DENTAL CARIES AND PUFA INDEX IN CHILDREN AGE 11 – 14 YEARS IN UNGOGO LOCAL GOVERNMENT AREA, KANO STATE, NIGERIA

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ABSTRACT

Background: Oral diseases are still a major problem in most developing countries. Dental caries is one of the most common forms of oral disease frequently resulting in loss of the affected teeth. In Western communities active caries is seen predominantly in childhood, adolescence and early adulthood. Within Nigeria, there remain areas where few or no studies have been done on the oral health status. Despite the fact that dental caries can be prevented and managed, it is still a major health problem affecting mankind, in that its manifestations persist throughout life in spite of treatment. There are practically no geographic areas in the world whose inhabitants do not exhibit some evidence of dental caries. It affects persons of both genders in all races, all socioeconomic strata and every age group.

Objectives: To assess the prevalence and severity of dental caries and PUFA Index of school children (11 – 14years old); evaluate the oral health practices pattern of the study population and to provide useful data for the planning of oral health services in this region.

Design: Descriptive analytical study.

Setting: School children (11 – 14years old) in Ungogo Local Government, Kano state, Nigeria

Subjects/Methods: Questionnaires on demographic variables and oral hygiene practices were administered to a total of 294 randomly selected school children in the age groups 11 – 14 years and then examined by the researcher for dental caries (DMFT) and the PUFA index which records the presence of severely decayed teeth with visible pulpal involvement (P/p), ulceration caused by dislocated tooth fragments (U/u), fistula (F/f) and abscess (A/a). The four schools in the study were randomly selected from the 15 junior secondary schools in the rural and peri-urban locations in the setting.

Results: Caries was diagnosed in 254 (86.4%) of the 294 school children. The total mean DMFT for the 294 children was 2.42 ± 1.53. The mean DMFT for boys was 2.35 ± 1.33 and for girls it was 2.54 ± 0.86. There was a general increase in mean DMFT with age. Caries was most prevalent on first molars (46.7%) and it was also observed more in the mandibular molars (69.8%). The decayed component constituted the main part of the DMFT scores. The overall caries prevalence was 82% while the overall PUFA ratio of 61% and ninety-nine percent of these lesions were untreated

Conclusion: The respondents had a poor knowledge of oral health, oral diseases and their causes, though the majority had a positive attitude concerning oral health care and rarely visited dentist. However, the overall knowledge of foods related oral diseases was good. Within the limitations of the study, the overall caries prevalence in the study population was high. Notwithstanding, ages11, 12 and 13 constitute the ages at greater risk. PUFA defines the various clinical stages and their different associations with health conditions. Presenting data based on the PUFA index may provide health planners with relevant information, which is complementary to the DMFT. This was an opportunity to validate the new PUFA index under field conditions in school children (11 -14years) in a low-income population in Ungogo local government of Kano state, with a population suffering from a high burden of untreated caries. This study indicates that preventive oral health measures should be implemented on the younger age groups in order to control dental caries.

DECLARATION

I declare that the work presented here has been done by me Dr. Sani Balarabe Auwalu under Supervision. I also declare that the work has not been submitted in part or in full to any other college for award of a degree/fellowship or submitted for publication elsewhere. The contributions of the authors referred to are duly acknowledged in the text.

Dr. Sani Balarabe Auwalu 22/04/2011

ATTESTATION

We certify that this dissertation is an original and independent work carried out by Dr. Sani Balarabe Auwalu under our supervision

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CHAPTER 1

1.1. INTRODUCTION

Dental caries is one of the most common forms of oral disease frequently resulting in loss of the affected teeth. In Western communities active caries is seen predominantly in childhood, adolescence and early adulthood[1].

Dental caries is an irreversible microbial disease of the calcified tissues of the teeth, characterised by demineralisation of the inorganic portion and destruction of the organic substances of the tooth, which often leads to cavitations[2]. It is a complex and dynamic process where bacteria act on tooth surface in the presence of substrate over time which influences and initiates the progression of disease. Despite the fact that dental caries can be prevented and managed, it is still a major health problem affecting mankind, in that its manifestations persist throughout life in spite of treatment[1, 2].

There are practically no geographic areas in the world whose inhabitants do not exhibit some evidence of dental caries. It affects persons of both genders in all races, all socioeconomic strata and every age group. For example, a more isolated community faces greater difficulties in ensuring the availability of high-quality health services which in turn affects their carious status. Children living in rural communities have a less favourable oral health status, which affects their performance at school. According to the American National Rural Health Association, rural Americans are twice as likely to lose their teeth compared with urban residents either due to caries, periodontal diseases or other oral conditions. This is somewhat similar in African regions; rural communities form a large part of African territories, they represent about 90% of the

population and 95% of the land mass. The rural environment, geography and demography have an impact on the needs of rural dwellers. For example, the communities in rural northern Alberta had a very high caries index in conjunction with a high rate of treatment needed for other oral health problems[3].

In spite of these, there were subjects who never developed carious lesions, they were designated 'caries – free' and no satisfactory explanation was given[3]. Many investigators have studied various aspects of dental caries for more than a century. Despite this, many aspects of aetiology are still obscure, and efforts at prevention have been only partially successful. Its prevalence has greatly declined, but it's still the chief dental disease of children in terms of frequency and destructiveness of the dentition, in view of these, an attempt was made to understand caries experience in rural school children in Kano State, Nigeria.

1.2. DENTAL CARIES

The word caries is derived from the Latin word meaning 'rot' or 'decay'. Dental caries is a progressive destruction of enamel, dentine and cementum initiated by microbial activity on the tooth surface. Certain types of bacteria preferentially colonize certain sites on the tooth surface and accumulate to form dental plaque, where they metabolize dietary constituents, principally carbohydrates, to form organic acids. The acids are formed close to the tooth surface and are thus less affected by the diluting and buffering effects of saliva[4, 5].

These acids are able to dissolve tooth substance and this occurs slowly, intermittently and initially below the enamel surface. Destruction of tissue is characteristically preceded by softening, brought about by partial dissolution of mineral ahead of the final destruction of the organic matrix

of the tissue and residual mineral. Due to the characteristic pattern of subsurface destruction, caries can be distinguished from other destructive processes of the crowns of the teeth such as abrasion due to mechanical wear and erosion due to chemical dissolution, most commonly due to acids which may be ingested in fruit juices, etc., or regurgitated from the stomach[5, 6].

1.3. DENTAL CARIES MEASUREMENT (DMFT INDEX)

It is important that the prevalence and pattern of disease can be studied by a quantitative measure that will accurately reflect the extent of the disease in a population. The most commonly employed method to measure the extent of previous damage to the permanent dentition, is by a measure known as the DMF index, where D represents the number of decayed teeth (surfaces), M the number of missing teeth and F the number of filled teeth (surfaces). DMF index is a sum of these components. It is an arithmetic index of the cumulative caries attack in a population[7].

The designation DMF (T) is used to denote decayed, missing, filled teeth, DMFS denotes decayed, missing and filled surfaces in permanent teeth and therefore takes into account the number of surfaces attacked on each tooth. A similar index for primary dentition is def (t) or def (s) index denoting the number of decayed, indicated for extraction or extracted due to caries (to differentiate from loss due to natural exfoliation)and filled teeth or surfaces respectively.

The DMF/def index can be used to quantify both caries prevalence and caries incidence in a given population. In case of dental caries, prevalence refers to the proportion of the population with caries experience, past or current. The incidence refers to numbers or proportion of persons developing disease in a specified interval of time, usually a year. It is more common to employ a modified form of this test called the caries increment. This latter measure refers to the number of

new carious lesions occurring in a specified time interval, either for an individual or average over a population[8]. The Index allows comparison of the number of subjects in groups who had experienced dental caries during their lifetime (dmft or DMFT>0) and had active and untreated caries (d or D>0)

1.4. PUFA INDEX

PUFA is an index used to assess the presence of oral conditions resulting from untreated caries. The index is recorded separately from the DMFT/dmft and scores the presence of either a visible pulp, ulceration of the oral mucosa due to root fragments, a fistula or an abscess. Lesions in the surrounding tissues that are not related to a tooth with visible pulpal involvement as a result of caries are not recorded. The assessment is made visually without the use of an instrument. Only one score is assigned per tooth. In case of doubt concerning the extent of odontogenic infection, the basic score (P/p for pulp involvement) is given. If the primary tooth and its permanent successor tooth are present and both present stages of odontogenic infection, both teeth will be scored. Uppercase letters are used for the permanent dentition and lowercase letters used for the primary dentition[8, 9].

The codes and criteria for PUFA index are as follows:

P/p: Pulpal involvement is recorded when the opening of the pulp chamber is visible or when the coronal tooth structures have been destroyed by the carious process and only roots or root fragments are left. No probing is performed to diagnose pulpal involvement.

U/u: Ulceration due to trauma from sharp pieces of tooth is recorded when sharp edges of a dislocated tooth with pulpal involvement or root fragments have caused traumatic ulceration of the surrounding soft tissues, e.g., tongue or buccal mucosa.

F / f: Fistula is scored when pus releasing sinus tract related to a tooth with pulpal involvement is present.

A / a: Abscess is scored when a pus containing swelling related to a tooth with pulpal involvement is present.

The PUFA/ pufa score per person is calculated in the same cumulative way as for the DMFT/dmft and represents the number of teeth that meet the PUFA/ pufa diagnostic criteria. The PUFA for permanent teeth and pufa for primary teeth are reported separately. Thus, for an individual person the score can range from 0 to 20 pufa for the primary dentition and from 0 to 32 PUFA for the permanent dentition. The prevalence of PUFA/ pufa is calculated as percentage of the population with a PUFA/ pufa score of one or more. The PUFA/ pufa experience for a population is computed as a mean figure and can therefore have decimal values[9].

The 'Untreated Caries, PUFA Ratio' is calculated as:

[(PUFA + pufa) ÷ (D+d)] × 100

Reproducibility of the PUFA/ pufa index

To assess the reproducibility of the PUFA/ pufa index three examiners were trained in its use. Fifty, 6-year-old children and 49, 12-year-old children were examined for PUFA/ pufa and the reproducibility assessed by the kappa statistic.

1.5. MINIMUM INTERVENTION (MI)

Minimum Intervention dentistry can be defined as a philosophy of professional care concerned with the first occurrence, early detection, and earliest possible cure of disease on micro levels, followed by minimally invasive, patient-friendly treatment to repair irreversible damage caused by such disease. The benefit for patients from MI lies in better oral health through disease

healing and not merely on symptom relief. Furthermore, minimally invasive treatment assists in reducing widespread patient dental anxieties. MI has the potential for dentists to apply a more conservative approach to caries treatment and simultaneously offer patients less invasive, health-oriented treatment options. MI aims to empower patients through information, skills, and motivation to be in charge of their own oral health, so they only require minimum intervention from the dental profession[7,9].

1. 6. CARIES CLASSIFICATION

Size
0 1 2 3 4
Site
1 1.0 1.1 1.2 1.3 1.4
2 2.0 2.1 2.2 2.3 2.4
3 3.0 3.1 3.2 3.3 3.4

Firstly – lesions are classified according to their location:

- Site 1: pits and fissures (occlusal and other smooth tooth surfaces)
- Site 2: contact area between two teeth
- Site 3: cervical area in contact with gingival tissues

Secondly – the new classification identifies carious lesions according to various sizes:

- Size 0: carious lesion without cavitation, can be remineralized;
- Size 1: small cavitation, just beyond healing through remineralization;
- Size 2: moderate cavity not extended to cusps;
- Size 3: enlarged cavity, with at least one cusp which is undermined and which needs protection from occlusal load;
- Size 4: extensive cavity, with at least one lost cusp or incisal edge.

It is recommended to use the new classification parallel to the widely adopted cavity classification described by Black. The latter remains beneficial for the classification of teeth requiring replacement dentistry[9, 10]. However, the new classification reflects the differences in caries progression in different locations and sizes of decay, and thus may be of benefit for the treatment of new caries lesions, particularly for monitoring and intervention purposes.

1.7. SITES OF OCCURRENCE

Dental caries is initiated by the metabolic activity of the microorganisms in dental plaque, the lesions commence at those sites on the tooth surface where plaque is retained, although plaque may be present without dental caries. These sites are determined largely by the shape of the teeth and gingivae which result in so called ‘stagnation areas’ protected from the mechanical cleaning afforded by tooth brushing and perhaps also by movement of the lips, cheeks and tongue. This traditional view, whilst undoubtedly true, is not, however, the complete explanation[10].

Dental plaque is not, for example, eliminated by fibrous food. Many factors influence the rate of formation of plaque, its volume and its distribution on the tooth and gingivae. The bacterial composition and biochemical activity of plaque differs markedly from tooth to tooth and from site to site on the same tooth and it is a key question of modern caries research to discover the essential differences between cariogenic and non – cariogenic plaque. Nevertheless, most lesions commence in stagnation areas and it is customary to classify both the lesion and any restoration placed to repair such a lesion according to these anatomical sites[10].

The sites are:

1. Pits, fissures and developmental grooves on the occlusal surfaces of posterior teeth or the palatal surfaces of upper anterior teeth (cingulum pits). These are known as **Class I** lesions. The likelihood of caries developing in any of these sites depends on the depth and width of its contour. Teeth with deep, narrow pits and fissures are more susceptible to caries, and prophylactic treatment of these areas with ‘fissure sealant’ is an important preventive measure. Nearly all other lesions arise on the smooth surfaces of teeth in stagnation areas created by concavities or the presence of adjacent teeth.

2. Approximal lesions, develop on surfaces which are in contact with an adjacent tooth. Lesions on the mesial or distal surfaces of premolar and molar teeth are called **Class II** lesions. Those on the mesial or distal surfaces of incisor and canine teeth are designated **Class III** lesions because of different problems involved in their restoration. If the lesion has extended to involve the incisal edge of an incisor or canine tooth it is termed a **Class IV** lesion. On approximal surfaces the stagnation area lies in a vertical direction from the gingival aspect of the contact area to the crest of the interdental papilla and laterally it extends out into the embrasures until these become cleansable. Conventional toothbrushing is an inefficient method of cleaning interdental areas and dental floss, wood points or ‘interspace’ brushes are now advocated for this purpose.

3. Gingival lesions. The gingival thirds of the labial and buccal, and of the lingual and palatal, surfaces of many teeth lie cervically to the maximum convexity of that surface and thus constitute stagnation areas. Lesions in these locations are defined as **Class V**. Labial or buccal surfaces are more commonly affected than lingual or palatal surfaces,

posterior teeth more commonly than anterior teeth, and deciduous molars more commonly than permanent molars. The predilection is explained largely by the more marked curvature of these surfaces.

4. Root surface or cemental caries. Root surface caries is initiated by bacterial plaque residing on the surface of cementum following gingival recession. It is more common in older patients and is becoming increasingly common in Western society with the lengthening of lifespan and the increased effectiveness of preventive measures directed against coronal caries. This form of the disease might have been neglected in the past because affected teeth frequently had advanced periodontal disease necessitating extraction. Plaque accumulates readily on exposed cementum partly because of the spaces created by recession of the gingival and also because cementum has a rough surface. The microorganisms within such plaque, and hence the organisms responsible for cemental caries, may differ from those producing enamel caries.

1.8. THE BURDEN OF DENTAL CARIES

Dental caries is one of the most common diseases in the world and it continues to be a common health problem among children. Its prevalence among population and its extent in individual varies however among nations and over time. Reports on tooth mortality among Nigerians indicated that the major cause of tooth loss in children was dental caries and its sequelae[10]. Though of interest, the prevalence of dental caries has been on the decline in developed countries and this has been attributed to increased use of fluoride in its various forms, while many developing African countries have reported an increased prevalence that has been closely linked

to changing life styles which include diet rich in sugars. Despite the paucity of information on caries prevalence among Nigerian adolescents, high caries prevalence among adolescents in Ibadan, Ife and Ilorin has been reported[10, 11].

Conversely, In England and Wales a national survey of adults conducted in 1968 found only 3 people in 1000 with 28 or more teeth present and free from caries. The survey of Children's Dental Health in the same regions, 1973 (Todd 1975) revealed a mean Decayed, Missing and Filled Teeth (DMFT) figure of 3.9 at age 6, 5.0 at age 8 and 8.4 at age 15 years[11]. DMFT denotes the total number of decayed, missing and filled teeth in each mouth. In 1968 the loss of all teeth, a state indicating total failure of past preventive and restorative treatment, had been reached by one-quarter of the population of England and Wales by the age 40 and three-quarters by the age of 60 years[11, 12].

The situation is now markedly improved, in children, in most of the industrial countries, though the effects of past neglect will take several decades to work through the community. Nevertheless, these few figures serve to show the enormity of the problem. In the United Kingdom the dental care delivered to the community over the past 30 years, as measured by the number of courses of treatment delivered within the National Health Service (NHS), has increased considerably. The improvement in dental health has, presumably, resulted from a combination of increased exposure to fluoride, increased efficiency by the profession and, in one part of the community, substitution of fats for carbohydrate as the major source of energy[13].

It is clear however that the gap between the need for care and the ability of the profession to meet that need will never be closed by increased treatment efficiency alone. If this is true of societies in Western Europe and North America, then it is clear that a system based on

professional dentists providing sophisticated restorative treatment will be even less likely to meet the needs of the developing countries of the world, where a vast number of the population have virtually no access to dental care services. Furthermore, in many of these countries, Nigeria inclusive, the incidence of dental caries is rising alarmingly[14].

These arguments point to the need for increased emphasis on prevention of oral disease as the best long – term solution. Quite a lot is known about the causes and mechanisms of dental caries and periodontal diseases for both to be regarded as completely preventable in the majority of individuals and this is illustrated by the families of many dentists, and other highly motivated individuals, who have a low incidence of these diseases[15]. The immediate challenge lies not only in devising and perfecting new, more efficient and more readily applicable, preventive measures, but in learning how best to apply known preventive measures to the community at large.

The importance of searching for those, perhaps relatively small, cohorts within a population who are at especially high risk of disease is becoming increasingly recognized. Another sense in which the cliché that 'prevention is better than cure' is undoubtedly true is in the effect of disease on the individual. The need to restore carious teeth and to replace lost teeth presents the individual with problems of discomfort, inconvenience and expense. Unless satisfactory prosthetic treatment is received, tooth loss will result in aesthetic deterioration and impairment of speech and mastication. Though rarely of life-threatening significance, the last can be important.

In aged individuals the impact on the quality of life of difficulty with chewing should not be underestimated. Loss of teeth leads inevitably to atrophy of the supporting alveolar bone which may be accelerated by the provision of inadequate dentures. Following tooth loss, there is a

progressive alteration in the whole of the facial skeleton and associated musculature, and disease of oral soft tissues and of the temporo-mandibular joint may supervene[16].

The pain and distress caused by inflammation of the pulp due to progression of caries is all too familiar. As this condition is caused by bacteria, the possibility of spread of infection to surrounding bone, to and through contiguous soft tissues, and to more distant sites via the blood stream and lymphatic system, must constantly be borne in mind. In most parts of the world, caries today rarely leads to fatal infection but deaths can and do occur, sometimes from brain abscess, particularly if the patient does not have access to surgical or antibiotic treatment[16]. A visit to any dental emergency clinic or hospital will confirm the extent of personal suffering which arises from dental infections. Oral infections in patients with rheumatic or congenital heart disease are particularly dangerous because of the risk of provoking infective endocarditis.

1.9. ECONOMIC IMPLICATION OF DENTAL CARIES

The treatment of dental caries is accomplished by the general dentist through restorative treatment. These processes are repeated over and again as successive teeth are neglected and eventually extracted due to dental caries. The treatment procedures are indeed expensive and require the attention, skills and time of majority of dentists. Approximately 15 % of the health care budget spent each year in the United States involves, dental treatment, and a majority of this financial outlay is directly implicated to dental caries, in Nigeria no statistics are available as regards dental spending[6, 9, 16]

The costs of primary dental care paid directly or indirectly to the dentist do not include the cost of oral hygiene products sold in the market place. However, the emerging economic scenario

globally, the impact of education, the growing number of dental graduates, insurance programs, retainership, National Health Insurance Service (NHIS) in Nigeria for example, commercial pressures and governmental policies are just some of the factors that have varying economic implications on dental care[17]. Nevertheless, the fact still remains that a huge segment of the population leaves dental caries unattended.

1.10. SIGNIFICANCE OF STUDY

Dental caries and periodontal disease have historically been considered the most important oral health problems around the world. In African countries, however, these appear to be neither as common nor of the same order of severity as in the developed world. The oral health profile of Africa today is very different from that perceived previously. This profile of oral disease is not homogeneous across Africa[1, 5, 7]. Thus, oral diseases known to exist in each community need to be individually assessed in terms of the basic epidemiological criteria of prevalence and severity. This is a prerequisite for the meaningful ranking of community needs and the development of intervention programs with which to address them.

Dental caries is one of the commonest oral diseases in children. Despite this fact, not much attention has been given to studies on this issue among rural Nigerian school children, in fact, there is little or no studies in the Northern part of Nigeria. Most studies that are carried out, especially in Nigeria, evaluate changes in the oral health status of individual subjects and populations and are often based on clinical indicators of disease; there are relatively few evaluation studies on oral health and welfare from the subject's perception. Over the last 30 years, the use of socio-dental indicators in oral epidemiology has been widely advocated, because single measures of clinical disease do not document the full impact of oral disorders[1, 8, 9]. These indicators were constructed and tested in epidemiological studies on different populations to build a more concrete relationship between subjective and objective oral health measures, which would help to estimate the real population needs.

This study would be conducted in a rural secondary school in Ungogo town in Ungogo Local Government area in Kano State. Ungogo Local Government is a rural local government and among the least developed local governments in Kano State. There are enormous pools of

potential research subjects in the chosen local government school in the age range of interest i.e. children 11 – 14 year for the study.

This study will serve to document:

1. The caries experience in school children from a rural setting in a northern state in Nigeria
2. Oral health practices, knowledge, attitudes, behaviour of children in this environment
3. Perceived oral health needs of the younger population in this community
4. The preventable measures or management options available in this environment

Recommendations may then be made towards ensuring that good quality oral health care services are provided to patients with dental caries, in order to enhance their overall quality of life, and that of all Nigerians. Thus, this study will add to the existing literature on dental caries and oral health quality of life in rural school children in Nigeria. The information from the study will also be useful to other researchers around the globe to stimulate further research in the field of oral health.

1.11. AIM OF THE STUDY

To study dental caries in children 11 – 14 year – old in Ungogo Local Government in Kano State, Nigeria

1.12. OBJECTIVES OF THE STUDY

1. To determine the prevalence of dental caries in children 11 – 14 years old in Ungogo Local Government (L.G.A) in Kano State
2. To determine the DMFT scores in children (11 – 14) years old in Ungogo L.G.A
3. To determine the PUFA scores in children (11 – 14) years old in Ungogo L.G.A
4. To evaluate the oral hygiene practices in the study group

Chapter 2

LITERATURE REVIEW

2.1. EPIDEMIOLOGY OF DENTAL CARIES

2.1.1. CARIES IN PREHISTORIC MAN

Dental caries may probably be considered a disease of modern civilisation, since prehistoric man rarely suffered from this form of tooth destruction. Data on the occurrence of dental caries in ancient populations are available owing to the fact that teeth are relatively imperishable in dry burial sites for many years and no caries – like lesions have been produced in cadavers. Anthropologic studies of Von Lenhossek revealed that the dolichocephalic skulls of men from preneolithic periods (12, 000 BC) did not exhibit dental caries, but skulls from brachycephalic man of the Neolithic period (12, 000 to 3000 BC) contained carious teeth[18]. In most instances the lesions were noted in older persons in teeth which showed severe attrition and impaction of food. Apparently the carious lesions were found at or just below the contact areas and an increased frequency of caries at cemento – enamel junction was noted[18,19].

2.1.2. CARIES INCIDENCE IN MODERN CULTURES

In about 17th Century, there was a significant increase in the total caries experience and a smaller increase in the number of carious lesions involving the interproximal contact areas of teeth, thus signalling a trend that was more characteristic of pattern and occurrence of caries in modern population[19]. Today, dental caries is virtually a universal disease as civilisation has penetrated

almost all areas of the world. Extensive studies have been made on the incidence of dental caries from various geographic areas involving different races to illustrate the apparent influence of civilisation on dental diseases.

Mellanby in 1934 reviewed the literature on caries in existing primitive races and noted that the incidence was invariably less than that in modern man. However, isolated populations that have not acquired the dietary habits of modern, industrialized man retain a relative freedom from dental caries[20]. Eskimos living in the northwest territories of Canada, Alaska and Greenland who consumed native food had a lower incidence of carious lesion compared to those living at trading post. Price reported that Alaskan Eskimos living in isolated conditions exhibited a caries incidence of approximately 0.1 %, while Eskimos living in areas with access to processed foods showed an incidence of 13 % of the teeth examined[18, 20].

A comparable effect of diet upon caries was demonstrated by Mellanby in studies on natives of Southern Rhodesia. Similar results have been reported in studies of native Samoans by Restarski, Maoris Pickerill and Bedouins by Clawson. The change in diet from one that is essentially primitive, to one characteristic of highly industrialized society indicates that modern civilization and increased dental caries are constant in their association, and that the determinants of the carious process are essentially local and limited to the oral cavity[21]. Although there may be a certain degree of racial resistance to dental caries, the dietary factor appears to be more significant, especially since caries incidence increased by contact with ‘civilized’ foods.

2.2. CARIES PREVALENCE IN MODERN DAY POPULATION

Dental caries is all pervading in modern man living in highly industrialized societies. However, the caries experience varies greatly among countries and even within countries. The difference in

caries rates noted in different parts of the world are extreme from rates fewer than one decayed, missing and filled (DMF) tooth per person at all ages up to 39 years in Ethiopia to 60 times greater in Alaska[22].

Findings from the Interdepartmental Committee on Nutrition for National Defence (ICNND) and World Health Organisation studies (Barmes, 1981) indicate that caries prevalence follows definite regional patterns. Caries prevalence is generally lowest 0.5 – 1.7 DMF in Asian and African countries and highest (12-18 DMF) in America and other Western countries[22, 23]. Consistently, low to moderate caries rates were found in populations of the Indonesia, Chinese peninsula, Malaysia, Central and Southern Thailand, Burma,, South Vietnam, Mainland China Taiwan, India and New Guinea. Generally, highly industrialized countries have the highest caries indices with decayed, missing and filled teeth (DMFT) of approximately 4.5. Nonetheless, within this large group of countries a very high caries pattern of over 5.6 DMFT occurs in New Zealand Australia, Brazil and Argentina[24].

Several epidemiologic studies have been carried out to establish baselines of the caries experience. These show that there are no areas in the United States for example, which have been investigated where the people are totally free of caries. Caries in children of many localities begins shortly after eruption of the deciduous teeth and may continue to increase at a remarkable rate. The exposure of children during the period of tooth formation to a water supply containing either naturally occurring or artificially added fluoride greatly influences the prevalence of caries[22, 24, 25].

2.3. FACTORS AFFECTING CARIES PREVALENCE

2.3.1. RACE

Some studies show notable differences in the caries experience between various races, American blacks and whites, living in the same geographic areas under similar conditions, offer an excellent opportunity for comparison. Investigations indicate that the blacks have fewer carious lesions than the whites[26]. Most studies concerning other races have been relatively unsatisfactory because of complicating factors such as differences in diet or exposure to fluoride, which tends to mask any differences due to racial background. Nevertheless there, is some evidence to indicate that blacks, Chinese and East Indians have considerably less caries than American Whites. The English have appallingly poor teeth and a higher caries incidence than Italians, Russian and Chinese[26, 27].

2.3.2. AGE

Carious lesions that result in cavitations are irreversible and therefore cumulative with age. There is a strong correlation between age and DMF indices. Several studies have shown that by the age of six years, about 20 per cent of children have experienced dental caries in their dentition and a DMFT of 0.5 can be expected. By age of 12 years, 90 per cent of children would have experienced a DMFT of approximately 5.5; the decayed, missing and filled surface (DMFS) accelerates at a greater rate than the DMFT beyond the age of eight years, so that, by age 12, a DMFS of 7.5 may be taken as an average figure[26, 28].

Weddell and Klein examined children who ranged in age from 6-36 months and resided in a community with water fluoridation. They found dental caries in 4.2 percent of the children of 12-17 months of age. 19.8 per cent of those of 24-29 months of age and 36.4 per cent of those of 30-36 months of age. Children in the middle and lower middle socioeconomic groups showed a trend towards higher caries incidence. Tang et al, performed dental caries examination in 5,171 pre-school children recruited from public health assistance programs in Arizona. They found caries in 6.4 percent of one-year-olds, 35 per cent of three year-olds, and 49 percent of four-year-old children in the study[29]. In general, other reports of caries prevalence among children in various parts of the world show rates that seem to be comparable to those cited here. Another common element is that children from families in low socioeconomic groups consistently have greater caries prevalence than their peers from families at a higher socioeconomic level[27, 29].

2.3.3. GENDER

One further variation in caries incidence, besides age and geographic areas, is that between the sexes. Studies indicate that the total caries experience in permanent teeth is greater in females than in males of the same age. This is attributable largely to the fact that the teeth of girls erupt at an earlier age than do the teeth of boys[2, 9, 30].

Conversely, the caries experience in deciduous teeth is greater in males. This time difference is particularly significant during the formative years because teeth have been shown to be maximally susceptible to dental caries immediately after eruption. This is due to the fact that the chemical structure of teeth in the immediate post eruptive stage is Suboptimal in terms of caries resistance, as teeth are exposed to saliva and constituents in the diet, the outer layers of the tooth

take up additional minerals from the oral environment in a process known as posteruptive maturation. This maturation process confers a greater resistance to dental caries on the tooth[27, 30].

2.3.4. FAMILIAL

The familial pattern of the caries experience seems to hold true, siblings of individuals with high caries susceptibility are also generally caries active, whereas siblings of caries immune individuals generally exhibit low caries rates. Children of parents with a low caries experience also tend to have low caries; the converse is true for children whose parents have a high caries rate[27]. Studies have been made of the dental caries experience in monozygotic and dizygotic twins; such studies indicate that concordance for carious sites in monozygotic twins is much higher than in dizygotic twin pairs[27, 31].

2.4. CURRENT TRENDS IN CARIES INCIDENCE

Significant data have been presented since 1980 to substantiate various observations that there has been marked improvement in dental health as measured by prevalence of dental caries, especially in children and young adults, throughout the 'civilized western world' since approximately 1960.

This trend has become so definitely established that the First International Conference on the Declining Prevalence of Dental Caries was held at the Forsyth Dental Centre in Boston in June 1982 to evaluate "the evidence and the impact on dental education, dental research and dental practice"[32].

Studies carried out under the National Caries Program in 1979-80 and reported by Brunelle and Carlos on 38,000 school children, aged 5-17years, and representative of approximately 48 million US school children, revealed a substantial decrease in the prevalence of dental caries[58]. Especially impressive was the increase in the percentage of children classified as caries-free in their permanent dentition, for example, in the 12-17 year group in the 1971-74 survey, 9.7 percent of the children were caries-free, while in the 1979-80 survey[33]. 17.2 percent were caries-free. Immediately since the previous survey in 1971-74 the decayed, missing and filled permanent tooth surfaces in the 5-17 year age group declined from 7.06 to 4.77.

Glass reported trends in caries prevalence in 1,775 children 7-13 years of age, analyzed over a period of 20 years in Massachusetts. He similarly reported that dental caries prevalence had decreased by about 50 percent and extractions due to caries decreased by 70 percent. In addition secondary caries had decreased to near zero. The changes had occurred in the absence of fluoridation and organized preventive programs[33, 34].

DePaola and his associates reported decreases of a similar magnitude in surveys on 9,000 school children over a 30-year span. Such an improvement in dental health was not limited to those living in the United States. Data revealing a decline in caries prevalence were also reported at this conference, representatives from England, Denmark, Ireland the Netherlands, New Zealand, Norway, Scotland and Sweden, Unfortunately, it was acknowledged that the prevalence of caries has been reported to be increasing in certain less developed countries[31 - 34].

The cause for this widespread decline in the prevalence of dental caries is a matter of speculations but almost certainly multi-factorial. In some instances, communal water fluoridation has been present in the areas studied, but in other instances it was not. Organized preventive

dentistry program had been available in some cases but not in others. However, the time period involved in most of these studies does coincide with the introduction and increased utilization of fluoride dentifrices and dietary fluoride supplements, as well as an increased awareness of the importance of oral health.

The very limited studies available give no evidence that there is any change, for example in the pervasiveness of streptococcus mutans or any changes in dominant serotypes. Finally, dietary habits and eating patterns are difficult factors to study and analyze. Still, there has been an obvious moment towards improved physical health through food and exercise, although this has been directed more towards adults than children. However, it is conceivable that a reduction in carbohydrate consumption might be related to this reduction of caries prevalence[35].

It now appears that the continued reduction of dental caries from whatever combination of factors, coupled with the use of additional agents and techniques now in various stages of research and development, could result in the near total elimination of dental caries in the very foreseeable future.

2.5. AETIOLOGY OF DENTAL CARIES

The aetiology of dental caries is generally agreed to be a complex problem complicated by many indirect factors that obscure the direct cause or causes. There is no universally accepted opinion of the aetiology of dental caries. Numerous references on dental caries, including early theories attempting to explain its aetiology, have been found in recorded history of ancient people. However, many theories have evolved through years of investigation and observation. The

acidogenic theory (Miller's chemicoparasitic theory), the proteolytic theory and the proteolysis – chelation theory, are among many which have stood the test of time.

2.6. THE EARLY THEORIES

2.6.1. THE LEGEND OF WORMS

The earliest reference to tooth decay is probably from the ancient Sumerian text known as the, Legend of worms. This dates from about 5,000 BC after its discovery on a clay tablet, excavated from an ancient city within the Euphrates valley of the lower Mesopotamian area[36]. The idea that caries is caused by worms was universal as is evident from the writings of Homer who made a reference to worms as the cause of toothache[36].

2.6.2. ENDOGENOUS THEORIES

With the fading away of the legend of worms in the early centuries, the humoral theory was advocated by Greek physicians who proposed that dental caries is produced by internal action of acids and corroding humors and an imbalance in these humors resulted in disease. Along with this, the early Greek physicians such as Hippocrates, Celsus, Galen and Avicenna, proposed the Vital theory of tooth decay, which postulated that tooth decay originated, like bone gangrene, from within the tooth itself[36, 37].

2.6.3. CHEMICAL THEORY

Parmly in the 1820s observed that dental decay affected externally, not internally, as had been claimed. It was proposed that an unidentified 'chymal agent' was responsible for caries. Support

for the chemical theory came after Robertson in 1835 proposed that dental decay was caused by acid formed by fermentation of food particles around the teeth[37].

2.6.4. PARASITIC THEORY

Apparently the first to relate microorganisms to caries on a causative basis as early as 1843 was Erdl who described filamentous organisms in the membrane removed from teeth. Shortly thereafter, Ficnus in 1847, a German physician in Dresden, definitely attributed dental caries to 'denticolae' the generic term he proposed for decay related microorganisms. The parasitic concept of dental caries was soon disseminated by two other German physicians, Leber and Rottenstein, who clearly expressed the concept that dental caries commenced as a purely chemical process, but that living microorganisms continued the disintegration in both enamel and dentin. In addition to these observations, Clark (1871, 1879) Tomes (1873) and Magitot (1878) concurred in the belief that bacteria were essential to caries although they suggested an exogenous source of the acids[33, 35, 37]. In 1880, Underwood and Milles presented a septic theory with the belief that acid capable of causing decalcification was actually produced by bacteria feeding on the organic fibrils of dentin. They reported section of decayed dentin having micrococci as well as oval and rod shaped forms.

2.7. POSTULATED THEORIES AND PATHOGENESIS

2.7.1. THE ACIDOGENIC THEORY

A number of explanations of the carious process which exist, majority of the available evidence support the 'acidogenic theory', or 'chemico-parasitic theory', propounded in some detail as long ago as 1890 by an American, W.D Miller. Miller based his ideas on a series of experiments

conducted in the laboratories of the German microbiologist, Robert Koch. He drew heavily on the new knowledge of bacteriology emerging in Europe at that time, from his studies of chemistry in Edinburgh and his clinical experience in dentistry and medicine in the United State[37].

A particular significance was the observation that organisms could produce acid from the fermentation of carbohydrate. Miller showed that a number of oral microorganisms had this property, which lactic acid was one of the major acids formed, and that extracted teeth could be decalcified by incubating them in mixtures of sugar or bread with saliva. At about the same time Williams (1897) recognized that bacteria adhered firmly to enamel surfaces, producing a film which he considered might localize acid to its most dangerous site – in contact with the tooth[37].

The chemico – parasitic theory postulates that acids are produced at or near the tooth surface by bacterial fermentation of dietary carbohydrates and that these acids dissolve the apatite crystals which make up some 95% of the mass of the enamel. Washing away of the acid is reduced by the presence of dental plaque, which also serves to hold the products of dissolution close to the tooth surface[37, 38,].

Many different kinds of bacteria aggregate in protected portions of the tooth surface to form dental plaque and if the kinds of organism recognized as cariogenic are present in substantial numbers, damaging amounts of acid can be formed. Readily fermentable carbohydrates, particularly those with a low molecular weight such as the sugars glucose, fructose and sucrose directly affect the pH when ingested. This causes the pH in plaque to fall to 4.5 or 5 within 1-3 minutes and takes 10-30 minutes to return to neutrality[38]. Subsequent intakes of carbohydrate may depress the pH even further. This characteristic grid is known as a ‘Stephan curve’. Such

levels of acidity are dangerous because, whereas at neutrality human saliva and dental plaque are supersaturated with calcium and phosphate, at about pH 5 this saturation is overcome and enamel solubility increases markedly[37, 38].

There is ample evidence that a direct correlation exists between type and frequency of carbohydrate ingestion and the lowering of intraoral pH, and a similar correlation exists between dietary carbohydrate levels and caries incidence. The acids involved can be identified in the test tube in sugar – saliva mixtures, after incubation of various other foodstuffs with saliva or dental plaque microorganisms, and with more difficulty in the dental plaque and in carious enamel itself. They are all organic acids, principally lactic acid, and are produced as end-products of the Embden – Myerhof glycolytic pathway, or of other pathways which these bacteria use in the catabolism of carbohydrate[38, 39]. These acids are capable of producing in the laboratory exact histological replicas of the early natural carious lesion when sound extracted teeth are placed in sterile artificial systems containing the acid in a colloidal medium such as gelatin, agar or hydroxymethyl cellulose. The gel apparently acts by providing a diffusion barrier for the products of enamel dissolution.

Several other theories of caries aetiology have been advanced over the years, though none has convincing experimental support. Nevertheless, they are by no means all mutually exclusive and some of these mechanisms may operate to a minor degree in the process of tooth destruction.

2.7.2. THE PROTEOLYTIC THEORY

This is attributed to Gottlieb, who in 1944, suggested that proteolytic enzymes liberated by oral bacteria destroyed the organic matrix of enamel so that the crystals became detached and the structure collapsed – rather like the mortar softening and weakening an old brick wall. In the

same year, a similar interpretation was published by Frisbie, and in 1949 Pincus extended the concept by proposing that sulphatases of Gram negative bacilli hydrolysed the sulphated muco – substances of the matrix, liberating sulphuric acid, which then dissolved mineral[36 - 40].

Whilst there is no doubt that a wide variety of proteolytic enzymes are produced by dental plaque, and these may be of importance in damaging soft tissue in the initiation and progression of periodontal disease, proteolysis is unlikely to be of prime importance in the initiation of enamel caries. In vitro studies show that tooth hard tissues require demineralization before proteolytic enzymes can hydrolyse their matrix proteins[40]. Furthermore, those portions of enamel with a relatively high organic content, such as tufts and lamellae, do not show greater susceptibility to decay. Nevertheless it would be unwise to ignore the proteolytic activity which is undoubtedly present, for, along with the more obvious destruction of mineral, alterations in the organic matrix of enamel occur and presumably influence the progress of the lesion[40].

These, however, are not easily detected because of the small amount of organic material present (less than 1% protein by weight in sound tissue). Organic changes certainly include breakdown of intrinsic enamel matrix, and it is now known that some of this is acid soluble. In addition, there is ingress of additional organic matter derived from oral fluids and bacteria, which fills the gaps between partially dissolved apatite crystals. In carious destruction of cementum, and of dentine, proteolysis is, undoubtedly more noticeable, though it may be necessary for the tissue to be first demineralized by acid, thus allowing the enzymes access to collagen and ground substance[39, 40].

2.7.3. THE PROTEOLYSIS-CHELATION THEORY

The proteolysis – chelation theory was proposed by Schatz, Martine and co-workers in 1954, and in a large number of subsequent publications. In this theory, It is proposed that products of the proteolysis of tooth substance, and possibly also of the acquired pellicle and foods, act as chelating agents which remove calcium ions from the tooth. The significance of this suggestion is that chelation, a process whereby metallic ions are complexed to another molecule by coordinate covalent bonds, is possible at neutral or even slightly alkaline pH. Thus enamel destruction could occur at times when plaque pH was close to neutrality[41].

The peptides and amino acids produced in this way do have chelating activity, as do a number of other molecules likely to be present in dental plaque. These latter include lactate, hydroxyl and keto esters of the Embden – Myerhof glycolytic pathway, and intermediates of the hexose monophosphate shunt, but all are weak chelating agents and are not present in sufficient concentration to account for the amount of demineralization that occurs. It is probable that chelators in plaque are exhausted by binding with the more readily available and more soluble calcium ions derived from saliva[41, 42, 43].

Nonetheless, this theory is striking in that it reconciles the conflict between whether it is destruction of matrix or mineral which is the key event by proposing that both occur simultaneously and interdependently. Some features of the histopathology of enamel caries can be simulated in vitro with chelating agent, and mixtures of oral bacteria and carbohydrates permitted to undergo glycolysis and then neutralized are capable of dissolving enamel. Thus, whilst chelation cannot be regarded as a major part of the destructive process in enamel caries, it

may play a minor role for a period after plaque returns to neutrality following an acid pulse such as is visualized in a Stephan curve pattern[39, 41, 42, 43.]

2.7.4. THE SUCROSE-CHELATION THEORY

Eggars – Lura has proposed in a series of papers (1948-1968) that the very high sucrose concentrations often encountered in the mouths of caries – active individuals form calcium saccharate, that is, a direct interaction between sucrose and calcium takes place. This however is unlikely to be a significant process, because of the rapidity with which sucrose is metabolized to acid and polysaccharide, and because calcium saccharates can only form at high pH, above the range usually found in the mouth[42, 43].

2.7.5. AUTOIMMUNITY

In 1798 the famous John Hunter proposed that the initial event in dental caries took place within the tooth, caries being secondary to inflammation of the pulp. Jackson and Burch, in recent years, have revived this 'intrinsic' concept of caries aetiology. They suggest that clones or regions of odontoblasts in specific sites within the pulp of teeth are damaged by an autoimmune process so that the defence capacity of the overlying dentine and enamel is compromised and conclude that caries should be regarded as a degenerative disease[43].

The hypothesis is based on complex statistical analysis of the frequency and distribution of lesions within the mouths of a large number of individuals, and comparison with statistical analyses of known autoimmune diseases. These authors argue that if, for example, caries develops on the mesial surface of a maxillary central incisor, it is reasonable to assume the disease will eventually involve the adjacent surface of the adjoining tooth, because of a common plaque environment. However they have shown that from the age of 22-60 years, the ratio of the

number of attacks on single central incisors to those on both incisors remains approximately constant at 1: 0.7. They conclude that the initiating events correspond to a form of somatic gene mutation in central growth control stem cells; descendant mutant cells synthesize auto – antibodies which damage specific groups of odontoblasts and thus determine the sites of caries susceptibility[42, 43, 44].

Difficulties in accepting these arguments arise from the fact that most of the epidemiological data used are derived from routine clinical studies and it is doubtful whether such data are accurate enough for mathematical analysis. In addition, the data are cross – sectional, not longitudinal, and it could be that the findings are a statistical artefact arising from selection of subjects with similar caries experience at different ages. It may be possible to reconcile Jackson's findings with events at the tooth surface because it is now known that there are marked local variations in plaque flora and thus in metabolic activity. Moreover, an incisor which is caries – free clinically may show caries in a histological section. Finally, no histological evidence of primary damage to odontoblasts has been produced[44].

2.8. CURRENT CARIES CONCEPT

Dental caries is a multifactorial disease in which there is interplay of three primary factors; the host, the microbial flora and the substrate, in addition a fourth factor – the time – must be considered in any discussion of the aetiology of caries. In other words, caries requires a susceptible host, a cariogenic flora and a suitable substrate that must be present for a sufficient length of time[45, 46]. Conversely, caries prevention is based upon attempts to increase the resistance of the host, lower the number of microorganisms in contact with the tooth and modify

the substrate by selecting noncariogenic foodstuffs and reduce the time that the substrate is in the mouth by limiting the frequency of intake.

2.9. DIAGNOSIS AND MANAGEMENT OF DENTAL CARIES

There has been remarkable progress in the reduction of dental caries globally over the past 30 years. The existence of children with no dental caries, a rarity in the past, is no longer unusual. The use of fluoride in public water supplies, in toothpaste, and in professional dental products, improved oral hygiene, and increased access to dental care have played major roles in this dramatic improvement. Nevertheless, dental caries remains a significant problem. Nearly 20 percent of children between the ages of 2 and 4 have detectable caries, and by the age of 17 almost 80 percent of young people have had a cavity — a late manifestation of dental caries infection[47].

In addition, more than two-thirds of adults aged 35 to 44 years have lost at least one permanent tooth due to dental caries, and older adults suffer from the problem of root caries. Moreover, there remain large segments of the population in which the disease remains a major problem. Noting that dental caries is an infectious, communicable disease resulting in destruction of tooth structure by acid-forming bacteria found in dental plaque, an intraoral biofilm, in the presence of sugar. The infection results in loss of tooth minerals. This begins on the outer surface of the tooth and can progress through the dentin to the pulp, ultimately compromising the vitality of the tooth.

During the past few decades, changes have been observed not only in the prevalence of dental caries, but also in the distribution and pattern of the disease in different populations worldwide. Specifically, it has been observed that the relative distribution of dental caries on tooth surfaces

has changed, and the rate of lesion progression through the teeth is relatively slow for most people[48]. These changes have important implications for diagnosis and management of incipient lesions, predicting caries risk, and conducting effective disease prevention and management programs for individuals and populations.

The application of multiple diagnostic tests to the individual patient increases the overall efficacy of caries diagnosis. Existing diagnostic modalities require stronger validation, and new modalities with appropriate sensitivities and specificities for different caries sites, caries severities, and degrees of caries activity are needed.

The use of sharp explorers in the detection of primary occlusal caries appears to add little diagnostic information to other modalities and may be detrimental. Studies employing receiver operating characteristic (ROC) analyses have shown radiology to have acceptable diagnostic efficacy in detecting larger cavitated lesions in numerous in vitro and in vivo studies[47, 49].

There was agreement that the literature is weak in the areas of diagnosis of caries on root surfaces and adjacent to existing restorations. The problems of assessing the microbiological load of demineralized dentin adjacent to or beneath existing restorations, and differentiating between residual and secondary caries, are substantial and important.

Digitally acquired and post-processed images have great potential in the detection of non – cavitated caries and in the diagnosis of secondary caries. Promising new diagnostic techniques are emerging, including fiber – optic transillumination and light and laser fluorescence.

These new modalities and developing digital imaging systems require robust laboratory and clinical evaluation.

Existing diagnostic modalities appear to have satisfactory sensitivity and specificity in diagnosing substantial, cavitated, dental caries; specifically radiographic methods are essential in

diagnosing interproximal carious lesions. However, these modalities do not appear to have sufficient sensitivity or specificity to efficaciously diagnose non – cavitating caries, root surface caries, or secondary caries. There is currently no diagnostic modality which can differentiate between microbiologically active caries and demineralized dentin without caries activity beneath a restoration[48, 49]. The need for the identification and clinical staging of the presence, activity, and severity of dental caries is of paramount importance in the deployment of treatment strategies that employ increasingly important nonsurgical modalities, such as fluoride, antimicrobials, sealants, and no treatment.

Accurate caries prognosis throughout the life span, however, can support an appropriate, individualized level of care for each patient and a more effective use of health care resources for the individual and for the population. In addition, as dentistry moves towards earlier detection of lesions and a more preventive rather than restorative orientation, good risk assessment will be essential for improving the predictive values of new screening and diagnostic methods by preselecting at-risk subpopulations.

2.10. PRIMARY PREVENTION OF DENTAL CARIES

In the last 30 years a number of community- and individual- level strategies for preventing caries, notably water fluoridation and the use of fluoridated toothpastes, have been highly successful. At the community level, water fluoridation has been widely accepted as both effective and of great importance in the primary prevention of dental caries. The individual interventions that may provide additional benefit in the primary prevention of dental caries included application of acidulated phosphate fluoride gel (APF), fluoride varnish, chlorhexedine

gels, pit and fissure sealants, and the use of dentrifices and other products containing non-cariogenic sweeteners[46, 49,50].

- **Acidulated phosphate fluoride gel (APF):** Evidence for the efficacy of APF gel applied 1–2 times per year was consistently positive.
- **Fluoride varnish:** The evidence for the benefit of applying fluoride varnish to permanent teeth is generally positive. In contrast, the evidence for effectiveness of fluoride varnish applied to primary teeth is incomplete and inconsistent.
- **Chlorhexedine gels:** The evidence for the use of chlorhexedine gel is moderately strong, although many of the studies demonstrating its effectiveness used concomitant preventive measures.
- **Pit and fissure sealants:** Pit and fissure sealants have been demonstrated to be effective in the primary prevention of caries, and their effectiveness remains strong as long as the sealants are maintained.
- **Products containing non-cariogenic sweeteners:** Noncariogenic sweeteners have been delivered to teeth as constituents of chewing gum, hard candy, and dentifrices. The evidence for both sorbitol and xylitol is positive, although the evidence for xylitol is stronger. Almost all studies of these agents included other interventions, such as fluoridated dentifrices, dietary modification, and oral hygiene instruction.
- **Combination interventions:** There is reason to believe that preventive strategies may be more effective when they are combined than when they are administered individually. Numerous combination interventions have been studied. These include combined fluoride interventions, Chlorhexedine plus fluoride, chlorhexedine plus sealants, and chlorhexedine plus xylitol. Majority of studies included instructions in dietary

modification and oral hygiene and instructions for control and experimental groups. In general, these combination treatments have been shown to be effective in preventing caries in children.

Consistent positive evidence was found for the effectiveness of all reviewed preventive interventions in unselected populations of children in different countries. Furthermore, the effectiveness of these interventions appears to increase as baseline DMFS (decayed, missing, and filled surfaces) scores increase, suggesting that they may be particularly effective in high-risk populations while raising questions about their cost-effectiveness in low-risk populations.

2.11. REVERSING OR ARRESTING PROGRESSION OF EARLY DENTAL CARIES

The caries process is endemic and potentially both preventable and curable. The latter can be achieved by identifying and arresting or reversing the disease at an early stage. Although more research is needed, there are clinical strategies to do this and they include application of fluorides, chlorhexedine, sealants, antimicrobials, salivary enhancers, and patient education. Fluorides and chlorhexedine can be delivered as varnishes, rinses, or gels. Many of these same strategies are also appropriate for primary prevention[44, 50, 51]. A number of these treatment methods have been tested in clinical populations with promising results.

1. **Fluoride:** is used as fluoride in water and dentifrices, fluoride varnishes. Available for use also as mouth rinses and gel applications.
2. **Chlorhexedine:** For varnishes and gels, are effective as Chlorhexedine mouth rinses
3. **Sealants:** The use of pit and fissure sealants is also capable of arresting caries.

4. **Combinations:** Combinations of Chlorhexedine, fluoride, and/or sealants are suggestive of efficacy.
5. **Antimicrobials:** Although mutans streptococci is recognized as part of the pathology of caries and therefore an antimicrobial approach would seem reasonable and act as antimicrobial treatments along with Chlorhexedine and fluorides, both of which have antibacterial properties.
6. **Salivary Enhancers:** Although there are indications that pathologically low salivary flow, as a consequence of Sjögren's syndrome or as an effect of head or neck radiation treatment or xerostomic medications, is associated with caries, there is no evidence that low normal salivary flow produces a similar outcome.
7. **Behavioural Modification:** Most interventions require patient adherence, and office-based behavioural interventions are useful in arresting or reversing caries progression.

Chapter 3

MATERIALS AND METHODS

3.1. STUDY AREA

This study was conducted at Ungogo town, which is situated in Ungogo Local Government area of Kano State. Its land mass covers the North-East wing of Kano metropolis, boarding Fagge and Dala Local Government areas to the south, Dawakin Tofa to the West, Minjibir to the North-East and Nassarawa to the South. Ungogo Local government has a total population of 168, 373 (2006 Census), with an almost equal male to female ratio (Male = 86, 579 and Female = 81, 794) according to the 2006 National Census.[52] Majority of the population are farmers, cattle/sheep rearers or petty traders. The people are predominantly Muslim Hausa/Fulani. The Local Government area has eleven political wards, four suburban and the rest rural. There are forty one villages and one hundred and thirty five traditional wards. There are twenty three health facilities in the Local Government area.[53]

Ungogo is among the least developed local governments in Kano State, since the creation of the state on April 1st, 1968. Kano State is made up of 44 Local Government areas. It is a homogenous state with a total population of 9, 383, 682 with an annual growth rate of 3.3 (2006 National Census). It has an almost equal distribution of Male (51%) and Female (49%). Over half of Kano state population are children. According to the state education board there are 9, 466 Quranic schools, 3, 421 primary schools, 388 junior secondary schools and 392 senior secondary schools of which 76. 6% of male children have access to education compared to 31.7% for female, 37. 6% have access to portable water supply (pipe – borne water); more than 75% of the populations live in the rural area. Kano State has a 35% literacy rate and average

school enrolment rate is 90% for primary education, 80% for secondary school education and 60% for tertiary education[53, 54]. The gap between male and female school enrolment has significant consequences in education, access to health care, economic and social facilities in the state.

3.2. STUDY DESIGN

This study is a cross-sectional descriptive study of 11 – 14 years old students seen in Ungogo Local Government of Kano State. The participants for this study would be taken from the selected junior secondary schools in the local government using table of random figures (simple random technique) with the subjects chosen from the list of students in the selected schools.

Thereafter, a duly signed informed consent to examine the participants would be obtained from the parent or guardian; the consent form would be distributed to all the selected participants to take home which is returned the next day prior to the examination of the subjects and administering the questionnaires. The clinical examination would be carried out by the researcher. The DMFT and other relevant values would be derived from the questionnaire. The participants would be examined while sitting in a school chair with the use of a disposable dental mirror and sterilised blunt dental probe to prevent cross infection. Caries diagnosis will be based solely on clinical examination.

3.3. STUDY POPULATION

The list of the junior secondary schools in Ungogo local government was provided by the Kano State Secondary School Management Board (KSSMB) and four schools were randomly selected from a sample frame of 15 junior secondary schools. The total population of the students aged 11 to 14 years in the 4 selected schools was 1519 males and 1027 females.

Schools	A	B	C	D	Total
Males	50	52	50	49	201
Females	23	22	24	24	93
Total	73	74	74	73	294

These subjects were randomly selected from the list of students using table of random numbers 201 males and 93 females (294 in total) within the age range (11 – 14years) and also satisfy other inclusion criteria.

3.4. SAMPLE SIZE DETERMINATION

The sample size (n) required for this study was calculated based on previous data from C. Udoye et al 2009[55], with a prevalence (P) of 24.1% (0.24) for caries and a confidence level of 95% (Z= 1.96) and a maximum tolerable error (E) not greater than + 0.05.

$n = (Z \div E)^2 \times P (1 - P)$ (Marchin and Campbell)

Confidence level (Z) at various levels:

- At 95% = 1.96
- At 98% = 2.53
- At 99% = 2.58

The higher the confidence level (Z), the lesser is the sampling error and the more representative is the sample size. Therefore, Z at 95%

Where

n = Sample size

Z = Desired confidence level = 1.96

P = Prevalence = 24.1% = 0.24

E = Maximum tolerable sample error = +0.05

Then,

$n = (1.96 \div 0.05)^2 \times 0.24\ (1 - 0.24)$

$= (39.2)^2 \times 0.24 \times 0.76$

$= 1536.64 \times 0.1824$

$= 280.28$

The actual sample size is 280.28;

Then,

$0.05 \times 280.28 = 14.014$

Total sample size =

$280.28 + 14.014 = 294.29$

The study sample of 294 subjects was required and was randomly selected from the chosen rural school in Ungogo Local Government area.

3.5. SAMPLING TECHNIQUE

A Multi stage random sampling was used, at each stage simple random sampling was employed in selecting the schools then simple random sample for the participants in the study sampling frame.

3.6. ETHICAL CONSIDERATIONS

To respect the participants´ autonomous choices and integrity, they were informed verbally that the study was voluntary, and if any student changed his or her mind before or while answering the questionnaire he/she was allowed to discontinue the participation at any time. The students were also informed that the answers in the questionnaire were confidential and would not be read by anyone else but the researcher. The answered questionnaire would be destroyed when the study results have been compiled.

- Permission was obtained from the Medical Ethics Committee of the Aminu Kano Teaching Hospital (Appendix II).
- Informed consent of the school authorities was obtained before carrying out the study.
- Informed consent of the parents and guardians was obtained before carrying out the study.
- All records were held in strict confidence.
- Clearance was not given to take clinical pictures

3.7. INCLUSION CRITERIA

- All randomly selected participants between 11 and 14 years in the school in the local government.

- Participants who gave consent to participate

3.8. EXCLUSION CRITERIA

- Those that were less than 11years or more than 14years of age
- Participants that failed to give their consent

3.9. INSTRUMENT OF MEASUREMENT

The instrument of measurement is a semi structured interviewer administered questionnaire, developed for the study. The questionnaires were administered to the randomly selected students to obtain information on socio demographic variables and oral hygiene practices and a dentition status (DMFT and PUFA). The total questions were 28 which were divided into four parts with two parts DMFT and PUFA respectively:

i. General information (2 – questions)
ii. Oral health attitudes (11 – questions)
iii. Knowledge about oral health and diseases (6 – questions)
iv. Personal attitudes concerning oral health care (9- questions)
v. DMFT (Decayed, Missing, Filled Teeth)
vi. PUFA (Pulpal Involvement, Ulceration, Fistula, Abscess)

3.10. INTRA-EXAMINER VARIABILITY

Only one examiner i.e. the author (intra-examiner) performed all oral examinations, all the participants were seen by the researcher alone in order to eliminate bias as well as inter – observer errors during caries identification. In order to eliminate misunderstandings in the

questionnaire and to ensure that important details would not be missed, questions were interpreted in the local language (hausa) which is well understood by the researcher.

The study took place during the participants´ break and they had to use their free time to answer the questions.

3.11. VALIDATION OF THE STUDY

There was randomisation of the sample population of the students, who displayed appropriate care and diligence. Though questionnaires were said to often lack validity for a number of reasons; participants may lie, give answers that are desired, fear of being looked down upon and so on, the questionnaire was pretested to ensure consistency.

3.12. DATA COLLATION

At each school visit, the questionnaires were administered by the author to the randomly selected students in the junior secondary classes 1, 2 & 3 of the study age groups (11 – 14years) of the four selected schools in the study area. In total, 201 males and 93 females (294) participated in the study. Oral examination was done and caries diagnosis was done by visual and tactile examination in a classroom setting with a plane mouth mirror, a blunt dental probe and wooden spatula, under natural illumination. Disposable items/examination materials were used to examine the subjects to prevent cross-infection. Study setting made the use of radiographic equipment or fibre optics impracticable. The decayed, missing and filled teeth were scored with DMFT index and PUFA index respectively. All of the 294 questionnaires were answered and collated.

3.13. DATA ANALYSIS

This was done using Statistical Package for Social Science (SPSS) version 14.0 and Microsoft Excel. Categorical variables were analyzed with Chi Square while Means were compared with Student t-test. Absolute numbers and simple percentages were used to describe categorical variables. Similarly, quantitative variables were described using measures of central tendency (mean, median) and measures of dispersion (range, standard deviation) as appropriate. A P-value of less than or equal to 0.05 was considered statistically significant. The entire result was done with Confidence Interval of 95%. Data presentations were done in tables, charts and graphs, and other relevant illustrations. Comparative statistics were generated, and tests of significance determined using the Chi Square test and other relevant statistical tools performed to compare the oral health indices in study age groups.

3.14. STUDY PROTOCOL

The list of the junior secondary schools in Ungogo local government was provided by the Kano State Secondary School Management Board (KSSMB) and four schools were randomly selected from a sample frame of 15 junior secondary schools. The total population of the students aged 11 to 14 years in the 4 selected schools was 1519 males and 1027 females.

Schools	A	B	C	D	Total
Males	50	52	50	49	201
Females	23	22	24	24	93
Total	73	74	74	73	294

These children were randomly selected from the list of students, using table of random numbers 201 males and 93 females who were within the age range (11 – 14years) and also satisfied other inclusion criteria. The participants were seen by the researcher alone.

Informed consent was then obtained from the Principals and caregivers of these selected children. The questionnaires were then administered to the selected students to obtain information on socio demographic variables and oral hygiene practices.

During each school visits, the questionnaires were handed out by the author to randomly selected students in the junior secondary classes 1, 2 & 3 students in the study age groups (11 – 14years) of the four selected schools in the study area. 294 randomly selected participants were taken, 201 were boys and 93 were girls. In order to eliminate misunderstandings in the questionnaire and to ensure that important details would not be missed, questions were interpreted and clarified in the local language (hausa) which is the language spoken by the researcher. The questionnaire administration and oral examinations took place during the participants´ break time. This was to avoid class disruptions and for relaxed atmosphere which would not be possible if it had been arranged during the students ordinary teaching time.

Oral examinations and caries diagnosis were done by visual and tactile examination in a classroom setting with a plane mouth mirror, a blunt dental probe and wooden spatula, under natural illumination. Disposable items and examination materials were used to examine the children to prevent cross-infection. The decayed, missing and filled teeth were scored with DMFT index and PUFA index respectively. All of the 294 distributed questionnaires were answered and retrieved. The study was done from 18th January, 2011 to 28th of January, 2011. Dental advice and referral to Aminu Kano Teaching Hospital (AKTH) was made for proper dental management of some of the participants and fears of attending dental clinic were alleviated during the study.

Chapter 4

4.0. RESULTS

In total, 294 students participated in the study. The majority of the respondents were males. The median age was 12, the mean age was 12.7

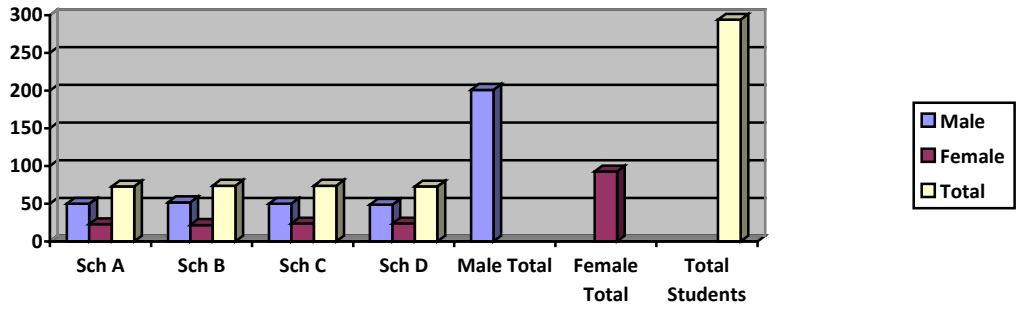


Table 1: Distribution according to gender and schools

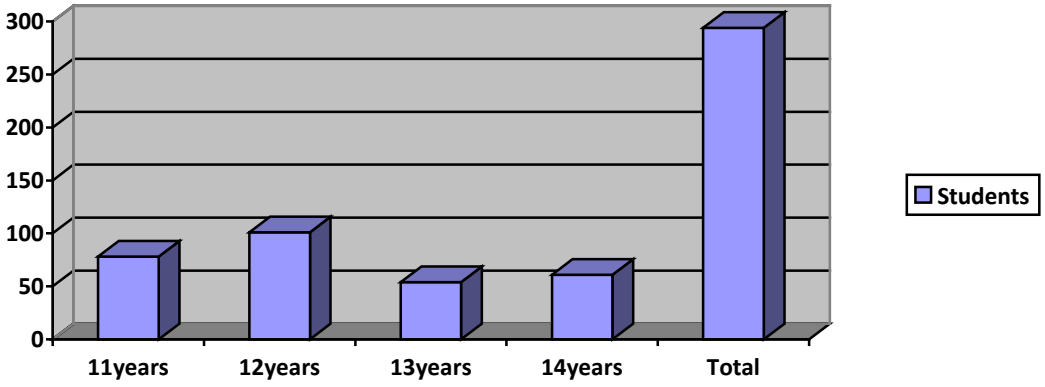


Table 2: Age distribution

Oral Health Attitudes (OHA)

Answer options	i	ii	iii	iv	v	vi	vii	viii	ix	x
Questions										
1-Visiting the dentist	161	33	36	40	0	0	0	21	3	0
2-When sick what you do	146	26	119	3	0					
3-Care of the teeth & gum	207	39	28	56	44	0				
4-Cleaning teeth regularly	73	221								
5-Infection of the gum	74	201	19	0						
6-Foods affecting teeth & gum	232	62								
7-Tooth ache & dentist	219	63	52	52						
8-Prefer tooth filled	39	31	7	2	256	9	0			
9-Scaling Teeth (clinic)	141	52	71	42	161	0				
10-Children loosing teeth	259	35								
11-Children gum disease	73	221								
Total										

Questionnaire: Dental caries in children 11-14 years, Ungogo Local government, Kano state (Sani, 2011)

Table 3: The distribution of answers on OHA

Oral Health Knowledge (OHK)

Answer Options	i	ii	iii	iv	v
Questions					
1-foods + toothache	200	19	15		
2-foods + healthy teeth & gum	189	80	7	18	
3-dentist visit + teeth & gum	142	100	52		
4-brushing + chewing stick	159	61	74		
5-Gum disease causes	38	152	84	57	
6-Tooth decay causes	31	90	89	172	44
Total					

Questionnaire: Dental caries in children 11-14 years, Ungogo Local government, Kano state (Sani, 2011)

Table 4: The distribution of answers on OHK

Oral Health Behaviour (OHB)

Answer Options	i	ii	iii	iv	v	vi	vii	viii	ix	x
Questions										
1-Brush teeth with	178	2	47	30	0	11	49	0	18	22
2-Times cleaning teeth	51	107	92	17	10	17				
3-Brushing with chewing stick	221	73								
4-Eating fresh fruits	54	99	68	30	43					
5-Eating vegetables	29	107	37	30	91					
6-Eating protein foods	36	87	48	52	71					
7-6months dentist visit	0	217	77							
8-Last dental visit	13	281								
9-Eating sweet foods	84	15	79	68	27	21				
Total										

Questionnaire: Dental caries in children 11-14 years, Ungogo Local government, Kano state (Sani, 2011)

Table 5: The distribution of answers on OHB

Table 3 shows that only 13 of 294 respondents had visited a dentist one to two times and nine had been to a dentist more than five times, during their life. The reasons for non dental visits are described in the table as 161 claimed they had no problems. In total, all respondents answered the questions and of these, ten gave more than one answer. 146 agreed to take drugs from their parents for any ailment. 207 answered on importance of care of the teeth and gum while 221 agreed to regular cleaning though 201 had no idea on seriousness of gum infection.

219 preferred extraction as opposed to only 9 who agreed to fillings even though 256 accepted ignorance of knowledge of dental treatment. 259 believed it's normal for children to lose their teeth and 221 accepted the fact that it's normal for children to have gum diseases even though 232 agreed that types of foods eaten do affect teeth and gum health.

The result showed that 159 respondents brushed their teeth twice daily with chewing stick or toothbrush, 61 respondents stated that they brushed their teeth once daily Table 4. The most frequently used aid for cleaning the teeth was chewing stick (n= 178) Table 5. Other mentioned

aids for cleaning the teeth were salt and branches of ‘dogonyaro’ tree. More than one alternative on the question could be given and 49 reported the use of both chewing stick and toothpaste, and 22 others used salt, toothpaste and chewing-stick. 141 respondents answered that they would like their teeth cleaned in the dental clinic (Table 3). 92 answered daily use of chewing stick or toothpaste, 51 not at all, only 17 twice daily, 10 more than twice another 17 were different variants and finally 107 respondents answered that they brushed not every day (Table 5).

The overall knowledge about oral health and diseases was high concerning caries and gingivitis. Within the study group, 200 answered that dental caries (tooth decay) is caused by sweet foods while 152 agreed that sweet foods also cause gum diseases (Table 4) though there were multiple combination of the causative substances as confirmed by multiple answers from the respondents. 172 claimed tooth decay is caused by little worms (The legend of worms?!) only a few, 31 believe eating garlic causes caries.

One hundred and seven answered that they eat vegetables once a week, 99 take fruits once a week also. Eighty seven claimed they take protein food once a week with associated taking of sweet foods by 79 respondents once a week while 84 everyday, only fifteen answered staying away from sweet foods for different reasons, 68 more than once a week, 27 more than three times a week, while 21 had various responses. In Table 5, in effect 221 brushed their teeth with chewing sticks. Two hundred and eighty one respondents have never been to a dental clinic while similarly 217 do not agree to visit the dentist because they claim they have no tooth problem while the remaining 77 gave different reason and no single respondent visited the dentist every six months. 142 considered that regular 6 monthly visits to the dentist are

necessary, 100 disagreed and 52 were indifferent. Interestingly, 159 agree on use of chewing stick or brushing for healthy teeth and gums while 61 clearly disagreed and 74 variants (Table 5), though majority of the participants were always eager to ask the researcher about oral health care at end of the questionnaire administration.

variable	Males	Females	Total
Dental caries present	175 (87.1%)	79 (84.9%)	254 (86.4%)
Dental caries absent	26 (12.9%)	14 (15.1%)	40 (13.6%)
Total	201 (100.0%)	93 (100.0%)	294 (100.0%)

Table 6: Sex distribution of caries among the 11 – 14 year olds

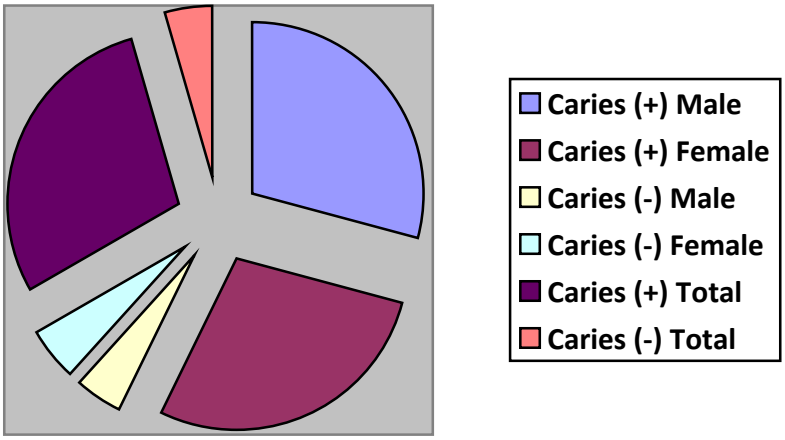


Figure 1: Sex distribution of caries in the subjects

variable	Number	Mean DMFT ± SD
Gender		
Male	201	2.35 ± 1.33
Female	93	2.54 ± 0.86
Total	294	2.42 ± 1.53
Age(years)		
11	78	2.17 ± 1.21
12	101	2.27 ± 0.62
13	54	2.35 ± 1.47
14	61	2.18 ± 1.20

Table 7: Age and Sex distribution of Mean DMFT

Tooth Type	Prevalence (%)
6	46.7
7	44.2
4	2.2
5	5.3
3	-
1	0.8
2	0.8
Total	100.0

Table 8: Caries Distribution among the Different types of tooth

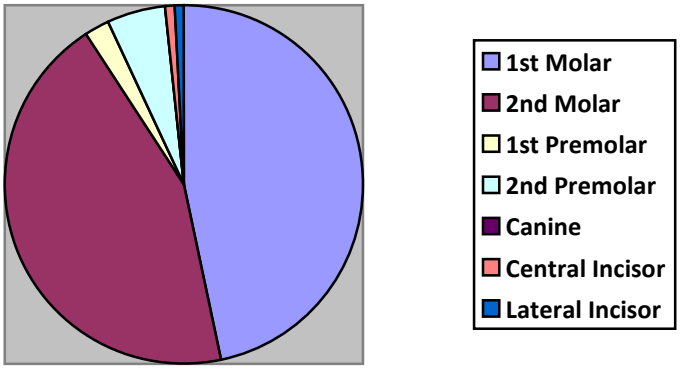


Figure 2: Caries prevalence in dentition

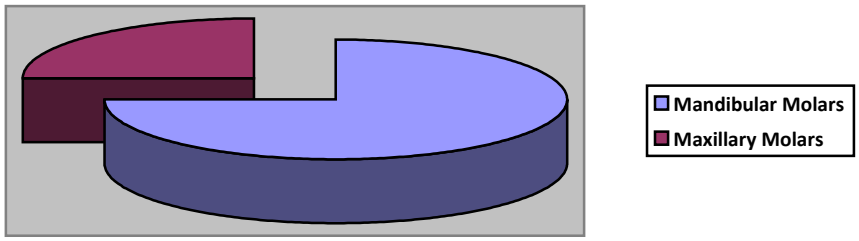
Maxilla

Tooth No.	7	6	5	4	3	2	1	1	2	3	4	5	6	7
Caries (%)	3.1	2.9	0	0	0	0.8	0.8	0	0	0	0	1.6	7.0	8.5

Mandible

Tooth No.	7	6	5	4	3	2	1	1	2	3	4	5	6	7
Caries (%)	0.8	14	22.5	0	0	0	0	0	0.8	13.2	18.6	0.8	11.0	0.9

Table 9: Percentages of intra arch distribution of dental caries



Mandibular molars = 69.8%, Maxillary molars = 23.2%
Figure 3: Percentage inter-arch molar caries

The mean DMFT increased with age, though a drop was observed at age 14. (Table 7) 2.18 ± 1.20 and for males it was 2.35 ± 1.33. The mean DMFT for females was 2.54 ± 0.86. A total of 294 school children aged 11 to 14 years were studied. Of this number, 201 (68.4%) were boys and 93 (31.6%) were girls. Also of the 294 children, 254 (86.4%) had caries, 40 (13.6%) caries free. Sex distribution of caries showed that 175 (87.1%) were boys while the remaining 79 (84.9%) were females. 26 (12.9%) males and 14 (15.1%) females were caries free. There was no significant difference in the sex distribution of caries (P = 0.141) (Table 6). The study population's mean DMFT was 2.42

Dental caries was most prevalent in the first molars (46.7%), followed by the second molars (44.2%), second premolar (5.3%), there was no caries in the canines (Table 8). Dental caries occurred more often in the mandibular molars (69.8%) than in the maxillary molars (23.2%). In

the maxilla, caries was more prevalent in the left half (17.9%) than in the right half (8.6%) and this was statistically significant (P<0.05) (Table 9).

	11(n=78)	12(n=101)	13(n=54)	14(n=61)
Prevalence DMFT > 0	36	78	69	45
Overall caries prevalence	80	82	88	73
Prevalence of a PUFA	58	50	61	53

Table 10: Prevalence of caries and PUFA of 11, 12, 13 and 14-year-old

	11(n=78)	12(n=101)	13(n=54)	14(n=61)
Mean DMFT	2.2 (1.2)	2.3 (0.6)	2.4 (1.5)	2.2 (1.2)
Mean PUFA	1.9 (0.5)	1.0 (1.3)	1.2 (1.5)	1.1 (1.2)
Mean P	2.1 (1.0)	2.8 (1.2)	2.9 (1.0)	2.7 (1.1)
Mean U	2.0 (0.9)	1.3 (0.9)	1.2 (0.9)	2.1 (0.8)
Mean F	1.0 (0.7)	0.9 (0.7)	1.0 (0.7)	1.0 (1.0)
Mean A	1.0 (1.0)	1.1 (1.2)	1.1 (1.3)	0.9 (0.6)

P=Pulpal involvement, U=Ulceration, F=Fistula, A=Abscess

Table 11: Mean caries experience (SD) and mean PUFA experience (SD) of 11, 12, 13 and 14-year-old

In the 11- 14 year-old group, 294 children with a mean age of 12.7 and median of 12 were examined. The overall caries prevalence (age) was 80% (11), 82% (12), 88% (13) and 73% (14) respectively while 85% of 13-year-olds present at least one tooth with pulp involvement (Table 10). The study group dentition presented 2.4 DMFT, purely concentrated on the D component and almost all decay occurring in the first molar (Table 8), no fillings were present and the M component was 0.2. The PUFA index for the dentition as presented (Table 11) showed the 'Untreated Caries PUFA Ratio' was 61%, indicating that 61% of the D component had progressed to an odontogenic infection. PUFA ratio as: [(PUFA) ÷ (D)] × 100

Chapter 5

5.0. DISCUSSION

The results showed that the majority of the respondents irregularly brushed while the most frequently used aid for cleaning the teeth was chewing sticks and about one third of the respondents cleaned their teeth once daily. Good oral health habits among school children were also shown in a Danish study (Christensen et al., 2003).

The overall knowledge about caries, gingivitis and their cause was good among the respondents, though more than half of the respondent were in support of the legend of worms' theory. There were some studies that show similar results (Mani et al., 2010; Wyne et al., 2002) but in contrast with other researchers, Al-Hussaini et al. (2003) showed a lack of knowledge about the causes and the prevention of dental caries among students in Kuwait. The knowledge about dental visit abhorrently was insufficient. This could be explained by the fact that the students never had oral health education or any health talk of some sort. According to Ranganathan & Hemalatha (2006), gingivitis was one of the most common oral diseases in Africa, so the students´ knowledge about the disease should have been higher even though they never had oral health awareness. The same lack in knowledge about gingivitis is shown in this study (Al-Hussaini et al., 2003).

The respondents had a very low attendance to dental services. This could be explained by many reasons, such as access to oral health services, socioeconomic factors and attitudes for oral health (Adeleke & Danfillo, 2005). Irregularity in dental visits was also been reported by another study performed in Jordan in school children (Al-Omiri et al., 2006). Although, the respondents showed a positive attitude concerning oral health care, so irregular dental visits may be explained by limited access to oral health services. The number of oral health services in Kano is not sufficient in relation to the numbers of residents in the state and most of the dental clinics are not

adequately equipped (Jeboda, 2009; Thorpe, 2003). This could also be due to the fact that dental treatments are expensive in some oral health care facilities especially in the private sector and some people cannot afford this dental care (Petersen, 2008). Oral health services may also be seen as luxury and dental attendance might have a low priority among people living in developing countries.

The majority gave one or more alternatives to diet related oral diseases. This result may be expected since Kano is involved in a local hausa programme Rigakafi (prevention) to reduce the prevalence of diseases in general. The results concerning the knowledge and the willingness to have oral health care awareness were widespread among the students. They had also positive attitudes concerning oral health care. The students considered oral health care as a high priority, and as an important issue. The results also showed that the students considered regular dental visits necessary, and that collaboration between dental care and regular health care practices is needed. Positive attitudes concerning oral health care have also been shown in other studies. Wårdh et al (2008) have reported in a Swedish study that the attitudes are more positive among school children compared to other children groups.

Good knowledge and positive attitudes about oral health and oral health care in school children could make it possible to improve oral health and quality of life for their patients (Quandt, 2009; Ranganathan & Hemalatha, 2006). Although the knowledge about oral health was good, the data showed irregularity in dental visits by the respondents.

The limitation of this study includes the impracticability of using radiographs to detect inter-proximal caries. As a result, caries experience figure may be an underestimation of the true caries prevalence if radiographs were to be used. Furthermore, the DMFT values did not show

the number of teeth at risk of developing caries. These limitations notwithstanding, DMFT has been found to be a rapid and universally applicable instrument that has been in use for decades.

The prevalence of caries (82%) in this study was high compared to the report of a previous study (Okeigbemen, 2004). The difference in both studies may be due to the differences in socioeconomic settings, pattern of dental visit, oral hygiene practices, school health programme, parent's education and job. Data from other studies observed that prevalence of caries is least in African countries when compared to Asia and Latin America (WHO, 2003).

These are similar to those observed by Okeigbemen (2004). Some researchers however did not obtain any significant difference between the sexes (Brekhus, 1931; Daneshkazemi et al, 2005). In the study by Hahn et al (1999) there were no gender specific differences with regard to DMFT when assessed by Student t-test but when assessed by multiple regressions, females showed significantly higher DMFT than males. Initial sex differences in prevalence of caries that might occur in children studies have shown that caries prevalence in both sexes tends to be similar in later life (WHO, 1997).± 0.86) when compared to boys (DMFT: 2.35 the significantly higher values of DMFT seen in girls (DMFT: 2.54

Using WHO's DMFT scale of severity and 12year old indicator age group, the finding in present study was high, the current finding of gradual increase in DMFT with increasing age was similar to the report of Okeigbemen (2004) who reported mean DMFT values of 0.51 and 0.66 for 11 and 14year olds respectively. These two reports are also in agreement with those of other studies (WHO, 1997; Alvares-Arenal et al, 1998). The drop in DMFT values noticed at 14years may be an incidental finding. It may however be that ages 11, 12 and 13 are high risk age groups for

caries development, which may be due to inefficient oral hygiene control, increase in plaque index, etc (Alamondi and Mosoud, 1995).

On the other hand, the drop at 14years may be due to transient decrease in the number of cariogenic bacteria and increase in the immuno globulin A (Tenovuo, 1986). This reduction in number of cariogenic bacteria has been shown to accompany the transition between late mixed dentition (Schlageahauf and Rosendhal, 1990). Overall, because the DMFT score of an individual cannot decrease overtime, a direct relationship exists between DMFT index and age (Woodmansay, 2005).

A marked variation noted in caries distribution in the present study agreed with the report of Brekhus (1931) in which the first molars, second molars and premolars are predominantly affected. Furthermore, the current work observed that the D component of the DMFT index accounted for 91.3% of all caries. These findings are also in agreement with the report of Okeigbemen (2004). The high D component is an indication of a high percentage of untreated caries and a high treatment need (Akpata and Shammary, 1992). High DMFT in certain tooth types has been ascribed to similarity in genetic, morphological, structural and ecological features of each type (Mc Donald and Avery, 1985).

Involvement of more mandibular teeth than maxillary teeth agrees with the report of Bajonio et al in USA but not with that of Brekhus (1931) who reported both greater prevalence in the maxillary arch and equal involvement of both halves of the maxillary arch by dental caries. Higher prevalence of caries in the mandibular molar in the current school children study may be related to faster caries progression in the mandibular molar teeth, relatively abundant saliva and its anticaries effect to the maxillary molars than to the mandibular molars, greater food packing

and plaque accumulation potential in the mandibular posterior region than in the maxillary region (Kleinberg and Jenkin, 1964).

During the last decade, international caries epidemiology has focused on the development of more sensitive diagnostic criteria to allow for assessment of the initial stages of caries[9, 58]. This is considered important in the light of the decline of cavitated caries lesions in high-income countries where non – operative and preventive interventions require an index that distinguishes between the different stages of initial caries lesions[9]. However, in low- and middle-income countries, as well as deprived communities within high-income countries, where people have little access even to the most basic forms of care there is a need for a diagnostic index that addresses the advanced stages of untreated carious lesions.

The way caries data are presented has a considerable impact on how it is interpreted by health decision makers. For example, the DMFT of 2.9 for 12-year-old in a Filipinos' study complies with the WHO⁄ FDI goal for the year 2000 of 3 DMFT for this age group[9]. This can lead to complacency among decision makers since the Philippines have already met WHO⁄ FDI goals based on the DMFT. The reality is that in this same age group 41% of the decay component has progressed to odontogenic infections, which clearly demonstrates the limited and often misleading explanatory power of the DMFT. By exposing decision makers only to DMFT data, leaves them uninformed of the high levels of untreated carious lesions, their severity and associated health and quality of life consequences.

The overall caries prevalence (age) was 80% (11), 82% (12), 88% (13) and 73% (14) respectively while 85% of 13-year-olds presented at least one tooth with pulp involvement.

There were no fillings in any of the subjects and very low M components for the age groups. That indicates that more than 90% of caries in the age groups remained untreated. The inclusion of data on pulpal involvement, traumatic ulceration, fistula and abscesses (PUFA) in this study provided a more comprehensive picture of caries and its consequences related to general health of school children population at Ungogo local government of Kano state. Forty three percent of decayed teeth in 11- year-olds and 41% of decayed teeth in 12-year olds had signs of odontogenic infection.

This information may be useful for treatment planning as it will help to calculate the treatment need (restorations, endodontic treatment and tooth extractions) depending on the capacity of the health care system available in the state.

The findings from the study showed a very high PUFA index for the age group, with Untreated Caries PUFA Ratio of 61%, indicating that 61% of the D component had progressed to an odontogenic infection. This clearly indicates the gravity of the caries experience and lack of treatment with consequent deteriorating outcome for the study population of school children 11 – 14year old at Ungogo local government area of Kano state. It is glaring in the results to make a case for urgent oral health action even though ethical approval was not given for clinical pictures to demonstrate some cases also, due to a rarity of similar studies it is difficult to compare the present findings with other countries; however, the prevalence of fistulae and abscesses of 18% among 11-year-old is consistent with the findings for 11-year-old Scottish children. There, the prevalence of sepsis (defined as the presence of an abscess or fistula) was reported to be 11% for children living in the most deprived areas in Scotland[68].

5.1 LIMITATIONS OF THE STUDY

- Oral examinations and caries diagnosis were done by visual and tactile examinations in classroom settings
- Poor light source with impracticability of use of radiographs or fiber – optics for proper caries diagnosis
- Lack of application of multiple diagnostic tests to the individual subjects
- The inability to use digitally acquired and post-processed images in the study, this would have had great potential in the detection of non – cavitated caries and in the diagnosis of secondary caries
- Unable to use new diagnostic techniques, light and laser fluorescence

Chapter 6

6.0. CONCLUSION

The respondents had a poor knowledge of oral health and oral diseases and their causes, though the majority had a positive attitude concerning oral health care and they seldom visited a dentist. However, the overall knowledge of foods related oral diseases was good. The majority of the respondents wanted, however to receive more information about this subject. The knowledge about oral health was poor, although the result showed irregularity in wanting to visit a dentist among the respondents. Within the limitations of the study, the overall caries prevalence in the study population was high. Notwithstanding, ages11, 12 and 13 constitute the ages at greater risk. It is recommended that this high level be brought lower by adopting cost effective preventive measures directed particularly at these age groups.

The dental profession should fulfill its ethical mandate and provide health decision makers with relevant information on disease levels. The PUFA index was developed in response to that need. The various clinical stages defined by PUFA have different associations with health conditions. The index defines four different clinical stages of advanced caries providing ‘a face of the reality’ to the prevailing and often ignored oral conditions. Presenting data based on the PUFA index will provide health planners with relevant information, which is complementary to the DMFT. This was an opportunity to validate the new PUFA index under field conditions in school children (11 -14years) in a low-income population in Ungogo local government of Kano state, with a population suffering from a high burden of untreated caries. The index proved to be appropriate in quantifying the consequences of severity of tooth decay as it is universally applicable in all settings, even under simple field conditions. The index is easy and safe to use,

even for non – dentists, takes little time to perform and does not require any additional equipment.

The use of the PUFA index in the Ungogo School children of Kano state has shown the relevance of this index to address the neglected problem of untreated caries and its consequences. Furthermore PUFA data may be used for planning, monitoring and evaluating access to emergency treatment and exposure to fluoride as components of the Basic package of oral care, national oral health plans, National Health Insurance Scheme (NHIS) and may have a higher potential than the DMFT to get oral health into political agendas.

6.1. RECOMMENDATIONS

- The Kano state government should provide at least one fully equipped dental clinic in each local government area with the full complement of dental personnel and ensure adequate funding for dental capital projects.
- The state and local governments should develop sustainable programmes for oral health promotion. There should be a concerted, focused and coordinated campaign to change the perceptions of dental health which has to be launched among the populace to address this very poor standard of oral health in Kano State in the Northern part of the country.
- The goals of this campaign would Promote Oral health, Improve quality of life especially children, Eliminate Oral health disparities.
- Organization and management of Oral Health Services with increased access by the rural populace

- Development of a monitoring system for management of Oral health programmes and personnel should be established to make the oral health services flexible and responsive to changing needs.
- Provision of promotive and preventive oral health care services integrated into the primary health system of the state
- There should be provision for oral health education to school children and the community as a whole in order to promote their awareness of involvement and participate in oral health activities.
- To identify target group easily available for Oral health education such as school children
- State based educational programmes using all available facilities like radio, television, mosques and churches should be utilized to mobilize community support for dental health and ensure its full participation.
- Further research in traditional practices that may influence oral health especially in children.

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APPENDIX I

PARENT/GUARDIAN INFORMATION SHEET

I am undergoing residency training in Family Dentistry at the Aminu Kano Teaching Hospital, Kano State. As part of my training, I am conducting a research on **Dental Caries and PUFA Index in Children age 11 – 14 years in Ungogo Local Government area in Kano State.** This information sheet serves to provide you with the relevant information regarding the study and the study topic.

The study is important to increase knowledge and awareness on dental caries among children. If you agree to participate, you will be asked simple questions and there is no risk in participating in the exercise. Participation is voluntary; your refusal to participate will not affect you in any way. Information is kept for the purpose of the research and will be held in strict confidence.

DENTAL CARIES

Dental caries is a term given to an oral disease that results in the destruction of tooth tissue usually presenting as a hole in a tooth. It is also known as 'tooth decay'. It is one of the most common oral diseases and is a major cause of tooth loss.

WHAT DO WE WANT FROM YOU?

For the purpose of this study, we wish to examine your children/wards mouth to determine the presence and extent of dental caries (tooth decay). We will record our observations in our files and your children/wards can return to their school activities after answering a few questions for our records.

WHY IS THIS STUDY IMPORTANT?

i. For prevention of development of caries thereby prolonging the teeth's lifespan
ii. Proper identification of carious teeth and prevent further deterioration
iii. Serve as a record of dental health needs of children in your community
iv. The study may be used to solicit for establishment of dental clinics or hospital in your community

IS THERE ANY RISK INVOLVED?

There is no risk involved with participating in the study. There will be no invasive procedures. We will only examine your wards/children's teeth and then ask questions. No form of treatment will be given in this study.

If you agree to participate in this study, please sign the consent form attached.

Dr. Sani Balarabe Auwalu

The researcher

APPENDIX II

ASSENT FORM

I ……have been fully informed about the study titled “Dental Caries and PUFA Index in Children age 11 – 14 years in Ungogo Local Government area in Kano State” and have agreed to participate in the research with the right to opt out anytime.

I am aware the study will be simply to examine the mouth to determine the presence and extent of dental caries (tooth decay). Any observations would be recorded and I return to my school activities after answering a few questions in the questionnaire. No risk involved

Information obtained would be confidential and the outcome of the research would help in the prevention of dental caries and treatment to prevent further deterioration.

Non – participation in this study would not affect me in any form.

Name of participant…….

Signature/thumb prints of participant……

Date…….

School……..

APPENDIX III

INFORMED CONSENT FORM

Title of investigation…….

……..

Purpose of study……...

……..

Procedure…….

…….

Discomfort and risks……

…….

Alternate procedure (if any)……

Potential benefits……

……

Period of time required……..

Contact person……

……

I FURTHER UNDERSTAND THAT I AM FREE TO WITHDRAW MY CONSENT AND TERMINATE MY (CHILD OR LEGAL WARD) PARTICIPATION AT ANY TIME.

_____	_____	_____
Date	Date of Birth	Subject's Signature

I hereby consent to the participation of……..my legal dependent, in the scientific investigation described.

_____

Signature of parent or guardian

_____

Date

I, the undersigned, have fully defined and explained the research investigation to the above subject.

_____	_____
Signature	Name of Investigator

_____

Date

This is to certify that I……hereby agree to (give permission to have my child or legal dependant) participate as a volunteer in the scientific investigation as an authorized part of education and research programme of the Aminu Kano Teaching Hospital, Kano Nigeria under the supervision of…… the investigation and my part in the investigation (my child or my legal dependant's part in the investigation) have been defined and fully explained to me by this investigator (s) and I understand this explanation. A copy of the procedure of this investigation and description of any risk and discomfort has been provided and described in details by me.

I have been given an opportunity to ask whatever question(s) I have had and all such questions and enquiries have been answered to my specific items or questions in interviews or medical history questionnaires.

I understand that any data or answers to questions will remain confidential with regard to my identity (or that of my child or legal dependant(s).

I certify that to the best of my knowledge and belief, I have fully disclosed (my child or legal dependant has) any physical or mental illness or weakness that would increase the risk to me (him or her) from participating in this investigation.

AMINU KANO TEACHING HOSPITAL

P. M. B. 3452, ZARIA ROAD, KANO. (☎: 064 - 947872)
FAX (064) 663354, www.akth.org, E-mail: enquiries@akth.org, email: (akthkano@yahoo.com)

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NNOM, OON, FAS, MD, FRCP	MBBS, FWACP, (Int. Med) DCP (Lond.)	MBBS, FMCP Path	B. Ed, CHPM

NHREC/21/08/2008a/AKTH/EC/227

OUR REF: AKTH/MAC/SUB/12A/P3/IV/730 DATE: 23rd June, 2010

Dr. Balarabe Sani
Department of Dental and Maxillofacial Surgery

Ufs:
The Head of Department,
Department of Dental and Maxillofacial Surgery
AKTH,
Kano.

RE: ETHICAL APPROVAL

RE: DENTAL CARIES IN CHILDREN 11 – 14 YEARS IN UNGOGO LOCAL GOVERNMENT IN KANO STATE

Further to your application in respect of the above titled research proposal and the subsequent response, the Committee considered your proposal and noted same as a prospective study.

In view of this, Ethical approval is hereby granted to conduct the research.

However, the proposal is subject to periodic reporting of the progress of the study and its completion to the Committee.

Best regards.

Bara'atu Kabir (Mrs)
Secretary
For: Chairman, Ethical Committee.

QUESTIONAIRE

DENTAL CARIES AND PUFA INDEX IN CHILDREN AGE 11 – 14 YEARS IN UNGOGO LOCAL GOVERNMENT AREA IN KANO STATE

School …… Serial No: ……

SECTION I: DEMOGRAPHICS

Age …… Sex ……

SECTION II: ORAL HEALTH ATTITUDES

1. **Do you like visiting the dentist**

    i. No because I have no problem

    ii. No because I am afraid the dental treatment will hurt

    iii. No because the dental clinic is too far away

    iv. No because I can't afford the cost

    v. No I don't need to because I have few teeth

    vi. No I don't have time

    vii. No because the workers are not friendly

    viii. No because no one to take me there

    ix. Yes

    x. Others

2. **When you are sick what do you do first**

    i. My parents buy drugs from the chemist and treat me

    ii. I am taken to the herbalist or medicine man first

    iii. I am taken to the hospital first

    iv. I am taken to the spiritualist first

v. Others

3. **Is care of the teeth and gum important?**

    i. It is important to care for the teeth and gum

    ii. No, diseases of the teeth are natural in life

    iii. No, children have few teeth so they have no teeth/gum problem

    iv. No, teeth problems are only for white people

    v. No, I don't usually have problems with my teeth/gum

    vi. Others

4. **It is not necessary to clean my teeth regularly every day?**

    i. I agree with this statement

    ii. I disagree with this statement

5. **Do you consider infection of the gum seriously?**

    i. Not necessarily

    ii. I have no idea

    iii. I consider it seriously

    iv. Others

6. **Do the types of foods you eat affect your teeth and gum?**

    i. Yes

    ii. No

7. **When your tooth is aching what would you like the dentist to do for you?**

    i. Extract (remove) the aching tooth

    ii. Drill and fill the aching tooth

    iii. Extract the aching tooth and put a new or artificial one

iv. Clean my teeth

v. Others

8. **Do you prefer your tooth to be filled?**

i. No it is easier for the dentist to extract it and put a new one

ii. No I don't like fillings because it takes too much time

iii. I don't like fillings because I can't stand the noise of the drill

iv. I don't like fillings because the hole is bigger when its filled

v. I don't know anything about dental treatment

vi. Yes

vii. Others

9. **Do you like your teeth to be cleaned and washed in the dental clinic?**

i. Yes, it will be cleaner and feels very smooth

ii. Yes so that I wouldn't need to clean my teeth

iii. Yes it makes my gum healthier

iv. No I have no problem with my teeth and gum

v. I don't know about dental treatment

vi. Others

10. **Is it normal for children to lose their teeth?**

i. It is normal

ii. It is not normal

11. **Is it normal for children to have gum problems?**

i. It is normal

ii. It is not normal

SECTION III: ORAL HEALTH KNOWLEDGE

1. **Sweet foods like sugar, coca cola, sweets etc can cause tooth decay**
    i. I agree with the above statement
    ii. I disagree with the above statement
    iii. Others
2. **For a healthy teeth & gum I need to eat fish, meat, fruits, vegetables, beans, eggs**
    i. I agree with the above statement
    ii. I disagree with the above statement
    iii. What I eat does not affect the health of my teeth and gum
    iv. Others
3. **For my teeth and gums to be healthy I need to visit the dentist every six month**
    i. I agree with the above statement
    ii. I disagree with the above statement
    iii. Others
4. **For my teeth and gums to be healthy I need to brush my teeth with toothbrush or chewing stick a minimum of two times every day**
    i. I agree with the above statement
    ii. I disagree with the above statement
    iii. Others
5. **Diseases of the gum can be caused by**
    i. Eating garlic
    ii. Eating sugary food
    iii. Dirt, stains and teeth tartar on the teeth

iv. Others

6. **Tooth decay can be caused by**

    i. Eating garlic

    ii. Eating sugary food

    iii. Dirt, satins and teeth tartar on the teeth

    iv. Little worms in the tooth

    v. Others

SECTION IV: ORAL HEALTH BEHAVIOUR

1. **What do you use when cleaning your teeth?**

    i. Chewing sticks

    ii. Chewing sponge

    iii. Ash and cotton wool or foam

    iv. Toothbrush and toothpaste

    v. Toothbrush and soap

    vi. Foam with water or cotton wool with water

    vii. Both toothbrush with toothpaste and chewing sticks

    viii. Foam or cotton wool with soap

    ix. Only water

    x. Others

2. **How many times do you clean your teeth either with toothbrush and paste or chewing stick or others?**

    i. Not at all

    ii. Not everyday

iii. Once daily

iv. Twice daily

v. More than twice daily

vi. Others

3. **If you are using stick do you brush your teeth with it?**

i. Yes

ii. No

4. **Do you eat fresh fruit?**

i. Not at all

ii. Once a week

iii. More than once a week

iv. More than three times a week

v. Others

5. **Do you eat fresh cooked vegetables?**

i. Not at all

ii. Once a week

iii. More than once a week

iv. More than three times a week

v. Others

6. **Do you eat meat, fish, eggs, beans (protein food)?**

i. Not at all

ii. Once a week

iii. More than once a week

iv. More than three times a week

v. Others

7. **Do you visit the dentist regularly for check up every six months?**

   i. I visit the dentist regularly every six months

   ii. No because I have no tooth problem

   iii. Others

8. **When was the last time you went for check up/treatment at the dental clinic?**

   i. My last visit was ……

   ii. I have never been to the dental clinic

9. **I eat sweet foods (chocolate, sweets, biscuits, soft drinks, tea, pap etc) regularly**

   i. Everyday

   ii. Not at all

   iii. Once a week

   iv. More than once a week

   v. More than three times a week

   vi. Others

SECTION V: DMFT INDEX

1. **Teeth present**

7	6	5	4	3	2	1	1	2	3	4	5	6	7
7	6	5	4	3	2	1	1	2	3	4	5	6	7

2. **Decayed teeth (D)**

7	6	5	4	3	2	1	1	2	3	4	5	6	7
7	6	5	4	3	2	1	1	2	3	4	5	6	7

3. **Missing teeth (M)**

7	6	5	4	3	2	1	1	2	3	4	5	6	7
7	6	5	4	3	2	1	1	2	3	4	5	6	7

4. **Filled teeth (F)**

7	6	5	4	3	2	1	1	2	3	4	5	6	7
7	6	5	4	3	2	1	1	2	3	4	5	6	7

5. **DMFT Score** =

SECTION VI: PUFA INDEX

1. **Pulp Involvement (P)**

7	6	5	4	3	2	1	1	2	3	4	5	6	7
7	6	5	4	3	2	1	1	2	3	4	5	6	7

2. **Ulceration (U)**

7	6	5	4	3	2	1	1	2	3	4	5	6	7
7	6	5	4	3	2	1	1	2	3	4	5	6	7

3. **Fistula (F)**

7	6	5	4	3	2	1	1	2	3	4	5	6	7
7	6	5	4	3	2	1	1	2	3	4	5	6	7

4. **Abscess (A)**

7	6	5	4	3	2	1	1	2	3	4	5	6	7
7	6	5	4	3	2	1	1	2	3	4	5	6	7

The Researcher would like to thank you for your time and help