered a major risk factor for non-communicable diseases such as: cardiovascular diseases (mainly heart disease and stroke), which were the leading cause of death in 2012; diabetes; musculoskeletal disorders (especially osteoarthritis – a highly disabling degenerative disease of the joints); some cancers (including endometrial, breast, ovarian, prostate, liver, gallbladder, kidney, and colon).The risk for these non-communicable diseases increases, with increases in BMI [(1)].

1.1.2.2 Measurements of obesity

By the early 1990s, the prevalence of childhood malnutrition had become a major political issue, so there was a need to ensure appropriate methods for its assessment. WHO convened four teams in 1993 to consider how to assess a nation's problem of either malnutrition or obesity in both children and adults [67].

The WHO [38] uses Body Mass Index (BMI) to categorize adults as overweight or obese. It is defined as a person's weight in kilograms divided by the square of height in meters (kg/m²). BMI is a simple index of weight for height, which provides the most useful population-level measure of underweight, overweight and obesity as it is the same for both sexes and for all ages of adults. However, it is significantly correlated with total body fat content, and it should be considered a rough guide because it may not correspond to the same degree of fatness in different individuals.

$$\text{i.e. BMI} = \frac{\text{Wt (kg)}}{\text{Ht (m)}^2}$$

The WHO [38] has recommended classifications of bodyweight that include degrees of underweight and gradations of excess weight or overweight that are associated with increased risk of some non-communicable disease. The internationally accepted ranges of BMI used to define degrees of overweight and the risk of its co-morbidities are shown in table 1:-

Table 1: Internationally accepted ranges of BMI used to define degrees of overweight.

WHO classification	BMI (kg/m²)	Risk of co-morbidities
▪ Underweight	< 18.5	Low (but risk of other clinical problems increased)
▪ Normal range	18.5-24.9	Average
▪ Overweight	25.0-29.9	Mildly increased
▪ Obese	> 30.0	
Class I	30.0-34.9	Moderate
Class II	35.0-39.9	Severe
Class III	> 40.0	Very severe

The 1997 WHO Expert Consultation on Obesity recognized the importance of abdominal fat mass (referred to as abdominal, central or visceral obesity), which can vary considerably within a narrow range of total body fat and body mass index (BMI). It also highlighted the need for other indicators to complementing the measurement of BMI, to identify individuals at increased risk of obesity–related morbidity due to accumulation of abdominal fat [(64)]. Waist–hip ratio (i.e. the waist circumference divided by the hip circumference) was suggested as an additional measure of body fat distribution. The ratio can be measured more precisely than skin folds, and it provides an index of both subcutaneous and intra–abdominal adipose tissue [(68)]. The suggestion for the use of proxy anthropometric indicators arose from a 12 year follow–up of middle–aged men, which showed that

abdominal obesity (measured as waist–hip ratio) was associated with an increased risk of myocardial infarction, stroke and premature death, whereas these diseases were not associated with measures of generalized obesity such as BMI [(69)]. In women, BMI was associated with an increased risk of these diseases; however, waist–hip ratio appeared to be a stronger independent risk factor than BMI [(70)].

Waist circumference is a convenient and simple measurement that is unrelated to height, correlates closely with BMI and is an approximate index and a valid measure of intra-abdominal fat mass and disease risk in individuals with body mass index less than 35. If BMI is 35 or more, waist circumference adds little to the absolute measure of risk provided by BMI. It also provides a clinically acceptable measurement for assessing a patient's abdominal fat content before and during weight loss treatment [(71)].

WHO uses two gender specific waist circumference categories (Table 2) to classify the risk for metabolic complications like Diabetes Mellitus type 2, hyperlipidemia, hypertension and cardiovascular disease.

Table 2: Gender specific waist circumference and risk of metabolic complications.*

Risk of metabolic complications	Male	Female
Increased risk	> (94cm) >37 inches	> (80cm) >32 inches
significantly increased risk	> (102cm) >40 inches	> (88cm) >35 inches

*Adapted from WHO consultation report Series 894.Obesity: Preventing and Managing the Global Epidemic. Geneva -2008.

Waist circumference should be measured at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest, using a stretch-resistant tape that provides a constant 100g tension. Hip circumference should be measured around the widest portion of the buttocks, with the tape parallel to the floor. For both measurements, the subject should stand with feet close together, arms at the side and body weight evenly distributed, and should wear little clothing (Figure 1). The subject should be relaxed, and the measurements should be taken at the end of a normal expiration. Each measurement should be repeated twice; if the measurements are within 1 cm of one another, the average should be calculated. If the difference between the two measurements exceeds 1 cm, the two measurements should be repeated [71].

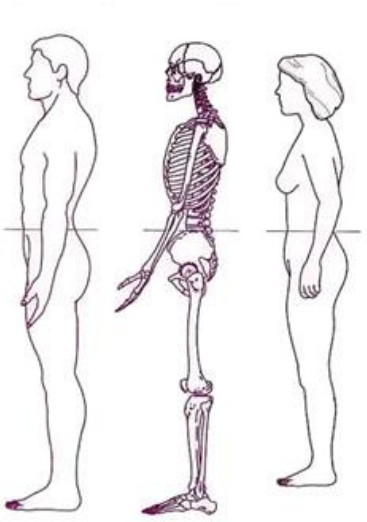

Figure 1: Measuring-Tape Position for Waist (Abdominal) Circumference in adults.

1.1.2.3 Prevalence and trends in obesity; Arab countries, GCC & Bahrain

The worldwide prevalence of obesity nearly tripled between 1975 and 2016. Overall, about 13% of the world's adult population (11% of men and 15% of women) were obese in 2016 [(1)]. Unlike other major global health risks such as tobacco and childhood malnutrition, obesity is not decreasing worldwide. Rates of obesity seem to be increasing in both developed and developing countries where almost two in three of the world's obese people live. Obesity is already a major public health challenge in many middle-income countries [(2)].

Across the Middle Eastern region the prevalence of obesity ranks among the highest in the world. The latest national data from the Middle East, reported by Kilpi et al. in 2014, revealed worryingly high obesity rates in many countries. The projections from this analysis demonstrated how the obesity epidemic is not abating. In particular, the results for Kuwait, Oman, Saudi Arabia, Lebanon, Turkey, Bahrain and Jordan showed that the problem of obesity is growing. Given that obesity is a risk factor for many Non Communicable Diseases (NCD), this has major implications for the public health systems of these countries. They also show the trends for combined overweight and obesity (BMI≥25kg/m2) prevalence (termed 'overweight') for men and women of all ages in each country. It is predicted that, by 2030, both Kuwaiti men and women will top the region with regard to overweight and obesity, both at exceedingly high levels. Saudi Arabian men and women also have consistently high levels of overweight, but the expected increase in overweight appears particularly steep for men in Oman. Bahrain and Jordan all have high levels of overweight, with moderate increases over time. The data for Egypt, on the other hand, suggest decreasing overweight for both men and women. Decreases are also shown for men in Iran. Tunisia and the United Arab Emirates were not included as not enough suitable data were available at the time of analyses. They also stated that over 115000 cases of coronary heart disease (CHD) and

stroke and over 200000 cases of type 2 diabetes would be avoided with a 1% decrease in obesity by 2030 [36].

In a previous review reported by Musaiger et al. in 2011, that the prevalence of overweight and obesity among adults in Eastern Mediterranean region ranged from 50 to 80%, with higher prevalence among women than men [35]. One study covering fifty-two countries from eight geographical regions worldwide showed that adult men and women in the Middle East have the highest mean BMI after the USA [72].

Additionally, the women in this region have the highest waist-to-hip ratio compared with all other regions [72]. Although the Middle Eastern region is characterized by different socio-economic levels, the prevalence in high-income countries (such as Arab Gulf states) and middle-income countries (such as Iran, Egypt, Jordan and Lebanon) was very similar, indicating that cultural rather than economic factors determine obesity in this region [36] .

Most evidence from several studies indicates that obesity is a major public health issue in Arab countries although it varies widely from one Arab country to the other [18]. Overweight and obesity have been reported as ranging from 25% to 38% among adult men, and 28% to 83% among adult women [73] (Table 3). It may be that the differences between countries may, in part, be influenced by dietary and /or climate differences.

Table 3: National prevalence of overweight and obesity among adults (15+ years) in selected Arab countries.

Country	Date of Survey	Sex	Overweight (%)	Obesity (%)
Bahrain	2007	**M**	34.8	32.3
		F	35.1	40.3
Egypt	–	**–**	–	–
Kuwait	2007	**M**	38.9	39.2
		F	28.9	53.0
Lebanon	1995–1996	**M**	43.4	14.3
		F	30.6	15.5
Libya	2000	**M**	19.2	5.8
		F	21.1	7.1
Morocco	1995–1999	**M**	28.0	05.7
		F	33.0	18.3
Oman	2000	**M**	30.6	15.5
		F	27.2	22.3
Qatar	–	**–**	–	–
Palestine	2002	**F**	–	10.9
S. Arabia	2005	**M**	43.0	31.5
		F	28.8	50.4
Tunisia	2005	**M**	51.7	37.0
		F	71.1	12.3
UAE	–	–	–	–

Ng et al. in 2011 reviewed studies on the prevalence of overweight, obesity and related nutrition-related non-communicable diseases in Bahrain, Kuwait, Qatar, Oman, Saudi Arabia and the UAE. They reported that obesity is common among women; while men have an equal or higher overweight prevalence. Among adults, overweight plus obesity rates are especially high in Kuwait, Qatar and Saudi Arabia, and especially among 30–60 year olds (70–85% among men; 75–88% among women), with lower levels among younger and elderly adults. The rate of increase in obesity was pronounced in Saudi Arabia and Kuwait. Prevalence of obesity was high among Kuwaiti and Saudi preschoolers (8–9%), while adolescent overweight and obesity are among the highest in the world, with Kuwait having the worst estimates (40–46%) [(74)].

Data on obesity in the Arab Region showed that even in middle and low income countries, the prevalence of obesity is high. The Arab Gulf countries showed the highest prevalence of obesity among adults, however countries like Egypt, Morocco and Tunisia have a similar trend in obesity. Characteristically, obesity in most Arab countries was reported to be more prevalent among women, urban dwellers, married, non-smokers, the inactive and high socio-economic classes. The prevalence of overweight and obesity among women may be attributed in part to the high parity among women in most Arab countries [(75)].

Unlike western countries, obesity in Arab countries is more prevalent among women in urban areas and in the higher social class. In Jordan, for example the prevalence of obesity was 56% in urban areas compared to 44% in rural areas. Similar trends were found in Egypt, Morocco, Oman and Tunisia [(73)]. Lebanon was an exception as obesity was more prevalent among rural than urban women. Interestingly, obesity occurred more among unemployed than employed women in several Arab countries. In Kuwait, for example, the percentage of obesity in unemployed women was 47% compared to 34% in employed women. In Saudi Arabia, the proportions were 79% and 53%, and in Tunisia the percentages were 24% and 15%, respectively [(75)]. This may be due to the fact that employed women are generally younger. It is also believed that the exposure of employed women to the community at work may possible put pressure on them to take care of their weight [(73)].

In Bahrain specifically, overweight and obesity are major causes of ill-health and create a huge burden on health care resources and the national economy [(39)] . The only published national non-communicable diseases risk factor survey from an adult sample of around 2037 revealed that the overall prevalence of obesity was 36.3% and was higher in females than males (40.3% and 32.3%, respectively). On the other hand, the overall prevalence of overweight was 32.9% and was higher in male respondents than females (34.8% Vs 31.1%). Obesity was most prevalent (46.1%) in the

age of 40-49 years and least prevalent (20%) in the youngest age of 20-29 years. On the other hand, overweight prevalence was highest (51.7%) in the oldest age group. The study also showed that among male respondents the prevalence was highest in the age group 40-49 years (43.4%), while in female respondents the prevalence was highest in the age group 50-59 years (51.4%). In contrast, the prevalence of obesity was the lowest in the age group 20-29 years for both male and female respondents [37].

1.1.2.4 Nutrition Clinics in Ministry of Health, Bahrain

The Bahrain government demonstrated increased awareness towards the growing obesity epidemic with widespread interest among health professionals and policy makers in the MOH to tackle the obesity-related health problems of the population in Bahrain. In response, a "Comprehensive National Action Plan for Prevention and Management of Overweight and Obesity" was drafted in 2005. Hereafter, the Nutrition Clinic project was established, aiming to provide high standards of obesity management to all overweight and obese adults in the Kingdom of Bahrain in the primary care setting, and to ensure that the huge number of patients in Bahrain society who suffer from weight related problems will receive early, appropriate and a long-term management of obesity and its associated morbidities, preventing the situation from additional deterioration [40].

The Nutrition Clinic was launched on 27th Nov 2007, and the first Nutrition Clinic was established in National Bahrain Banks (NBB) –Arad Health Centre in Muharraq Governorate in 2009. Among the reasons for opening the first Nutrition Clinic in Muharraq was the high prevalence rate of overweight and obesity in the region served by this center. After successful results of the pilot phase, the nutrition section supported opening the second nutrition clinic in September 2009; Al-Dair Health Centre in Muharraq Governorate [40].

Currently, there are five nutrition clinics distributed in different Governorates in the Kingdom of Bahrain for treatment of obesity in adults 19 years and above, and funded by the Nutrition Section, Ministry of Health.

Different health professionals are involved in delivering the care management to overweight patients; family physicians, nurses and nutritionists. The care management involves [40];

1- Assessment of patients includes the following;

a. Clinical assessment including taking different blood tests (Hb, FBS, S.Creatinine, Uric Acid, Total Cholesterol, LDL, HDL, Triglyceride, Thyroid Function Test, ...).
b. Blood Pressure (BP).
c. General Physical examination.
d. Dietary history.
e. Lifestyle Assessment.
f. Anthropometrics;

- BMI = $\frac{\text{weight (kg)}}{\text{height (m)}^2}$
- Waist Circumference

2- Assessment of risk factor includes the following;

a. Determine the relative risk status.

b. Identify cardiovascular risk factors.

c. Assess the presence of underlying disease and conditions.

d. Assess other risk factors.

3- Patient motivation which is a principal factor in any successful weight loss program aims;

a. To try to predict patient's readiness for weight loss and willingness to make the necessary lifestyle changes.
b. To identify potential variables associated with weight loss success.
c. To explain to patients the negative health effect of overweight or obesity and enumerate the dangers that accompany persistent obesity such a hypercholesterolemia, hypertension, diabetes, etc.

In addition, the Nutrition Section has developed a number of patient assessment tools and educational materials. These include the following [40]:

- Patient Assessment Sheet.
- Food frequency questionnaire.
- 7-days menu plans.
- A patient Educational booklet: Obesity Management Guide and Food Composition Booklet.
- Patient self-care booklet.
- Nutrition education package (flip chart and educator manual guide).
- Posters and flyers.
- Appointments cards.

1.1.3 Review of association between periodontal disease and obesity

1.1.3.1 Association between periodontal disease and obesity

The relationship between obesity/ overweight and periodontal disease had been explored in many cross-sectional studies, cohort studies, systematic reviews and meta-analysis, which report a significant positive association between obesity and periodontal disease prevalence, and suggest that overweight/obese individuals are more likely to suffer from periodontitis compared to normal weight individuals. Thus, indicating that obesity could be a potential risk factor for periodontal disease.

This relationship was first reported by Perlstein and Bissada in 1977 in experimental animals, to evaluate the extent to which obesity and /or hypertension may modify the response of rats' periodontium to chronic gingival irritation. The histopathological evaluation of the periodontal structure showed both hyperplasia and hypertrophy of the walls of blood vessels supplying the periodontium in the hypertensive and obese animals. The results also indicated that obesity significantly contributed to the severity of periodontal disease. Hypertension alone was not a significant factor. They concluded that obese –hypertensive rats showed the most severe periodontal response to local irritation [(76)]. In another animal study, Verzeletti et al in 2012 found that obesity potentially influences the pathogenesis of experimental periodontitis (ligature-induced periodontitis), leading to higher alveolar bone loss in female Wistar rats [(77)] .

A subsequent human study conducted by Saito et al. in 1998 in Japan including 241 healthy individuals in whom the CPITN was measured, reported that obese Japanese subjects were more

likely to have periodontal disease, and a RR , and a significant association was found between obesity and an increase risk of periodontitis [22].

Al-Zahrani et al. in 2003, analyzed the data collected from a cross-sectional survey conducted by a U.S. national center for Health Statistics, the Third National Health and Nutrition Examination Survey (NHANES III) involving 13,665 individuals, who were ≥18 years and had undergone a periodontal examination were selected, BMI and WC were used as measures of overall and abdominal fat content, respectively. A significant association was found between the measures of body fat and periodontal disease among the younger adults aged 18 to 34, but not in the middle-aged 35 to 59 and older 60 to 90 years. Young subjects with BMI ≥ 30kg/m² and high WC had an adjusted OR of 2.27 (1.480 to 3.487) for having periodontal disease. The prevalence of periodontal disease in obese young people was, by approximately 76% significantly higher compared with those of the same age with normal body weight [23].

In addition Dalla Vecchia et al. in 2005, investigated a sample comprising 706 subjects aged 30 to 65 years from south Brazil. They were examined clinically and completed a structured interview. Overweight and obesity were assessed by body mass index (BMI) using (WHO) criteria. Individuals with ≥ 30% teeth with attachment loss ≥ 5 mm were classified as having periodontitis. They found that Overweight and obesity were prevalent in this adult Brazilian population. Obesity was significantly associated with periodontitis among females, with obese females showing an 80% higher chance of having periodontitis than females of normal weight. The association of periodontitis with obesity was stronger in non-smoker females adult. Hence, female nonsmokers were 3.4 times more likely to be diagnosed with periodontitis compared to female non-smokers with normal weight. No significant association was found between periodontitis and overweight/obesity among males in this population, irrespective of smoking status. They

concluded that obesity was significantly associated with periodontitis in adult, non-smoking women. Overweight was not significantly associated with periodontitis. Smoking may attenuate the association of periodontitis with obesity [24].

In Arab countries the relationship between obesity and periodontitis was first reported by an epidemiological study conducted by Khader et al. in 2008 in Jordan, they aimed to determine the relationship between periodontitis and overweight/obesity among a sample of 340 Jordanians aged between 18 and 70 years, using different indicators including BMI, WC, WHR, and body subcutaneous fat per cent. All participants underwent periodontal examination. Periodontitis was defined as presence of four or more teeth with one or more sites with probing pocket depth ≥4mm and clinical attachment loss ≥3 mm. The results showed that 51.9% of obese participants had periodontal disease. It was more prevalent among subjects with high waist circumference (WC) and among subjects with high waist-to-hip ratio. After adjusting for important variables, only body mass index (BMI)-defined obesity, (OR= 2.9, 95% CI: 1.3, 6.1), high WC (OR=2.1, 95%CI: 1.2, 3.7), and high fat per cent (OR=1.8, 95% CI: 1.03, 3.3) remained significantly associated with increased odds of having periodontitis. [19]. In addition, El-Sayed Amin in 2010 investigated the relationship between overall and abdominal obesity and periodontal disease among 380 adults (170 males and 210 females) aged 20–26 years in Egypt. Body mass index (BMI) and waist circumference (WC) were measured and clinical attachment loss (CAL), gingival index (GI) and Community Periodontal Index (CPI) were estimated. His findings showed a statistically significant positive correlation between obesity in terms of BMI and WC and periodontal measurements of CAL, GI and CPI in female participants. In males, a significant correlation was only recorded between WC, GI and CPI. He concluded that the overall and abdominal obesity in young adult females and abdominal obesity in males were significantly associated with periodontal disease [20].

Several systematic reviews and meta-analyses have been conducted to pool studies evaluating the association between obesity / overweight and periodontitis in adults. Chaffee and Weston in 2010 aimed to systematically compile the evidence of an obesity-periodontal relationship from epidemiologic studies and to derive a quantitative summary of the association between these disease states. Nearly all studies (70 studies) matching inclusion criteria were cross- sectional in design. Of the 70 studies, 41 suggested a positive association. The fixed –effect summary odd ratio was 1.35(95% confidence interval: 1.23 to 1.47), with strong evidence of a stronger association found among younger adults, women, and non-smokers. Additional summary estimates suggested a greater mean clinical attachment loss among obese individuals, a higher mean body mass index (BMI) among periodontal patients, and a trend of increasing odds of prevalent periodontal disease with increasing BMI [(7)].

One year later, Suvan et al. in 2011 provided a systematic review and meta-analysis of the available evidence investigating such an association, based on BMI, and to provide a standardized guide for future study design. Their results support an association between BMI overweight and obesity and periodontitis although the magnitude was unclear, and suggested appositive association, based upon cross- sectional surveys and case control analyses. They also suggested additional prospective studies to further quantify or understand the mechanism of this association [(6)]. Therefore, Suvan et al. in 2015 did another study to investigate periodontitis as a co-morbidity of overweight/obesity in an age-matched sample of periodontitis cases or periodontally healthy controls in a sample representative of the UK population. The odds ratio was used for diagnosis of periodontitis when BMI (kg/m^2), (overweight BMI 25-29.9 kg/m^2, or obese BMI $\geq$ $30kg/m^2$) were the explanatory variables. The results showed that BMI had a dose-response association with increased odds (1.12 per increase of 1 kg/m^2, 95% CI 1.05-1.20, P=0.001) of being a case

compared to control independent of gender, ethnicity, smoking status and dental plaque level. Similarly overweight/obese were independently associated with increase odds of diagnosis of periodontitis for overweight (OR = 2.56, 95% CI 1.210-5.400, p= 0.014) and for obese (OR = 3.11, 95% CI 1.052-6.482, p= 0.015) compared to normal weight individuals. They concluded that the odds of having periodontitis are more likely if an individual is overweight or obese compared to normal weight individuals in this case-control sample [8].

Moreover, Nascimento et al. in 2015 conducted the first systematic review and meta-analyses analyzing only prospective longitudinal studies exploring the association between weight gain and its effects on the incidence of periodontitis in adults. Only five studies were eligible to be included in this review. All studies were conducted in high income countries, with one in Finland (Saxlin et al. 2010), two in Japan (Morita et al. in 2011, Ekuni et al. in 2014) and two in United States of America (Gorman et al. in 2012b, Jimenez et al.in 2012). Body mass index was used in all articles, however only WC and WHR were used in conjunction with BMI in studies conducted in United States of America. Pooled relative risks(RR) for becoming overweight and obese on the incidence of periodontitis were estimated by meta-analysis, and showed that subjects who became overweight and obese presented higher risk to develop new cases of periodontitis (RR 1.13; 95% CI 1.106-1.20 and RR 1.33; 95% CI 1.21-1.47 respectively) compared with counterparts who stayed at a normal weight. They concluded that a clear positive association between weight gain and new cases of periodontitis was found. However, these results originated from limited evidence (limited studies identified after extensive and careful searches), thus, more studies with longitudinal prospective design are needed not only in high-income countries, but especially in those of low-and middle-income, where the prevalence of obesity and periodontitis is greater [9].

One prospective study conducted by Gaio et al. in 2016 investigated the effect of obesity on periodontal attachment loss (PAL) progression in an urban population from south Brazil, 582 (333 males/249 females) individuals were included. The progression of periodontitis was defined as proximal PAL of ≥3mm in ≥4 teeth over 5 years of follow-up. They found that females who were obese at baseline were at statistically higher risk of periodontitis progression than those who were normal weight, with a relative risk of 1.64(95% CI 1.11-2.43). No association was observed for males with a relative risk of 1.13(95% CI 0.75-1.69).This suggests obesity in this population from south Brazil was a risk factor for PAL progression in females but not in males. A major limitation was the high dropout rate, at baseline,1568 subjects were screened but only 755 re-attended at 5 years. A further limitation was the lack of diabetes assessments. This is particularly important in obesity studies as it is well established that diabetes is associated with both obesity and periodontitis [(31)].

Conversely, few cross-sectional studies indicated that there is lack of association between obesity and periodontitis. Kongstad et al. study in 2009 was to evaluate the possible relationship between body mass index (BMI) and periodontitis, measured as mean clinical AL and BOP, in a sample of adult Danes, 878 women and 719 men aged 20 to 95 years (participation rate 54%) underwent an oral examination, including full-mouth recording of clinical attachment level (AL) and BOP. Overweight and obesity were assessed by body mass index (BMI) using the WHO criteria. BMI was related to clinical AL (defined as mean ≥3 mm) and BOP (defined as ≥25%).They found that the obese participants had a lower odds ratio (OR) for clinical AL compared to participants with normal weight (OR: 0.60; 95% CI: 0.36 to 0.99). The same tendency was observed in subjects stratified by smoking habit. Obese never-smokers had a lower OR for clinical AL compared to never-smoking participants with normal weight (OR: 0.32; 95% CI: 0.11 to 0.91). Overweight

participants had a higher OR for BOP compared to subjects with normal weight (OR: 1.36; 95% CI: 1.04 to 1.78). In addition, overweight never-smokers had a higher OR for BOP compared to normal weight never-smokers (OR: 1.63; 95% CI: 1.03 to 2.59).Accordingly, they reported an inverse relationship between obesity and clinical AL and a slightly positive association between BMI and BOP. BMI was not a very strong risk indicator for periodontitis in the present cohort [(78)].

In addition, de Castilhos et al. in 2012 reported that among a group of 720 individuals, aged between 15 and 23 years old (a representative sample of young adults participating in a birth cohort oral health survey for 24 years), the presence of periodontal pockets was neither related to obesity (BMI $\geq$30kg/m^2) nor to waist circumference, and that the cumulative effect of obesity was only observed for dental calculus. They concluded that although no association was found between measures of obesity and periodontitis in this cohort, the study identified an association between the number of episodes of obesity and risk for dental calculus and also between waist circumference and the number of teeth with gingivitis. They also mentioned that by using only the measurement of periodontal pocket depth to assess the presence of periodontitis, its prevalence may have been underestimated due to possible clinical attachment loss without the presence of pockets [(79)].

Recently, Awad et al. in 2015 in a cross-sectional study, reported that among 186 Arab patients with type 2 diabetes mellitus (over half (61%) had a BMI $\geq$ 30kg/m^2, (90%) were non-smokers), there was however, no relationship between periodontitis and BMI. Periodontitis was measured by full mouth recording of clinical attachment loss (CAL; >2mm vs $\leq$2mm). They mentioned that although the age of cases and controls were similar, there was no gender balance between the obese and control group, also they did not assess participants' oral habits and other life factors and therefore, the effect of variables such as frequency of brushing may have been informative [(21)].

1.1.3.2 The Mechanisms linking obesity and periodontal disease

Numerous studies have been explored the mechanisms by which obesity may lead to increased periodontal disease. If an association between obesity and periodontitis exists, that association most likely lies in the commonality of their inflammatory pathway [(17)]. A heightened inflammatory response is regularly observed in individuals in who are obese .Obesity is considered as a chronic low-grade inflammatory disease because of changes in adipose tissues [(16)]. Adipose tissue is an active endocrine organ, and it performs many important functions in the body, such as thermal insulation and protection, storage, and secretion. The cells of adipose tissue, called adipocytes, secrete dozens of biologically active molecules, which can significantly modulate reactions occurring in the body. These include leptin, resistin, tumor necrosis factor α (TNF-α), interleukins:IL-1, IL-6, IL-8 and IL-10, growth factors, complement components, angiotensinogen, plasma plasminogen, activator-1(PAI-1), and a number of other substances. Many cytokines are secreted proportionally to the amount of fat present and are actively involved in the metabolism of the whole system. Therefore, obesity may alter the response of the host antigen derived from bacterial plaque, and thus cause disturbances in the inflammatory response in the course of the periodontal disease [(14),(15),(80)]. Beck et al. in 1996 reported an up to tenfold increase in local and systemic inflammatory cytokines including TNF-α and IL-6 in some subjects with periodontitis [(12)]. These same cytokines can then trigger an increase in the production of acute phase proteins such as C-reactive protein, which results in an upregulation of the inflammatory response similar to that seen in obesity [(12),(81)] .

Pro-inflammatory cytokines may play a crucial role in the close relationships between periodontitis, obesity, and chronic diseases [10],[82]. In fact this association may be multidirectional (Figure 2) [83].

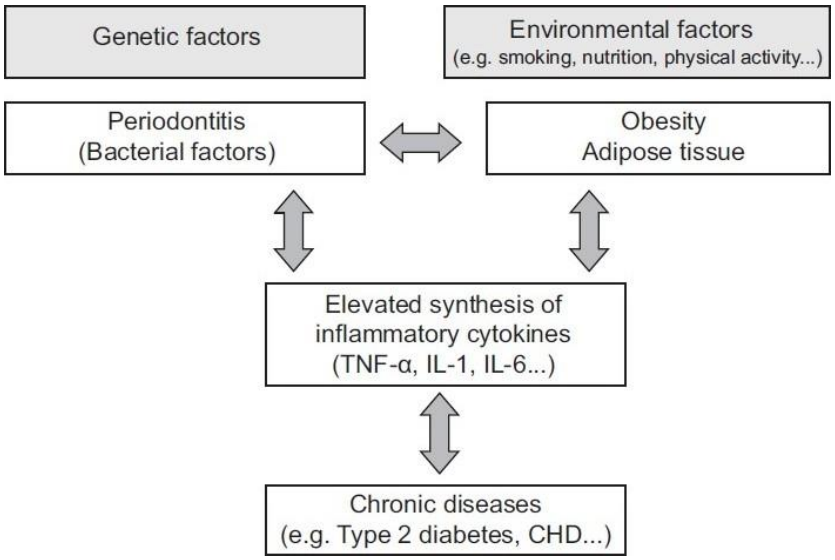

Figure 2: Model linking periodontitis, and obesity with inflammatory related chronic diseases.

For example, it has been well–established that inflammation is an essential component in the development of atherosclerosis, and observational studies have shown that periodontitis is associated with a moderate, but significantly, higher risk of coronary heart disease [10],[82]

Thanakun et al. in 2016 did a study to evaluate the effect of periodontitis on adiponectin, C-reactive protein(CRP) and immunoglobulin G against porphyromonas gingivalis in Thai people who were overweight or obese. They found that periodontitis and an overweight or obese BMI changed plasma levels of the inflammatory mediators adiponectin and CRP, independently. This study suggested the role of periodontitis in systemic inflammatory response in Thai people who were overweight or obese [84].

According to Genco et al. in 2005 , the relentless release of pro- inflammatory cytokines into the systemic circulation from adipose tissue in obese individuals provides a systemic inflammatory overload, increased cytokines may directly injure the periodontal tissues or reduce the vascular blood flow to the periodontal tissues [10], thus promoting the development of periodontitis as first suggested by the Perlstein and Bissada in 1977 study on a group of obese rats and their lean littermates, periodontal disease was induced, and the level of TNF-α was found to be significantly higher in the periodontitis and obesity group of rats compared with the control group. It was concluded that systemic low-grade inflammation combined with increase gene expression for hepatic levels of TNF-α and C-reactive protein and elevated adipose IL6 and C-reactive proteins, combined to make the obese rats more susceptible to periodontal disease [76].

In addition, the pro-inflammatory mediators (TNF-α receptor 1 and receptor 2) produced by adipose tissue and /or periodontal inflammation may contribute to insulin resistance. Thus in obesity, insulin resistance may occur due to an increase in TNF-α [85]. Obese individuals with high insulin resistance appeared to have the most severe periodontitis [10] .

On the other hand, multiple mediators of inflammation and the immune response are detected in gingival crevicular fluid in periodontitis. Patients with the periodontitis show a significant increase in the levels of IL-6, IL-8 and TNF-α in gingival crevicular fluid [11],[86]. A positive correlation was demonstrated between the concentration of cytokines in gingival fluid and the BMI value. Bruun et al in 2003 found that plasma level of IL-6, IL-8 were 30-40% higher in obese as compared with lean subjects. In addition they found that weight loss in obese men was associated with a decrease in the circulatory levels of IL-6 and TNF-α by 25-30% ($p<0.001$), but an increase in circulatory levels IL-8 by 30% [13].

Blood vessels in the periodontium of obese people have a thickening on the most inner wall. It is believed that this is an effect of the action of plasma plasminogen activator -1 adipokine. Which is one of the pathogenic mechanisms of the development of periodontal disease [87].

Boesing et al. in 2009 stated that in obesity, factors such a hyperglycemic status, the presence of advanced glycation end products, modifications of saliva pH, and neutrophil dysfunction amplify the immune stress and affect the biological phenotype of periodontitis. Thus obesity appears to participate in the multifactorial phenomenon of causality of periodontitis through the increased production of reactive oxygen species (oxidative stress) and the inflammatory response, and this is likely because of the increase in proinflammatory cytokine [88].

There is evidence suggesting that changes in the pro-inflammatory and immune responses associated with obesity are due to epigenetic changes that may very well modify periodontal inflammation, increasing the susceptibility of obese individuals to periodontal disease.

Kalea et al. in 2015 identified differences in the microRNA gene expression profiles of gingival tissue in periodontitis when obesity is present; these genes play central roles in pathways involved in re-epitheliazation of a gingival wound, which is an important process in periodontal regeneration and re-attachment, affecting treatment outcome of tissue integrity [89]. However the specific molecular and cellular mechanisms responsible for the changes in pro-inflammatory and immune responses associated with obesity, are not yet clear and further studies are needed to unravel the mechanisms, which may target for prevention or treatment [5].

Haffajee and Socransky in 2009 compared the oral microbiome in obese and non-obese subjects with periodontally health and gingivitis or with chronic periodontitis. They found that the number of T.forsythia was significantly higher in obese individuals suffering from gingivitis, but who were

otherwise periodontally healthy, compared with the other group. They hypothesized that overgrowth of T.forsythia may occur in periodontally healthy individuals who are obese, putting them at risk for the initiation and progression of periodontitis [90].

In another study Rangé et al. in 2012 identify changes in the salivary protein/peptide profiles by differential proteomics in obese patients with or without periodontitis. They identified six increased putative markers (albumin, α and β haemoglobin chains, α defensins 1, 2 and 3) in obese subjects versus controls. Alpha defensins were more abundant in obese patients (40.99 ± 26.66 $\mathcal{M}$A) versus controls (27.1 ± 23.98 $\mathcal{M}$A). They conclude that periodontal status modifies the salivary proteome in obese patients. Alpha defensins may play a role in gingival inflammation, and be involved in the higher susceptibility of obese patients to periodontal diseases [91].

Recently, the Al Abdaly study in 2017 was designed to determine the relationship between severity of chronic periodontitis and salivary calcium (Ca_{+}) and phosphorous (P_{-}) among 450 overweight and obese Yemenis aged 20-50 years living in Abha city/Kingdom of Saudi Arabia. He found that salivary Ca_{+} and P_{-} levels were significantly and positively associated with periodontal pocket depth formation (PPD) ($P<0.05$), and concluded that the overweight and obesity in relation to PPD showed an increase in the salivary calcium and phosphorus values compared to the NO obese patients group [92].

1.1.4 Aim

The aim of this was to investigate the prevalence of periodontitis in obese adults in the Kingdom of Bahrain.

Specific objectives:

- To determine the prevalence of periodontitis among obese adult patients.
- To determine which measure of obesity (BMI, WC) is a better predictor of periodontitis.

CHAPTER TWO

1. MATERIALS AND METHODS

2.1 Study design, location and population

A cross-sectional study design was used to study the prevalence of periodontitis in obese adults. The obese/overweight participants were recruited from the Nutrition Clinics at Primary Health Centers, Ministry of Health in Bahrain from 28th December 2016 until 13th April 2017. Only cases who fulfilled the inclusion and exclusion criteria were invited to participate for periodontal examination. All participants had to provide voluntarily written consent.

2.1.1 Sample size

The sample size was calculated based on the probability of having chronic periodontitis among obese patients. For this purpose, the findings of Shahrukh Khan et al. in 2015 [30], which showed a prevalence of 73.6% in obese Malaysians was chosen for sample size calculation. In 2016 the number of registered patients in obesity clinics in Bahrain was approximately 1000. The calculated sample size was 354 using the formula of Cochran's sample size calculation for cross-sectional design:

$$N = \frac{Z^2_{\frac{\alpha}{2}}\, pq}{B^2}\, \mathrm{D}$$

Where

$$B = z_{\alpha/2}\sqrt{\frac{pq}{n}}$$

Where

P is a prevalence of periodontitis from the reference study.

q is (1-p)

zα/2 is the quartile of 95%, and B is the width of the confidence interval of 95% (error).

Using the above formula with error 0.05, the calculation yield 296 and adding 20% of nonresponse the size will be **354** participants.

2.1.2 Sampling technique

A convenience sampling method was used for the participant's recruitment. All obese/overweight patients who had appointments in the Nutrition Clinics at Primary Health Centers were invited to participate for periodontal examination.

2.1.3 Participating health centers

Five Nutrition Clinics for treatment of obesity at MOH Primary Health Centers in Bahrain were involved;

- Arad Health Center.
- Al-Dair Health Center.
- Bank of Bahrain and Kuwait Health Center.
- Hamad Kanoo Health Center.
- Hamad Town Health Center.

2.2 Inclusion and exclusion criteria

2.2.1 Inclusion criteria

- All participants were ≥ 18 years old.
- A minimum of 20 natural teeth excluding wisdom teeth.
- Systemically healthy (No active infectious diseases such as hepatitis, HIV or TB, uncontrolled metabolic diseases including diabetes, kidney, liver or cardiovascular diseases).

2.2.2 Exclusion criteria

- Pregnant women.
- Chronic use of non-steroidal anti-inflammatory drugs.
- Antibiotic use in the last 6 months.

2.3 Data collection

Data was collected using a standardized questionnaire (Appendix 1). Initially the data sheet was identifiable by the participant's civil personal number. Once the data sheet was checked for completeness the sheets were coded. Data collection was in 2 steps;

Step 1**:** Information regarding the subject's personal data, education status, medical history, health related behavior (smoking, alcohol drinking) as well as anthropometric measurements (BMI, WC, height and weight) was taken from the Nutrition Clinic assessment sheet. Measurement of BMI &WC were already recorded by nurses or family physicians in the Nutrition Clinics. The diagnosis of diabetes mellitus, hyperlipidaemia, and hypertension was made by the family physician according to the Bahrain National guidelines for the management of non- communicable diseases in primary care setting in the Kingdom of Bahrain.

Step 2: Periodontal examination of participants was performed by the author. An assistant recorded the findings in the data sheet. All visits were carried out in the same setting, and using the same measurements.

At the study visit the following was done:

- Informed consent was signed by the participant.
- Medical history was checked and updated.
- Record of participant oral hygiene practices.
- Update of participant smoking/ alcohol habits.

2.3.1 Examiner calibration

A pilot study was conducted before starting the data collection. Intra-examiner reliability was calculated using Kappa statistics prior to starting the data collection.

2.3.2 Assessment of periodontitis

The periodontal condition was assessed by using the WHO community periodontal index (CPI), it was selected as a tool for evaluating the periodontal health status because it is used widely in the survey of large populations.

The five CPI scores which was used to evaluate the periodontal health status as follows:

CPI 0 = Normal.

CPI 1 = gingival bleeding.

CPI 2 = calculus.

CPI 3 = shallow periodontal pocket of 3.5-5.5mm.

CPI 4 = deep periodontal pocket of > 5.5 mm.

The measurements were made using a WHO periodontal probe (E- probe) at six sites (MB, Mid B, DB, ML, Mid L, DL) per tooth. Ten teeth were selected for the periodontal examination, the two molars in each sextant and the upper right and lower left central incisors. If the index teeth were not present in a qualifying sextant, the adjacent remaining teeth in the sextant was examined. When a code 4 was gained, no further probing in that sextant was needed and a diagnosis of periodontitis was made. The presence or absence of periodontitis was dichotomized, and the subjects were categorized into two groups according to CPI 0, 1, 2 or CPI 3, 4 :

- Periodontitis absent = CPI 0 to CPI 2 (healthy periodontal tissues , gingivitis, and or plaque retentive factors)
- Periodontitis present = CPI 3 or CPI 4 (probing depth >3.5mm)

The individual CPI score per subject was recorded. Periodontitis was considered severe if the individual CPI score was 4. Mild-moderate if the individual CPI score was 3.

The number of sextants with periodontitis (CPI 3 or CPI 4) was also recorded per subject. Periodontitis was considered mild if the number of sextants with CPI 3 & 4 was 1 or 2, moderate if the number of sextants with CPI 3 & 4 was 3-4, and severe if number of sextants with CPI 3 & 4 was 5 or 6.

2.3.3 Assessment of obesity:

The clinical assessment of Body Mass Index (BMI) calculation, Waist Circumference Measurement (WC), Weight and Height was measured using standard technique at the primary health care in Nutrition clinics by Nurses or Family physicians and they obtained BMI electronically using a special scale (seca gmbh & co.kg.Germany).

The WHO classification (2000) was used to define the degrees of overweight in the Nutrition clinics;

- Overweight if BMI ranges 25.0-29.9 (kg/m^2)
- Obese if BMI $\geq$ 30.0, also sub-divided into:
  - Class I (moderate obese) : BMI 30.0-34.9 (kg/m^2).
  - Class II (severe obese): BMI 35.0-39.9 (kg/m^2).
  - Class III (very severe obese): BMI > 40.0 (kg/m^2).

2.3.3.1 Waist Circumference Measurement (WC):

Two gender specific waist circumference categories were used to classify WC into high or normal. The cut-point of >94cm (>37 inches) for male and of >80cm (>32 inches) for female was selected as high WC in our study, because at these cut-points the risk categories for both genders are considered (increase risk/significantly increased risk), according to WHO 2008, as follows;

High WC

- Male, if WC is > 94cm (>37 inches).
- Female, if WC > 80cm (>32 inches).

Normal WC

- Male, if WC is ≤ 94 (≤ 37 inches).
- Female, if WC is ≤ 80cm (≤ 32 inches).

2.4 Statistical analysis

The collected data were entered in to computer spreadsheets and analyzed using Statistical Package for Social Sciences (SPSS, version 20, Chicago, SPSS Inc). Descriptive statistics were performed for the general description of the data. Chi-square and Exact Fisher test were performed to examine differences between categorical data and t-test was performed to compare continuous variable. ANOVA test were performed to analyze categorical data with a continuous variable. In order to determine which obesity measure is associated with the severity and extent of periodontal disease, cross-tabulation and Pearson Chi-Square analyses were performed. A stepwise regression model

were performed to explain periodontitis as a function of different variables. The level of statistical significance was set at 5%.

2.5 Ethical considerations

Informed consent was signed by the participants and participation in this study was completely voluntary (Appendix 2).Moreover confidentiality of Research Records was maintained, participant examination records was completely anonymous, and only the investigators had access to the research data. The confidentiality of individual information will be maintained in any publications or presentations regarding this study.

2.6 Institutional ethical clearance

This study was conducted in full conformance with the principles of the "Declaration of Helsinki", Good Clinical Practice (GCP), and within the Ethical Guidelines for Health Research/Ministry of health in Bahrain. Ethical approval was obtained from the Research and Ethics Committee, HBMCDM/MBRU in Dubai and Ministry of Health Medical Research Committee in Bahrain (Appendix 3).

CHAPTER THREE

2. RESULTS

A total of 372 obese and overweight participants aged between 18 and 70 years with a mean age of 42.9 years (SD) (±11) participated in the study. Table 4 shows the age range and gender distribution of obese participants. Most of the participants 135 (36.3%) were aged between 40–50 years, and females predominated in the sample at 262 (70.4%). There was no significant difference in age between males and females (t=1.19, p=0.234)

Table 4: Number of participants by age and gender.

Age groups(years)	***N (%)***	
18-30	57 (15.3%)	
31-39	83 (22.3%)	
40-50	135 (36.3%)	
≥51	97 (26.1%)	
Mean age (SD) 42.9(±11)	372 (100%)	
Gender	***N (%)***	**Mean age(years)**
Male	110 (29.6%)	43.96 (±10.5)
Female	262 (70.4%)	42.46 (±11.24)
Total	372 (100%)	42.9 (±11)

Table 5 shows the prevalence of various demographic and behavioral factors in the obese participants. 338(90.9 %) of the participants were married. 303(81.44 %) of the participants had at least secondary education and 186(50%) were employed. Smoking and alcohol intake were found to be uncommon characteristics of the sample (31(8.3%), and 0.0% respectively).Most of the participants 352(94.62 %) brushed their teeth more than once daily.

Table 5: Prevalence of demographic and behavioral factors in the obese participants.

Marital status	***N (%)***
Single	34 (9.1%)
Officially Married*	338 (90.9%)
Occupation	
Employed	186 (50.0%)
Unemployed*	58 (15.6%)
House wife	128 (34.4%)
Education status	
Intermediate &less*	69 (18.5%)
Secondary	147 (39.5%)
University	156 (41.9%)
Smoking	
No	341 (91.7%)
Yes	31 (8.3%)
Alcohol	
Yes	0 (00.0%)
No	372 (100%)
Tooth brushing frequency	
‹ 1 time/day	20 (5.4%)
≥ 1/day	352 (94.6%)

Officially married:** Married, Divorced, WidowedUnemployed:** Student, Retired, Unemployed***Intermediate &less:** illiterate, primary, intermediate

The various medical conditions in the participants are shown in table 6. Around 261 (70%) of the participants had at least one medical problem. Hypertension was the most common medical problem 87(23.4%) noted in this sample, followed by diabetes mellitus 60(16.1%). Many participants had more than one medical problem, hence the number are not mutually exclusive. The mean number of teeth of the obese participants was 26.28 (±2.6) as shown in table 7 according to different categories of BMI.

Table 6: Prevalence of medical conditions secondary to obesity.

Medical conditions	***N%***
Hypertension	87 (23.4%)
Heart disease	12 (3.2%)
Diabetes Mellitus	60 (16.1%)
Hyperlipidaemia	48 (12.9%)
Endocrine system	23 (6.2%)
Others	31 (8.3%)
Total	261 (70%)

Table 7: Mean No. of teeth according to different categories of BMI

Categories of BMI	***Mean No. of teeth(SD)***
Overweight	26.45 (±2.8)
Obese I	26.3 (±2.5)
Obese II	26.48 (±2.1)
Obese III	25.94 (±3)
Total	26.28 (±2.6)

Table 8 shows the number of obese participants grouped by different categories of BMI and gender specific WC categories (WHO guideline). The majority of the participants were females in the obese class I category at 141(37.9%). According to gender specific WC categories, more of females had the highest WC N =258(69.4%) compared to male N =105 . However, mean WC was significantly greater in males at 114cm (±15.6) compared to females 109.5cm (±12.5) ($p<0.001$).

Table 8: Number of obese participants grouped by different categories of BMI and gender specific WC categories (WHO guideline).

Categories of BMI	***Male*** ***N (%)***	***Female*** ***N (%)***	***Total*** ***N (%)***
Overweight	16 (14.5%)	28 (10.7%)	44 (11.8%)
Obese I	49 (44.5%)	92 (35.1%)	141 (37.9%)
Obese II	22 (20.0%)	77 (29.4%)	99 (26.6%)
Obese III	23 (20.9%)	65 (24.8%)	88 (23.7%)
Total	110 (100%)	262 (100%)	372 (100%)
Gender WC categories	***N (%)***		
Male			
WC ≤ 94cm	20 (5.4%)		
WC >94cm	105 (24.2%)		
Female			
WC ≤ 80cm	4 (1.1%)		
WC >80cm	258 (69.4%)		
Total	372 (100%)		

Gender	***Mean WC(SD)***
Male	114.945 (±15.6)
Female	109.523 (±12.5)
Overall	111.126 (±13.7)

$t=3.538$, $p<0.001$

Table 9 shows the prevalence of periodontitis was very high at 361(97%) and out of this, 223(59.9%) had CPI score 3(mild- moderate periodontitis) and 138(37.1%) had CPI score 4 (severe periodontitis).

Table 9: Prevalence of periodontitis among obese participants by CPI score.

	Periodontitis absent N%	Periodontitis present N%		Total
	CPI=0,1,2	CPI=3	CPI=4	
	11 (3.0%)	223 (59.9)%	138 (37.1)	
Total	11 (3.0%)	361 (97.0%)		372 (100%)

CPI score 2= gingivitis, CPI score 3= mild-moderate periodontitis, CPI score 4= severe periodontitis

Table 10 shows the prevalence and the severity of periodontitis according to socio-demographic factors, habits, oral and general health status. Cross-tabulation and Chi-Square tests were sequentially carried out on all the factors and significance determined. Periodontitis (CPI 3& 4) was most prevalent in the 40–50 years age group at 36.3%. Significant severity was found if the obese participants were aged more than 40 years (P=0.012). Significantly more females had periodontitis than males (P=0.001). The prevalence of hypertension was significantly greater in the CPI 4 group compared to CPI 3 group ($P< 0.05$), but none of the other medical conditions were significantly different.

Table 10: Prevalence of periodontitis according to socio-demographic factors, habits, and oral and general health status of obese participants.

Variables	**Periodontitis**		**Total**	**P-value**
	CPI = 3	CPI = 4		
	N%	N%	N%	
Age(year)				
18-30	37(16.6%)	18(13.0%)	55(15.2%)	0.012*
31-39	57(25.6%)	22(15.9%)	79(21.9%)	
40-50	82(36.8%)	49(35.5%)	131(36.3%)	
≥51	47(21.1%)	49(35.5%)	96(26.6%)	
Total	223(100%)	138(100%)	361(100%)	
Gender				
Male	54(24.2%)	55(39.9%)	109 (30.2%)	0.001*
Female	169(75.8%)	83(60.1%)	252 (69.8%)	
Total	223(100%)	138(100%)	361(100%)	
Marital status				
Single	18(8.1%)	12(8.7%)	30(8.3%)	0.490
Official Married*	205(91.9%)	126(91.3%)	331(91.7%)	
Total	223(100%)	138(100%)	361(100%)	
Occupation				
Employed	107(48.0%)	74(53.6%)	181(50.1%)	0.375
Unemployed*	32(14.3%)	22(15.9%)	54(15.0%)	
House wife	84(37.7%)	42(30.4%)	126(34.9%)	
Total	223(100%)	138(100%)	361(100%)	
Education status				
Intermediate &less*	39(17.5%)	30(21.7%)	69(19.1%)	0.570
Secondary	88(39.5%)	54(39.1%)	142(39.3%)	
University	96(43.0%)	54(39.1%)	150(41.6%)	
Total	223(100%)	138(100%)	361(100%)	
Smoking	18(8.1%)	13(9.4%)	31(8.6%)	0.396
Tooth brushing frequency				
‹ 1 time/day	12(5.4%)	7(5.1%)	19(5.3%)	0.552
≥ 1/day	211(94.6%)	131(4.9%)	342(94.7%)	
Total	223(100%)	138(100%)	361(100%)	
Medical conditions				
Hypertension	46(20.6%)	40(29.0%)	86(23.8%)	0.047*
Heart disease	8(3.6%)	4(2.9%)	12(3.3%)	0.488
Diabetes Mellitus	32(14.3%)	27(19.6%)	59(16.3%)	0.124
Hyperlipidaemia	32(14.3%)	16(11.6%)	48(13.3%)	0.280
Endocrine system	14(6.3%)	8(5.8%)	22(6.1%)	0.523
Other	21(9.4%)	9(6.5%)	30(8.3%)	0.222

*$\mathbf{P} < 0.05$, **Official married:** Married, Divorced, Widowed***Unemployed:** Student, Retired, Unemployed***Intermediate &less:** illiterate, primary, intermediate, CPI score 3= mild-moderate periodontitis, CPI score 4= severe periodontitis

In order to determine which obesity measure is associated with the severity and extent of periodontal disease, cross-tabulation and Chi-Square analyses were performed and the results are shown in tables 11 and 12.

Table 11 shows the prevalence and severity of periodontitis if the CPI score was 3 or 4 per participant, according to BMI and gender specific WC categories using WHO guidelines. Participants with BMI obese I were the most prevalent at 138 (38.2%).

Table 11: Prevalence and severity of periodontitis according to categories of BMI and gender specific WC by using WHO guideline.

Variables	**Periodontitis**		**Total**	**p-value**
	CPI = 3	CPI = 4		
	N%	N%	N%	
Categories of BMI				
Overweight	28 (12.6%)	13 (9.4%)	41 (11.4%)	0.743
Obese I	86 (38.6%)	52 (37.7%)	138 (38.2)	
Obese II	59 (26.5%)	37 (26.8%)	96 (26.6%)	
Obese III	50 (22.4%)	36 (26.1%)	86 (23.8%)	
Total	223 (100%)	138 (100%)	361 (100%)	
Gender WC categories				
Male				0.037*
WC ≤ 94	14 (25.9%)	6 (10.9%)	20 (18.3%)	
WC >94	40 (74.1%)	49 (89.1%)	89 (81.7%)	
Total	54 (100%)	55 (100%)	109 (100%)	Spearman rho=+0.2
Female				0.600
WC ≤ 80	3 (1.8%)	1 (1.2%)	4 (1.6%)	
WC >80	166 (98.2%)	82 (98.8%)	248 (98.4%)	
Total	169 (100%)	83 (100%)	252 (100%)	

*P < 0.05. CPI score 3= mild-moderate periodontitis, CPI score 4= severe periodontitis

Analysis of the relationship between BMI and severity of periodontitis did not reveal any significant differences in the observed and expected frequencies for any of the categories of BMI (P=0.743). The gender WC cut-off of 94cm for male and 80cm for females did find that significantly more males (N=49) had a CPI code 4 and a WC >94cm (Spearman rho=+0.2). The WHO cut-off of ≤ 80cm and >80 for WC in females was not associated with the severity of periodontitis.

Table 12 shows the extent of periodontitis determined by the number of sextants with CPI 3 or 4. There were no significant differences in the distribution of participants by WHO BMI category and the extent of periodontitis, but the overall mean WC increased with the greater extent of periodontitis (ANOVA =2.767, P<0.05). It should be remembered that BMI categories do not differentiate by gender but WC does, further analysis to see if BMI was associated with periodontal disease was carried out. To simplify the analysis, BMI categories of overweight and obese I, were amalgamated into "moderate obesity" and BMI obese II and III were combined into "severe obesity" as shown in table 13. The observed frequencies in this 2x2 contingency table were not statistically significant (X^2=0.551, P =0.263).

A stepwise logistic regression model to explain periodontitis as a function of different variables is shown in table 14. The fitness of the model is significant (p-value < 0.001), and the severity of periodontitis was explained 11% by the overall WC and age adjusted for gender and blood pressure, the overall WC OR was 1.02 with 95% CI(1.00-1.04) and for age it was 1.05 with 95% CI(1.00-1.07).

Table 12: Extent of periodontitis by number of sextants with CPI 3 and 4 per participant according to categories of BMI and overall WC mean.

Categories of BMI	**Extent of Periodontitis(CPI 3 and 4) sextants N%**			**Total**	**P-value**
	1-2 sextants	3-4 sextants	5-6 sextants		
Overweight (25-29.9)	6(10.3%)	30(15.1%)	5(4.8%)	41(11.4%)	0.161
Obese class I (30-34.9)	27(46.6%)	74(37.2%)	37(35.6%)	138(38.2)	
Obese class II (35-39.9)	14(24.1%)	49(24.6%)	33(31.7%)	96(26.6%)	
Obese class III (>= 40)	11(19.0%)	46(23.1)	29(27.9%)	86(23.8%)	
Total	58(100%)	199(100%)	104(100%)	361(100%)	

Extent of Periodontitis(CPI 3 and 4) by number of affected sextants	Number of participants	Overall WC Mean(SD)	P-value
1-2 sextants	58	109.4(12.9)	0.042*
3-4 sextants	199	110(14.1)	
5-6 sextants	104	114.3(12.9)	
Total	361(100%)	111.2(13.7)	

*$\mathbf{ANOVA} = 2.74, \mathbf{P} < 0.05$

Table 13: Combined BMI categories and severity of periodontitis.

New BMI categories	**Periodontitis**		**Total**	**p-value**
	CPI = 3	CPI = 4		
	N%	N%	N%	
Moderate obesity (Overweight and Obese I)	114(51.1%)	65(47.1%)	179(49.6%)	0.263
Severe Obesity (Obese II and Obese III)	109(48.9%)	73(52.9%)	182(50.4%)	
Total	223(100%)	138(100%)	361(100%)	

X^2=0.551. CPI score 3= mild-moderate periodontitis, CPI score 4= severe periodontitis

Table 14: Logistic regression to explain periodontitis on function of different variables.

Variables	**B**	**S.E**	**Wald**	**df**	**Sig.**	**OR(95%CI)**
WC	0.021	0.009	5.334	1	0.021	1.021(1.003-1.039)
Gender	-0.433	0.257	2.843	1	0.092	0.648(0.392-1.073)
Age	0.047	0.012	14.546	1	0.000	1.048(1.023-1.074)
BP	0.039	0.290	0.018	1	0.893	1.040(0.589-1.834)
Constant*	-4.615	1.302	12.573	1	0.000	0.010

CHAPTER FOUR

4. DISCUSSION

The present study found that the prevalence of periodontitis in this sample of adults in the Kingdom of Bahrain was very high at 361(97%). Studies in the Jordanian, Malaysian and the USA populations, reported that the prevalence of chronic periodontitis among obese was 51.9%, 74% and 35% respectively (Khader et al. in 2008 [19], Khan et al. in 2015 [30], Al Zahrani et al. in 2003 [23]). The observed high prevalence of periodontitis in the Bahraini obese participants is mainly due to the shortcomings of CPI that was used to define periodontitis. Therefore, the possibility to overestimate the prevalence may exist. In this study, 37.1% were categorized as having severe periodontitis, as compared to a 10-15% worldwide prevalence of severe chronic periodontitis in the general adult population [65], or ranged from 5–15% of the adult global population [60].

Our results found that periodontitis was more prevalent in the 40–50 years age group, with a significance in obese participants aged over 40 years. However this result is in contrast to previous studies that have reported higher prevalence of periodontitis in younger obese adults. Al Zahrani et al. in 2003 [23] reported that BMI & WC were crudely associated with prevalence of periodontal disease in persons aged 18 to 34, but not in the middle-aged 35 to 59 and older 60 to 90 years in the United States. In addition, Ekuni et al. in 2014 [29] reported that BMI was associated with periodontitis in young obese Japanese university students (age range; 18-25years), and Khan et al. in 2015 [30] found that among an obese Malaysian population CP was more prevalent in the age group of 30-39 years.

Ageing is commonly associated with periodontal disease, and is one risk factor that is generally reported by all epidemiological studies [60], although this relationship is thought to be more related

to the cumulative periodontal breakdown over time than to an age-related, intrinsic deficiency that contributes to susceptibility to periodontal disease [86]. In general, as we age, adiposity and especially percentage body fat increase whereas lean mass and bone mineral density decrease [93]. Another major change is that fat mass tends to be preferentially distributed in the abdominal region, a phenomenon that has been reported in both sexes [93]. Ageing is associated with progressive changes in total and regional fat distribution that have negative health consequences [94]. Our study found that the severity of periodontitis was more pronounced in obese participants aged more than 40 years. Moreover, the logistic regression model found that the severity of periodontitis was marginally predicted by age, the adjusted OR was 1.02 with 95% CI: 1.00-1.04.

In this study periodontitis was more prevalent in obese female participants (69.8%). This finding was similar with the findings of the study conducted by Khan et al. in 2015 [30] on obese Malaysian participants. He reported that the prevalence of CP was much higher in females (61.3%) as compared to male participants. In addition Dalla Vecchia et al. in 2005 [24] found that obese females had an 80% higher chance of having periodontitis, than females of normal weight. Under obese conditions, females and males are both equal in terms of the obesity-induced burden of inflammation, however, in females, changes in hormonal levels of estrogen and progesterone during pre-menstrual and menstrual phases influence the periodontium and induce inflammatory cytokines in the periodontium [95].

The prevalence of periodontitis with smoking habits was not reflected (P=0.396) in our study probably, due to the small sample size of the obese participants who smoke (8.3%). It could be speculated that the low number of smokers in obese Bahraini participants could be due to high tobacco-quit rate among smokers seeking treatment at Quit Tobacco Clinics in MOH-Bahrain which is encouraging, and indicates that the clinics contributed to tobacco cessation in Bahrain [96].

Several epidemiological studies indicate that metabolic syndrome, obesity and hypertension increase the risk of periodontal disease [16],[22],[97]. The present study found that the prevalence of hypertension was significantly greater in the CPI 4 groups compare to CPI 3 groups, but none of the other medical conditions were significantly different ($P< 0.05$). This finding was reported in the Kitagawa et al. in 2017 study [34] that higher systolic blood pressure (≥140 mmHg) was a significant independent predictor of ≥ CPI score 3 (pocket depth ≥4mm) in Japanese individuals with BMI ≥30 kg/m², indicating an association with periodontitis.

The current study also showed no significant prevalence of periodontitis in association with the tooth brushing frequency ($P=0.579$). Our findings disagree with the finding of a systematic review and meta-analysis conducted by Zimmermann et al. in 2015 [98] which indicated that infrequent tooth brushing was associated with severe forms of periodontal disease, with a significant overall odds ratio estimate of 1.41 (95%CI: 1.25–1.58, $p < 0.0001$) for infrequent compared to frequent tooth brushing, although, the obese participants in their study suffered from moderate and severe periodontitis.

In the present study we found that the increase in the degree of obesity using the WHO guidelines had no significant association with the prevalence & severity of periodontitis in this obese sample of Bahraini participants ($p=0.743$). Moreover, the extent of periodontitis (The number of sextants with CPI 3 or 4) was found to have no significant association with the degree of BMI level ($p=0.161$). This finding was similar to a previous study [30] which reported that the increase in obesity level had no significant association with CP in an obese sample of Malaysians. In addition, a study of the Fourth Korean National Health and Examination Survey [27] found that obesity as defined by BMI does not seem to be correlated to periodontal disease. Recently, Chatzopoulos et al. in 2016 [99] investigated the relationship between BMI and CPITN score in a sample of 262

individuals aged 55years and over. They reported that there was no correlation between the two variables (p=0.499), with high CPITN scores not associated with higher levels of BMI. In disagreement with our results, several recent studies conducted on US, Jordanian and Japanese obese populations found that an increase in BMI was associated with periodontal disease. Al Zahrani et al. in 2003 [23] found that BMI > 30kg/m² was crudely associated with prevalence of periodontal disease in young adults. Khader et al. in 2008 [19] reported that periodontitis was more prevalent among Jordanian people with BMI ≥30 kg/ m². Ekuni et al. in 2014 [29] found that the risk of an increased CPI score was significantly associated with increase in BMI after adjusting for confounding factors in Japanese university students.

The current study found that there is a significant relationship between the extent of periodontitis measured by the number of sextants with CPI 3 or 4 and the overall mean of WC in obese participants (P=0.042). The highest mean of WC was in obese participants who had high number of sextants with CPI 3 or 4. Similar results were shown in many recent studies. Kangas et al. in 2017 [33] found that central adipose measures WC which seemed to be associated with periodontal pocketing in non-diabetic, never smoking Finland adults aged 30- 49 years old. Kim et al. in 2011 [27] also found a significant association between obesity and the prevalence of periodontitis, especially using the WC criteria established by WHO. In addition, Al Zahrani et al. in 2003 [23], and Khader et al. in 2008 [19] reported that WC was significantly associated with the prevalence of periodontal disease. Moreover, our finding suggested that in males both the Mean WC at 114cm (±15.6) ($p<0.001$) and WHO cutoff of WC >94cm ($p<0.05$) were significantly associated with the severity and extent of periodontitis; more males had a CPI 3-4 (Spearman rho=+0.2) compared to females WC mean at 109.5cm (±12.5) and WHO cut-off WC > 80cm.

Gorman et al. in 2012 [100] recorded similar finding, while they used a higher WC cutoff point (≤ or >120cm) and included WHR percentage as another measure of central obesity in adult white males. They found that central obesity (WC, WHR) was significantly associated with an increased risk of periodontal disease progression events in men. In addition, Han et al. in 2010 [25] found that visceral adiposity (WC; cut-off ≥90cm for male and ≥85cm for female, VFA) had a dose – effect relationship with the number of sextants with periodontitis (CPI 3-4). VFA was significantly associated with periodontitis (OR=1.47, 95%CL: 1.04-2.09). However, they reported that when considering the results of the subgroup analysis by age groups, the greatest association between VFA and periodontitis was found in males, aged 45-54(OR=3.30; 95%CI: 1.53-7.09).

Moreover, El-Sayed in 2010 [20] found that abdominal obesity in Egyptian males were significantly associated with periodontal disease.

The finding of the significant association between the severity of periodontitis and WC in males as compared to females, is perhaps partially due to men of all ages, race /ethnic groups, and geographic locations having significantly more periodontal disease than women, assessed by prevalence, extent, and severity, as well as by any parameter and case definition of periodontitis [5]. Additionally, men have more body fat in the abdominal (visceral) region than women [101].

In the stepwise logistic regression model the overall WC mean and age were found to be significantly associated with the severity of periodontitis ($p<0.001$), when adjusted for gender and blood pressure. The overall WC mean OR was 1.02 with 95% CI: 1.00-1.04 and the age OR was 1.05 with 95% CI: 1.00-1.07.

Our data suggest a weak positive correlation of WC with the severity of periodontitis irrespective of gender, whereas there was no correlation with BMI. Kim et al. in 2011 [27] reported similar findings to our study, in that there was no association between BMI and the prevalence of periodontitis, while a significant association was found using the WC criteria established by WHO (cutoff point > 90cm for males and > 85 for females). After adjusting for all variable the OR for periodontitis was 1.358 with 95% CI: 1.003-1.839. Khader et al. in 2008 [19] found a greater OR of periodontitis with high WC (>120cm for males and > 88 for females), OR=2.1 with 95% CI: 1.2-3.7. Moreover, Al Zahrani et al. in 2003 [23] reported an adjusted OR of 2.27 with 95% CI: 1.480-3.487 in young subjects with high WC. It seems that our data might be refined by using a higher WC cutoff point as the measurement of central obesity, and taking the subgroup analysis by age groups into account, as was previously mentioned by Han et al. in 2010 [25].

Thus, we can report that the overall WC mean is a predictor of the severity of periodontitis irrespective of gender, whereas BMI is not. In some periodontal studies, adiposity measures such as WC and WHR, that take visceral fat into account, have reported to be associated more strongly with different parameters of infectious periodontal diseases than the commonly used BMI. These finding suggested that adiposity measures that measure visceral fat accumulation accurately indicate the obesity-related health risk for periodontal health, at least more accurately than the commonly used BMI [33]. A shortcoming of BMI is that it does not take into account body composition nor distribution of fat, although visceral fat accumulation has harmful health effects; it increases the risk of Cardiovascular disease more than does subcutaneous fat, and increases the risk of cardiovascular diseases regardless of BMI [38].

4.1 Study limitations and strengths

There were certain limitations in the present study:

(i) Cross sectional study design did not allow us to infer relationships. This could be better done with a longitudinal cohort study design.

(ii) The subjects in our study were a convenience sample. Participation bias could have occurred.

(iii) The absence of a non-obese group, limits our ability to assess any association between obesity and periodontal disease.

(iv) The limited number of smokers and other health related problems such as diabetes mellitus, limit our ability for evaluating their risk on periodontal health.

(v) Although the CPI is an easy way for identifying periodontitis, it has several shortcomings [61]. A CPI 3 or 4 is very unlikely to be associated with destructive disease among young subjects, and older subjects are likely to have gingival recession and shallow pockets. Therefore, the possibility of an underestimation or overestimate the prevalence of periodontitis using CPI exists. A misclassification bias in using CPI could affect both the magnitude and the direction of the observed prevalence of this study.

Nevertheless, we could find several strengths and advantages of this study:

(i) The clinical assessment of BMI Calculation, WC, Weight and Height were measured at the primary health care in Nutrition clinics by Nurses or Family physicians.

(ii) This study confirmed that WC which is easier to measure and calculate than BMI, can effectively being used as a predictor of the extant of periodontal disease.

CHAPTER FIVE

5. CONCLUSION

This study reported a high prevalence of periodontitis in obese adults in the Kingdom of Bahrain. In this particular sample of Bahrainis, BMI was not correlated with periodontitis but WC in males had a weak positive correlation but not in females. Furthermore, the extent of periodontitis was significantly greater in subjects with a greater WC compared to subjects with lower WC.

5.1 Recommendation

- This study is a reflective of a selected obese Bahraini population and could highlight to the public, the effect of obesity on periodontal health of the adult population.
- Implementation of a program to combat periodontal disease among obese/overweight Bahrainis and to increase their awareness on the importance of good oral hygiene.
- Introducing periodontal health screening as a routine part of the Nutrition clinic program in MOH/Bahrain.
- By taking a preventive approach to management, it is anticipated that a positive cost to benefit outcome can be achieved.

5.1 Recommendation for further studies

- Study with larger sample size including normal weight participants as a control group to investigate the association between obesity and periodontal disease.
- Collecting data on waist hip ratio and visceral fat area to continue and examine multiple obesity measurements and determine the most suitable indicator of obesity in relation to periodontitis.

CHAPTER SIX

6. BIBLIOGRAPHY

1. World Health Organization. Overweight and Obesity. Fact sheet. Updated October. 2017.

2. Ng M, Fleming T, Robinson M, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: A systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2014; 384: 766-781.

3. World Health Organization. Burden of oral disease.2004.Available at; https://www.who.int/oral_health/disease_burden/global/en/index.html

4. Van Dyke T, Van Winkelhoff A. Infection and inflammatory mechanisms. J Clin Periodontol. 2013; 40: S1-S7.

5. Genco R, Borgnakee W. Risk factors for periodontal disease. Periodontol 2000. 2013; 62: 59-94.

6. Suvan J, D'Aiuto F, Moles D, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults. A systematic review. Obes Rev. 2011; 12: e381-e404.

7. Chaffee B & Westonn S. Association between chronic periodontal disease and obesity; a systematic review and meta-analysis. J Periodontol. 2010; 81: 1708-1724.

8. Suvan J, Petrie A, Nibali L, Darbar U, Rakmanee T, Donos N, D'Aiuto F. Association between overweight/obesity and increased risk of periodontitis. J Clin Periodontol. 2015; 42: 733-739.

9. Nascimento G, Leitre F, Do L, Peres K, Correa M, Demarco F, Peres M. Is weight gain associated with the incidence of periodontitis? A systematic review and meta-analysis. J Clin Periodontol. 2015; 42: 495-505.

10. Genco R, Grossi S, Ho A, Nishimura F, Murayama Y. A proposed model linking inflammation to obesity, diabetes and periodontal infections. J Periodontol. 2005; 76: 2075-2084.

11. Williams R. Periodontal disease. N Engl J Med. 1990; 322: 373-382.

12. Beck J, Garacia R, Heiss G, Vokonas P, Offenbacher S. Periodontal disease and cardiovascular disease. J Periodontol. 1996; 67: 1123-1137.

13. Bruun J, Verdich C, Toubro S, Astrup A, Rihelsen B. Association between measures of insulin sensitivity and circulating levels of interleukin-8,interleukin-6 and tumor necrosis factors-alpha. effect of weight loss in obese men. Eur J Endocrinol. 2003; 148: 535-542.

14. Esposito K, Giugliano G, Scuderi N, Giugliano D. Role of adipokines in the obesity-inflammation relationship: the effect of fat removal. Plast Reconstr Surg. 2006; 118: 1048-1057.

15. Zeyda M, Stulnig T. Adipose tissue macrophages. Immunol Lett. 2007; 112: 61-67.

16. Saito T, Shimazaki Y, Kiyohara Y, Kato I, Kubo M, Iida M, Yamashita Y. Relationship between obesity, glucose tolerance, and periodontal disease in Japanese women: the Hisayama study. J Periodontal Res. 2005; 40: 346-353.

17. Krejci C, Bissada N. Obesity and periodontitis: a link. General Dentistry. 2013; 61: 60-63.

18. World Health Organization. Obesity: Preventing and Managing the Global Epidemic. 894 WHO Technical Report Series; Geneva, Switzerland: 1988.

19. Khader Y, Bawadi H, Haroun T, Alomari M, Tayyem R. The association between periodontal disease and obesity among young adult in Jordan. J Clin Periodontol. 2009; 36: 18-24.

20. El Sayed Amin H. Relationship between overall and abdominal obesity and periodontal disease among young adults. East Medit H J. 2010; 16: 429-433.

21. Awad H, Rahman B, Hasan H, Ali H. The Relationship between body mass index and periodontitis in Arab patients with type 2 diabetes mellitus. Oman Med J. 2015; 30: 36-41.

22. Saito T, Shimazaki Y, Skamoto M. Obesity and periodontitis. N Engl J Med. 1998; 339: 482-483.

23. Al Zahrani M, Bissada N, Borawskit E. Obesity and periodontal disease in young, middle-aged, and older adults. J Periodontol. 2003; 74: 610-615.

24. Dalla Vecchia C, Susin C, Rosing C, Oppermann R, Albandar J. Overweight and obesity as risk indicators for periodontitis in adults. J Periodontol. 2005; 76:1721-1728.

25. Han D, Lim S, Sun B, Paek D, Kim H. Visceral fat area-defined obesity and periodontitis among Koreans. J Clin Periodontol. 2010; 37: 172- 179.

26. Saxlin T, Ylöstalo P, Suominen-Taipale L, Männistö S, Knuuttila M. Association between periodontal infection and obesity: results of the Health 2000 Survey. J Clin Periodontol. 2011; 38: 236-242.

27. Kim E, Jin B, Bae K. Periodontitis and obesity: A study of the fourth Korean National Health and Nutrition Examination Survey. J Periodontol .2011; 82: 533-542.

28. Nascimento G, Seeig L, Vargas-Ferreira F, Correa F, Leite F, Demarco F. Are obesity and obesity and overweight associated with gingivitis occurrence in Brazilian schoolchildren?. J Clin Periodontol. 2013; 40: 1072-1078.

29. Ekuni D, Mizutani S, Kojima A, Tomofuji T, Irie K, Azuma T, Yoneda T, Furuta M, Eshima N, Iwasaki Y, Morita M. Relationship between increase in BMI and changes in periodontal status: a prospective cohort study. J Clin Periodontol. 2014; 41: 772-778.

30. Khan S, Saub R, Vaithilingam R, Safii S, Vethakkan S, Baharuddin N. Prevalence of chronic periodontitis in an obese population: a preliminary study. BMC Oral Health. 2015; 15: 114.

31. Gaio E**,** Haas A, Rösing C. Effect of obesity on periodontal attachment loss progression: a 5-year population-based prospective study. J Clin Periodontol. 2016; 43: 557-565.

32. Peruchi C, Poli-Frederico R, Cardelli A, Fracasso M, Bispo C, Neves-Souza R, Cardoso J , Maciel S. Association between oral health status and central obesity among Brazilian independent-living elderly. Braz Oral Res. 2016; 30: e116.

33. Kanga S, Timonen P, Knuuttila M, Jula A, Ylöstalo P, Syrjälä A. Waist circumference and waist-to-height ratio are associated with periodontal pocketing- results of Health 2000 survey. BMC Oral Health. 2017; 17: 48. DOI 10.1186/s12903-017-0336-y.

34. Kitagawa M, Kurahashi T, Matsukbo T. Relationship between general health, lifestyle, oral health, and periodontal disease in adults: A large cross-sectional study in Japan. Bull Tokyo Dent Coll. 2017; 58: 1-8.

35. Musaiger A. Overweight and obesity in Eastern Mediterranean Region: Prevalence and possible causes. J Obesity. 2011; 2011: 1-17.

36. Kilpi F, Webber F, Musaigner A, Selmi A, Marsh T, Rtveladze K, McPherson K, Brown M. Alarming predictions for obesity and non-communicable diseases in the Middle East . Public Health Nutrition. 2014; 17: 1078-1086.

37. National Non-communicable Diseases Risk Factors Survey 2007 Booklet. Ministry of Health, Kingdom of Bahrain. 2010.

38. World Health Organization. Waist circumference and waist–hip ratio. Report of a WHO expert consultation, Geneva. 2008.

39. Evidence-based Guidelines for Obesity Management in Primary Health Care. Ministry of Health. Kingdom of Bahrain. 2008.

40. Gharib N, Alsalehi S, AlAmer M. Nutrition Clinics. Management and prevention booklet. Directorate of Public Health. Nutrition Section. Ministry of Health. Kingdom of Bahrain. 2012.

41. American Academy of Periodontology. Fact sheet 2015. Available at; https://www.perio.org/newsroom/periodontal-disease-fact-sheet

42. Socransky S. Microbiology of periodontal disease-Present status consideration. J Periodontol. 1977; 48: 497-504.

43. Theilade E. The non-specific theory in microbial etiology of inflammatory periodontal disease. J Clin Periodontol. 1986; 13: 905-911.

44. Loesche W. The specific plaque hypotheses and the antimicrobial treatment of periodontal disease. Dent Update. 1992; 19: 70-74.

45. Slot J. Bacterial specificity in adult periodontitis. a summary of recent work. J Clin Periodontol. 1986; 13: 912-917.

46. Socransky S, Haffajee A. The bacterial etiology of destructive periodontal disease: Current concepts. J Periodontol. 1992; 63: 322-331.

47. Lindhe J, Lang N, Karring T. Clinical periodontology and implant dentistry, 5th edn. Oxford: Wiley-Blackwell. 2008.

48. Borrell L, Papapanou P. Analytical epidemiology of periodontitis. J Clin Periodontol. 2005; 32: 132-158.

49. Tonetti M. Cigarette smoking and periodontal diseases: etiology and management of disease. Ann Periodontol. 1998; 3: 88-101.

50. Martinez-Maestre M, Gonzalez-Cejudo C, Machuca G, Torrejon R, Castelo-Branco C. Periodontitis and osteoporosis: a systematic review. Climacteric. 2010; 13: 523-529.

51. Nishida M, Grossi S, Dunford R, Ho A, Trevisan M, Genco R. Calcium and the risk for periodontal disease. J Periodontol. 2000; 71: 1057-1066.

52. Peruzzo D, Benatti B, Ambrosano G, Nogueira G, Sallum E, Casati-Filho M, Nociti F. A systematic review of stress and psychological factors as possible risk factors for periodontal disease. J Periodontol. 2007; 78: 1491-1504.

53. McCracken G. Positive relationship between stress and periodontal disease. Evid Based Dent. 2009; 10: 42.

54. Michalowicz B, Diehl S, Gunsolley J, Sparks B, Brooks C, Koertge T, Califano J, Burmeister J, Schenkein H. Evidence of a substantial genetic basis for risk of adult periodontitis. J Periodontol. 2000; 71: 1699-1707.

55. Meng H, Ren X, Tian Y, Feng X, Xu L, Zhang L, Lu R, Shi D, Chen Z. Genetic study of families affected with aggressive periodontitis. Periodontol 2000. 2011; 56: 87-101.

56. Laine M, Loos B, Crielaard W. Gene polymorphisms in chronic periodontitis. Int J Dent. 2010; 324719.

57. Lilienfeld D. Definition of epidemiology. Am J Epidemiol. 1978; 107: 87-90.

58. Papapanuo P. Periodontal disease: epidemiology. Ann Periodontol. 1996; 1: 1-36.

59. Garcia R, Dietrich T. Introduction to periodontal epidemiology. Periodontol 2000. 2012; 58: 7-9.

60. Dye B. Global periodontal disease epidemiology. J Periodontol. 2012; 58: 10-25.

61. Kingman A, Albandar J. Methodological aspects of epidemiological studies of periodontal diseases. Periodontol 2000. 2002; 29: 11-30.

62. Ainamo J, Barmes D, Beagrie G. Development of the World Health Organization (WHO) Community Periodontal Index of Treatment needs (CPTIN). Int Dent J. 1982; 32: 281-291.

63. Petersen P, Ogawa H. The global burden of periodontal disease: towards integration with chronic disease prevention and control. Periodontol 2000. 2012; 60: 15-39.

64. World Health Organization. Oral health surveys basic methods, 4th edition. Geneva. 1997.

65. World Health Organization. The WHO global oral health data bank. Geneva. 2007.

66. Petersen P, Ogawa H. Strengthening the prevention of periodontal disease: the WHO approach. J Periodontol. 2005; 76: 2187-2193.

67. James W. WHO recognition of the global obesity epidemic. Int J Obesity. 2008; 32: S120-S126.

68. Bjorntorp P. Fat cell distribution and metabolism. Ann N Y Acad Sci. 1987; 499: 66-72.

69. Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Bjorntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. Br Med J. 1984; 288: 1401-1404.

70. Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjöström L. Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow up of participants in the population study of women in Gothenburg, Sweden. Br Med J. 1984; 289: 1257-1261.

71. World Health Organization. Obesity: Preventing and Management the Global Epidemic, Report of a WHO Consultation, Geneva. 2000.

72. Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi M, Commerford P, Lang C, Rumboldt Z, Onen C, Lisheng L, Tanomsup S, Jr P, Razak F, Sharma A, Anand S. Obesity and the risk of myocardial infarction in 27000 participants from 52 countries: a case-control study. Lancet. 2005; 366: 1640-1649.

73. Musaiger A, Hassan A , Obeid O. The Paradox of Nutrition-Related Diseases in the Arab Countries: The Need for Action. Int J Environ Res Public Health. 2011; 8: 3637-3671.

74. Ng S, Zaghloul S, Ali H, Harrison A, Popkin B. The prevalence and trends of overweight, obesity and nutrition-related non-communicable diseases in the Arabian Gulf States. Obes Rev. 2011; 12: 1-13.

75. Musaiger A. Overweight and Obesity in the Arab Countries: The Need for Action. Technical Report. Bahrain Center for Studies and Research. Bahrain. 2007.

76. Perlstein M, Bissada N. Influence of obesity and hypertension on the severity of periodontitis in rats. Oral Surg Oral Med Oral Pathol. 1977; 43: 707-719.

77. Verzeletti G, Gaio E, Linhare D, Rösing C. Effect of obesity on alveolar bone loss in experimental periodontitis in Wistar rats. J Appl Oral Sci. 2012; 20: 218-221.

78. Kongstad J, Hvidtfeldt U, Grønbæk M, Stoltze K, Holmstrup P. The Relationship Between Body Mass Index and Periodontitis in the Copenhagen City Heart Study. J Periodontol. 2009; 80: 1246-1253.

79. De Castilhos E, Horta B, Gigante D, Demarco F, Peres K, Peres M. Association between obesity and periodontal disease in young adults: a population-based birth cohort. J Periodontol. 2012; 39: 717-724.

80. Garcia R, Dietrich T. Association between periodontal disease and systemic disease: evaluating the strength of the evidence. J Periodontol. 2005; 76: 2175-2184.

81. Loos B. Systemic markers of inflammation in periodontitis. J Periodontol. 2005; 76: 2106-2115.

82. Beck J, Offenbacher S. Systemic effect of periodontitis: epidemiology of periodontal disease and cardiovascular disease. J Periodontol. 2005; 76: 2089-2100.

83. Pichon N, Heng N, Bernimoulin J, Kleber B, Willich S, Pischon T. Obesity, inflammation and periodontal disease. J Dent Rest. 2007; 86: 400-409.

84. Thanakun S, Izumi Y. Effect of periodontitis on adiponectin, C-reactive protein, and immunoglobulin G against porphyromonas gingivalis in Thai people with overweight or obese status. J Clin Periodontol. 2016; 87: 566-576.

85. Kanety H, Feinstein R, Papa M, Hemi R, Karasik A. Tumor necrosis factor alpha-induced phosphorylation of insulin receptor substrate-1(IRS-1). Possible mechanism for suppression of insulin-stimulated tyrosine phosphorylation of IRS-1. J Biol Chem. 1995; 270: 23780-23784.

86. Genco R. Current View of Risk Factors for Periodontal Diseases. J Periodontol. 1996; 67: 1041-1049.

87. Saito T, Shimazaki Y. Metabolic disorders related to obesity and periodontal disease. Periodontol 2000. 2007; 43: 254-266.

88. Boesing F, Patino J, Da S, Moreira E. The interface between obesity and periodontitis with emphasis on oxidative stress and inflammatory response. Obes Rev. 2009; 1: 290-297.

89. Kalea A, Hoteit R, Suvn J, Lovering R, Palmen J, Cooper J, Khodiyar V, Harrington Z, Humphries S, D'Aiuto F. Upregulation of gingival tissue miR-200b in obese periodontitis subjects. J Dent Res. 2015; 94: 595-695.

90. Socransky S, Haffajee A. Relation of body mass index, periodontitis and Tannerella Forsythia. J Clin Periodontol. 2009; 36: 89-99.

91. Rangé H, Léger T, Huchon C, Ciangura C, Diallo D, Poitou C, Meilhac O, Bouchard P, Chaussain C. Salivary proteome modifications associated with periodontitis in obese patients. J Clin Periodontol. 2012; 39: 799-806.

92. Al Abdaly M. Periodontal pocket depth and salivary calcium and phosphorous levels among obese Yemenis patients. Inter J of Curr Med And Pharma Research. 2017; 3: 1684-1688.

93. Enzi G, Gasparo M, Biondetti P, Fiore D, Semisa M, Zurlo F. Subcutaneous and visceral fat distribution according to sex, age, and overweight, evaluated by computed tomography. Am J Clin Nutr. 1986; 44: 739-746.

94. Kuk J, Saunders T, Davidson L, Ross R. Age- related changes in total and regional fat distribution. Ageing Research Reviews. 2009; 8: 339-348.

95. Khosravisamani M, Maliji G, Seyfi S, Azadmehr A, Abd Nikfarjam B, Madadi S, Jafari S. Effect of the menstrual cycle on inflammatory cytokines in the periodontium. J Periodontal Res. 2014; 49: 770-776.

96. Hamadeh R, Ahmed J, AlKawari M, Bucheeri S. Quit tobacco clinics in Bahrain: smoking cessation rates and patient satisfaction. Tob Indu Dis. 2017; 15: 1-7.

97. Shimazaki Y, Saito T, Yonemoto K, Kiyohara Y, Iida M, Yamashita Y. Relationship of metabolic syndrome to periodontal disease in Japanese women: the Hisayama study. J Dent Res. 2007; 86: 271-275.

98. Zimmermann H, Zimmermann N, Hagenfeld D, Veile A, Kim T, Becher H. Is frequency of tooth brushing a risk factor for periodontitis? A systematic review and meta-analysis. Community Dent Oral. 2015; 43: 116-27.

99. Chatzopoulos G, Tsalikis L. Periodontal treatment needs and systemic disease in an older population in Greece. J clin Exp Dent. 2016; 8: e32-7.

100. Gorman A, Kaye E, Apovian C, Fung T, Nunn M, Garcia R. Overweight and obesity predict time to periodontal disease progression in men. J Clin Peridontol. 2012; 39: 107-114.

101. Lemieux S, Prud'homme D, Bouchard C, Tremblay A, Després J. Sex differences in the relation of visceral adipose tissue accumulation to total body fatness. Am J Clin Nutr. 1993; 58: 463-467.

CHAPTER SEVEN

APPENDIX 1

Study Assessment sheet

Sheet No: ------

CPR No----------

Date: ---------- **Telephone**:----------

Gender: M ☐ F ☐ **Age:** ----------

Marital status: ☐ Single ☐ Married ☐ Divorced/Widowed
For female: ☐ Pregnant ☐ Not pregnant

Occupation: ☐ Employed (sedentary/light manual/heavy manual)
☐ Unemployed
☐ Housewife (sedentary/light manual/heavy manual)

Education status: ☐ Illiterate ☐ Primary/Intermediate/Secondary ☐ University

Medical History: ☐ Hypertension ☐ Coronary artery disease ☐ Stroke
☐ Diabetes ☐ Hyperlipidaemia ☐ Premature Cardiovascular Disease
☐ Endocrine disease. Type----------

Habit of smoking & alcohol drinking:

Smoke: ☐ Yes ☐ NO

Alcohol drinker: ☐ Yes ☐ NO

Anthropometrics:

Height----------cm Weight----------Kg Body Mass Index----------kg/m2

Waist Circumference----------cm

Degree of Overweight BMI SCORE:

☐ Overweight (25.0-29.9)

☐ Obese class I (30.0-34.9)

☐ Obese class II (35.0-39.9)

☐ Obese class III (≥ 40.0)

Periodontal assessment:

- Frequency of daily teeth brushing: ☐ ‹ 1 ☐ ≥ 1
- No. of teeth: ----------
- CPI measurement for each sextant:

UR 6,7	UR 1	UL 6,7
LR 6,7	LL 1	LL6,7

- CPI score for the individual: ----------
- Periodontitis ☐ Yes ☐ No.
- Number of sextants with periodontitis (CPI 3or CPI 4) ----------.

APPENDIX 2

إقرار بالموافقة للمشاركة في البحث

اسم الباحث: د. لينا عبدالله الصالحي

عنوان الدراسة : قياس مدى انتشار امراض اللثة في فئة البالغين ممن يعانون من السمنة في مملكة البحرين.
هدف البحث: يهدف البحث بشكل أساسي بدراسة صحة اللثة في فئة البالغين ممن يعانون من السمنة ، حيث تعتبر السمنة في ازدياد بشكل كبير في هذه الفئة في مملكة البحرين. ويسعى هذا البحث لتحديد حجم انتشار أمراض اللثة لديهم.

عملية الاختيار: جميع الاشخاص البالغيين الذين يتابعون مع عيادات السمنة التابعة لوزارة الصحة، هم مدعوون للمشاركة في البحث في حالة تم اختيارهم. المشاركة في هذا البحث طوعية وليست إجبارية ، أي كان اختيارك ، لن يكون لذلك تأثير في الخدمات المقدمة لك في المركز الصحي.

خطوات البحث:
1. سيتم اختيارك من قبل عيادات السمنة وذلك حسب المعايير المطلوبة للبحث.
2. بعد موافقتك على البحث سيتم جمع بعض المعلومات المتعلقة بوزنك وطولك من ملفك التابع لعيادة السمنة.
3. يقوم الباحث (د. لينا عبدالله) بفحص بسيط للثة لمدة 5-10 دقائق تقريباً.

الفوائد/ والأعراض الجانبية: هذه خدمات عادية تقدم لجميع المرضى، لا يوجد خطر معين على صحتك نتيجة هذا البحث. فائدة هذا البحث تكمن في قياس انتشار أو غياب أمراض اللثة في البالغيين ممن يعانون من السمنة ، وهذا سيوفر معلومات بقدر من الأهمية ، قد يستفاد منها لإنشاء برنامج وقائي.

الخصوصية: المعلومات التي يتم جمعها من هذا البحث سرية وخاصة، ستحفظ في مكان لا يستطيع أي أحد الوصول إليه سوى فريق البحث، سيكون لجميع المعلومات الخاصة بك رقم معين بدل اسمك أو رقمك الشخصي، فريق البحث فقط سيمكنه التعرف على رقمك، لن يتم مشاركة أي أحد بهذه المعلومات.

كيفية التواصل: إذا كان لديك أي سؤال ، حالياً أو مستقبلاً، أو حتى بعد الانتهاء من الدراسة ، بإمكانك التواصل مع الباحث بطرق التواصل التالية:
الاسم : لينا عبدالله الصالحي
العنوان: وزارة الصحة – قسم البرنامج التدريبي لأطباء الأسنان- مملكة البحرين ص ب 12
هاتف: 00973-39691361
البريد الالكتروني: leena.alsalihi@mbru.ac.ae

هذا البحث تمت الموافقة عليه من قبل لجنة البحوث في جامعة محمد بن راشد للطب والعلوم الصحية في دبي، ومن قبل فريق دعم البحوث في مكتب المراجعة الطبية في وزارة الصحة بمملكة البحرين. هذه اللجان من مهام عملها، التأكد من عدم وجود أي نوع من الضرر على المشاركين في البحث. إذا كنت ترغب بمزيد عن المعلومات عن اللجنة البحرينية، بإمكانك التواصل مع مكتب المراجعة الطبية في وزارة الصحة ، هاتف 00973-17286052، فاكس 00973-17286651، والبريد الإلكتروني . RTST@health.gov.bh

لقد قمت بقراءة المعلومات السابقة، أو قد تمت قراءتها لي. لقد كانت لدي الفرصة الكافية لتوجيه الأسئلة لفريق البحث، وقد تمت الإجابة عليها بصورة مناسبة. إني أقر بالموافقة الطوعية للمشاركة في هذا البحث.

اسم المشارك: ---------- توقيع المشارك: ----------

التاريخ : ----------

Informed Consent Form

Principal Researcher: Leena Alsalihi.

Title of project: The Prevalence of Periodontitis in obese adults in the Kingdom of Bahrain.

I am asking for your voluntary participation in my science fair project, please read the following information about the project. If you would like to participate please sign in the appropriate box below.

Purpose of the project: You are being asked to participate in a research study examining the periodontal/gums health of obese adults.

If you participate, some information concerning your weight and height will be taken from your medical records at the nutrition clinic. Moreover you will be asked to be screened to examine the status of your gums.

Time required for participation: about 5-10 minutes of your time.

Potential risks of study: No foreseeable risks are involved in this study.

Benefits: We expect the project to benefit you by gaining a better understanding of factors contributing to the development of gums diseases.

How confidentiality will be maintained:

Your examination records to this study will be completely anonymous, and only the investigators will have access to the research data. The confidentiality of your individual information will be maintained in any publications or presentations regarding this study.

If you have any questions about this study feel free to contact:

Dr. Leena Alsalihi .Phone no: 00973-39691361. E-mail: leena.alsalihi@mbru.ac.ae

Voluntary Participation:

Participation in this study is completely voluntary, if you decide not to participate there will not be any negative consequence. Please be aware that if you decide to participate, you may stop participation at any time.
By signing this form I am attesting that I have read and understand the information above and I freely give my consent to participate in this study.

Printed name of Research Participant: Date reviewed & signed----------
---------- Signature----------

APPENDIX 3

MBRU
جامعة محمد بن راشد للطب والعلوم الصحية
MOHAMMED BIN RASHID UNIVERSITY OF MEDICINE AND HEALTH SCIENCES

Date: 09/11/2016

Dear Dr Leena Alsalihi Periodontology Resident

Re: Your research protocol

Titled: The prevalence of periodontitis in obese adults in The Kingdom of Bahrain

Thank you for submitting your research protocol to the Research and Ethics committee of the Hamdan Bin Mohammed College of Dental Medicine, MBRU.

It was considered at the meeting held on: 23/10/2016

It was agreed to approve the protocol. Please make sure you see your research supervisor regularly during the project in order to maintain close collaboration and support. The committee would like to remind you that it is a requirement of the programme that you complete a research dissertation, which comprises 15% of credits within the 3-year MSc programme.

With best wishes

Yours sincerely,

Prof A Milosevic

Chair, Research and Ethics Committee, HBMCDM

Prof. Alexander Milosevic
Program Director
Professor of Prosthodontics

KINGDOM OF BAHRAIN
MINISTRY OF HEALTH
Office of Asst. Undersecretary for Training & Planning

مملكة البحرين
وزارة الصحة
مكتب الوكيل المساعد للتدريب والتخطيط

No:MA/JE/647/2016
Date: 12/12/2016

To: Mrs. Leena Alsalihi

Subject: Letter of Approval for Research Proposal:

"The prevalence of periodontitis in obese adults in the Kingdom of Bahrain"

Dear Mrs. Leena,

Thank you for submitting your revised research proposal documents, which have been considered by members of the Research Technical Support Team (RTST) on 13 December 2016

We would like to inform you that the team found no major ethical issues or methodological problems that would hinder the conduct of this survey. We are thus pleased to approve the above application.

This approval is subject to the following conditions:

1. We expect that the study will begin within 6 months of the date of this approval.
2. Approval from an MOH Research Committee does not automatically imply that the researcher is granted access to data, medical records or biological samples from MOH healthcare facilities. Researchers must seek permission and follow procedures as dictated by the concerned departments after presenting them with a valid MOH approval letter.
3. Any significant change, which occurs in connection with this study and/or which may alter its ethical consideration, must be reported immediately to the RTST.
4. This approval is valid for up to **1 year** from the date of approval. If the study extends beyond this date, a progress report must be sent to the RTST to renew the approval.
5. The RTST must be informed when the research has been completed and a copy of the final research report must be submitted for our records.

We wish you all the best in this study.

Yours sincerely,

Dr. Mohammed Amin Al-Awadhi
Assistant Undersecretary for Training and Planning
Chairperson, Research Technical Support Team

CC: Team file