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Epidemiology: It's Application in Periodontics

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1. Introduction

From immemorial time man has been interested in trying to cure and/or control of periodontal diseases. In view of the limitations of several theories (i.e., 'Focal infection theory', 'Theory of contagion' and 'Germ theory of disease'), scientific thought began to search the other factors or causes in the etiology of periodontal diseases namely social, economic, genetic, environmental and behavioral factors. Thus, a newer concept (Multi-factorial causation) of periodontal diseases has been evolved by investigators. Recently, evidence has also shed light on the relationship between systemic health and periodontal diseases, that is, possible adverse effects of periodontal disease on a wide range of organ systems (i.e., cardiovascular, endocrine, reproductive, respiratory). Hence, the application of epidemiology in the field of periodontics has utmost importance to measure prevalence, extent and severity of periodontal diseases, its relationship to other factors (age, oral hygiene, and nutrition), to assess the degree of association between periodontal diseases and systemic health and to improve treatment modalities for the prevention and control of periodontal diseases.

The word 'Epidemiology' (Epi- among, demos- people, logos- study) is derived from the term 'epidemic'. 'Epidemiology' is defined as the study of the distribution and determinants of health related states or events in population and the application of this study for the prevention and control of health problems (Last, 1983). Epidemiology is more often concerned with the well being of society as a whole rather than the well being of individual. Three most important components of epidemiology are study of disease frequency (incidence/prevalence), study of disease distribution (i.e., age, sex, race) and (3) Study of determinants (causative/risk factor) of disease. Etiologic (causative) agent is defined as the living/nonliving substances or forces which may initiate or exaggerate the disease process by its excessive presence or relative lack. Risk factor is a subjective determinant of some disease processes (periodontitis, cancer) when true disease causing agent is not fully established. In epidemiologic study, three types of causal relationship are identified between different variables and manifestation of disease (Brownson, 1998) as they are shown in Fig. 1. Variable is a characteristic that helps to measure changes of disease processes varies from person to person. It may be dependent/ uncontrolled variable (i.e., age, genetics) or independent variable that can be controlled or manipulated (i.e., smoking). The presence of risk factor does not imply that always disease will occur and in its absence, disease will not occur. In a disease, they are additive/synergistic, observable/identifiable, can be modified or non-modifiable. The basic aims of epidemiology are: (1) to explain

distribution and magnitude of disease in population, (2) to identify causative/risk factors of disease, (3) to assess the risk in population (4) to study the complete course of disease, (5) to provide the data essential for treatment planning (6) implementation of programme for prevention and control of a disease and finally (7) to promote, protect and restore health of population. Dentist is concerned with the disease of a patient, where as, epidemiologist is concerned with the disease patterns in whole population and to determine preventive or control measures.

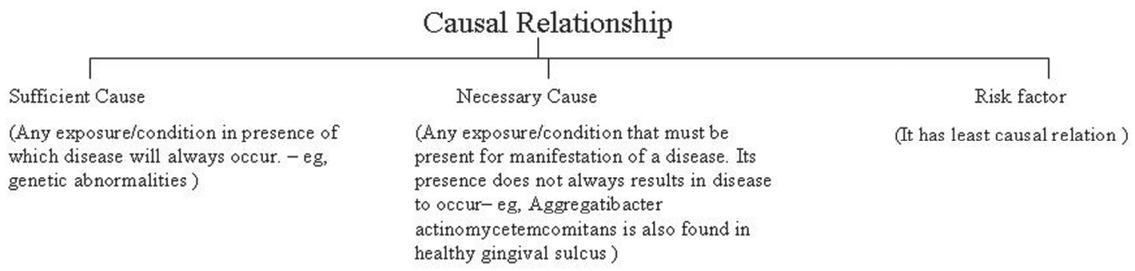

Fig. 1. Different types of causal relationship between variables and disease.

2. Tools for measurement of epidemiology

Incidence is defined as the number of new cases of a disease occurring in a defined population during a given time period (Park, 2002). It is measured as number of new cases of a disease during specific time/ population at risk x 1000 (X/1000/year). Prevalence is the number of cases (old/new cases) of a disease within a specific point of time (point prevalence) or over a given period of time (period prevalence) in a designated population. Point prevalence is more commonly used. When population is stable, incidence and duration are not changing then the relationship between incidence (I) and prevalence (P) can be expressed as, $P = I \times D$ [D = mean duration]. As longer the disease process, prevalence will be increased (i.e., chronic periodontitis, tuberculosis) whereas in acute short lived cases, incidence rate will be higher than prevalence rate. Incidence and prevalence rate are used to assess the magnitude of communal health problems, to identify potential high risk population and useful for administrative and planning purposes. Usually two types of epidemiological methods (i.e., observational & experimental) are used in periodontics to assess different variables and control measures of diseases.

a. Observational studies-
I. Descriptive- eg, cross-sectional study and longitudinal study
II. Analytical- a) Case-control/Retrospective study
b) Cohort /Longitudinal/Follow-up study
b. Experimental studies-
I. Randomized control trial, II. Community trial, III. Field trial

Descriptive study: Descriptive study is the first phase of an epidemiological investigation. In periodontology, it is concerned with the occurrence and distribution of periodontal diseases in human populations and identifies the variables related with the disease. The variables most frequently examined in descriptive studies for periodontal diseases are time related, place related (urban/rural, geographical comparisons) and person related (age, sex, stress, social status, education etc.) characteristics. By comparing the distribution of

periodontal diseases with the help of cross-sectional (prevalence assessment) and longitudinal design (incidence assessment) in different populations, it is possible to set hypotheses relating to disease etiology. The hypotheses can be accepted or rejected with the further application of analytical epidemiology. Cross-Sectional Study (i.e., also known as "prevalence study") is an observational study based on single examination of a cross section of population at given point of time. It provides gross idea about the defined population when sampling has been done correctly. Longitudinal Studies in which observations are repeated in the same population over a period of time by means of follow up examinations. Longitudinal studies are useful to study the natural course of disease and its future outcome, to identify the etiologic/risk factors and to find out the incidence rate which can not be achieved by cross-section study. Cross-sectional study is like a photograph whereas longitudinal study can be considered as a cine film.

Case-Control (retrospective) Study: It has three distinct features: i) exposure and disease have occurred before starting of study, ii) Study proceeds backward from effect to cause, and iii) control group is used to compare the study group.

Risk Factor (smoker)	Cases (periodontitis + ve)	Control (periodontitis -ve)
< 10 cigarette/day	33(a)	55 (b)
Non-smoker	2 (c)	27 (d)

Table 1. Frequency of periodontitis in smoker and non-smoker.

Frequency of exposure to cigarette for cases are (a/a+c) 94.2% and with control (b/b+d) 67.7%. If the frequency of smoking is higher in cases than control (non periodontitis), a association is said to be existed between smoking and periodontitis and vice versa. If 'p' value (statistical association) is less than 0.05, the association is regarded as statistically significant but does not imply causation. Exposure rate of 94.2% does not mean that all the smokers would develop periodontitis. In the case-control study, Odds ratio (measure of strength of the association between risk factor and disease) is the common end point. In Table 1, Odds ratio (ad/bc) is 8.1. It means that there is 8 times greater risk of smokers to develop periodontitis than non smokers. It is a key parameter in the analysis of case-control study which is rapid, in expensive and easy to carry out.

Cohort Study: It is a forward looking, observational study to obtain additional supportive evidence about the existence of association between suspected cause and disease. It is also called prospective, longitudinal, incidence study. In this study, cohorts are identified before appearance of disease, study groups are observed over a period, and study proceeds forward from cause to effect and establish a firm relationship between exposure and disease. Cohort is defined as a group of people who share a common characteristic or experience (age, exposure to drug) with in defined time period. It is indicated when association between exposure and outcome is already established by a case control study. In case control study, exposure and disease have already occurred when the study is initiated, where as in cohort study exposure had occur but disease has not. In general, both groups should be equally susceptible to disease, all the variables should be compared that influences disease frequency and all groups are followed under same condition over a period to determine the exposure.

Relative risk (Incidence among exposed / incidence among non-exposed) is exactly determined by cohort study because incidence rate in the case-control study is not accurate.

It is a direct and reliable measure of strength of association between exposure and outcome. If, it is 1 or less, indicates no association whereas >1 indicates positive association between cause and effect. It is '2' means, two times higher incidence rate of disease in exposed group than unexposed group and 100% increase in risk. 0.25 means less chance of disease in exposed person. Larger the relative risk, greater will be the strength of association between disease and the suspected factor which can be considered as a risk factor for the disease. Relative risk may alter as a result of bias (systematic error in the determination of association) that should be removed by matching (age, sex, etc.) in cohort study. The natural history of the disease is a key concept in epidemiology and is best established by cohort study.

Randomized control trial: To guard against different biased conditions (memory bias, observer bias), 'Single Blind Trial' (patient unaware), 'Double Blind Trial' (doctor and patient unaware about the group allocation and treatment received), and Triple Blind Trial (ideal, where doctor, participant and data analyzer unaware) are adopted. It is used in preventive and clinical trial, trial of etiological agent and evaluation of effectiveness of health services. In Fig.2, the strength of association increases in progressing toward the peak of the pyramid and simultaneously number of biases decrease. 2 or more systematic reviews (i.e., summary of multiple research studies investigated for same parameters) and meta-analysis (i.e., merging of statistical values of multiple studies into one analysis) provide strong causal relationship between risk factor and disease. The association is defined as the occurrence of two variables more often than would be expect by chance. Association does not always imply causal relationship. Correlation is the degree of association between characteristics or variables. Correlation coefficient ranges from -1 to +1. Correlation does not imply causation but causation always implies correlation. '1' indicates perfect association between two variables, '>0.75-1' implies high correlation, '0.75-0.4' for moderate correlation and < 0.4 indicates none or weak correlation. '0' means no association and '-1' indicates perfectly opposite relation.

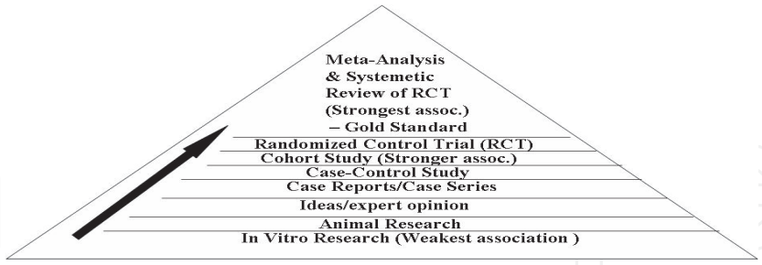

Fig. 2. Strength of association increases toward the peak of the pyramid.

2.1 Sensitivity and specificity

A less accurate and inexpensive screening test is applied by observer on a group of healthy population for an unrecognized disease (ELISA for blood donor). Diagnostic test is applied in a single sick person (ELISA for suspected patient), which is more accurate and expensive. Validity refers to what extent the test accurately measures the variables. It has two components; sensitivity and specificity. Sensitivity is defined as the ability of a test to identify maximum true positive and minimum false negative results. 90% sensitivity means, 90% diseased patients screened by a test will give true positive and 10% a false negative

results. Specificity is defined as the ability of a test to identify maximum true negative and minimum false positive cases or results. In fact, no screening/diagnostic test provides 100% specificity and 100% sensitivity. Sensitivity come in expense of specificity (inversely related). Two or more tests are required to enhance the sensitivity and specificity of the screening programme. Predictive value depends on sensitivity, specificity and prevalence. A highly sensitive test will be rarely false negative when someone has the disease. So clinician should choose it for screening during routine examination. As a highly specific test rarely gives false positive results, it is indicated when a positive results may harm to a person emotionally, physically or financially. Usually a highly sensitive test is done first to rule out non-diseased persons then a highly specific test is advocated to rule out false positive patients.

2.2 Statistical averages

Mean is simply the arithmetic mean of data. It is the most frequently used value for data analysis and presented along with the standard deviation. For the data 1, 2, 3, 4, the mean will be 2.5, but it is usually presented as 2.5 ± S.D [Standard Deviation]. 30 or more samples should preferably be analyzed to get acceptable value of SD. Greater variation of mean values and low sample size of a test provides higher value of standard deviation, which may hamper the test of significance. Median and mode have limited use in periodontology.

3. Concepts of prevention

Successful prevention of disease depends upon knowledge of causation, transmission, availability of prophylactic and therapeutic measures etc. It has four levels:

Primordial prevention: It is a type of primary prevention in present form. For example, many adult health problems (i.e., periodontitis, diabetes, cardiovascular disease) have their early origins in childhood when lifestyles are formed. Efforts are directed toward discouraging children from adopting harmful lifestyles (food, habits, smoking etc.) through proper education and motivation.

Primary prevention: Action taken to prevent the onset of a disease which removes the possibility that disease ever occurs. Intervention is taken during pre-pathogenesis phase of disease. Example- pit and fissure sealant application, plaque control instruction, daily tooth brushing and flossing, fluoridation/ defluridation of water, vaccination.

Secondary Prevention: Action which halts the process of disease at its incipient stage and prevents complications. Interventions are early diagnosis and treatment. In case of infectious disease, it provides secondary prevention to infected individual and primary prevention to community. Example- filling, oral prophylaxis by dentist, gingivectomy.

Tertiary prevention: Action taken to reduce or limits suffering, impairments and disabilities and to promotes rehabilitation. Intervention taken at let pathogenesis phase. Example- Root canal treatment, removable and fixed partial denture, extraction, dental implant retained removable and fixed prostheses.

4. Dental (periodontal) epidemiology

Dental epidemiology is the study of distribution and dynamics (time, pattern, etiologic agent) of dental diseases in human population. The periodontal disease epidemiology is one of the most important but complex part of dental epidemiology because pathologic changes in periodontal diseases involve both soft and hard tissues and there are so many subjective

variation in objective measurement of periodontal indices like color change, pocket depth and swelling. In order to measure the incidence, prevalence and severity of periodontal diseases, its relationship to other factors and for assessment of treatment needs, special indices have been designed to provide objective measurement of identifiable features. It is a quantitative science and is measured by biostatistics. Using these indices and applying the appropriate statistical tests should allow the observer to make a valid comparison of periodontal disease conditions in respect to different variables and to measure the efficacy of therapeutic agents.

4.1 Indices used to assess gingival inflammation

4.1.1 Papillary Marginal Attachment Index

The first dental index, Papillary Marginal Attachment (PMA) Index was developed to count number of gingival unit affected with gingivitis that correlated with severity of gingival inflammation (Schour & Massler, 1948). The facial surface of gingiva around a tooth divided into three units: Mesial interdental papilla (P), Marginal gingiva (M), and Attached gingiva (A). Presence or absence of inflammation on each gingival unit recorded as 1 or 0 respectively. Summation of these three units of a tooth is considered as PMA score of the tooth and summation of score of all teeth and divided by number of teeth; is considered as PMA score of the person. Usually central incisor to second premolars was examined. In 1967, they added severity component for assessing gingivitis likewise- Papillary unit - 0-5, Marginal gingiva - 0-3, Attached gingiva - 0-3. It is used for epidemiological survey, in clinical trials and for patients' education.

4.1.2 Gingival Index

Gingival Index (GI) was developed to assess the severity and quality of gingival inflammation in individual or population (Loe & Silness, 1963). Only gingival tissue is assessed by this index. Blunt periodontal probe is used to assess and palpate the bleeding tendency by running the probe along the soft tissue wall of the entrance of gingival sulcus. Gingiva surrounding the tooth divided into 4 scoring units- Mesio-facial papilla, Facial marginal gingiva, Disto- facial papilla, Lingual marginal gingiva (to minimize examiners' variability in scoring, lingual gingiva were not subdivided). All 4 scoring units are examined by visual examination (dental mirror) and periodontal probe and scored from 0-3 for each of them. Gingival index may be used for selected or all teeth. The scoring criteria are 0 (normal), 1 (mild inflammation, slight color change, slight edema, no bleeding on palpation), 2 (moderate inflammation, redness, edema, bleeding on probing), and 3 (severe inflammation, marked redness & edema, tendency to spontaneous bleeding). The GI score of 4 units are totaled and then divided by 4 (surfaces) to yield the GI score of a tooth. The GI score per person is obtained by totaling all of the tooth scores and dividing by the number of teeth examined (Table 2).

Gingival index score	Degree of gingivitis
0.1-1	Mild
1.1-2	Moderate
2.1-3	Severe

Table 2. Degree of gingivitis in relation to gingival index score.

4.1.3 Modified Gingival Index

Modified Gingival Index (MGI) was introduced with two important changes in gingival index by eliminating sulcus probing, and by redefining the scoring system for mild inflammation to increase the sensitivity of lower values of scoring scale (Lobene et al., 1986). This non-invasive index allows for repeated evaluation of the sites without disturbing the plaque or irritating the gingiva. The scoring criteria are: 0 (Absence of inflammation), 1 (Mild inflammation, slight color change, little change in texture of a portion of papillary or marginal gingiva but not in entire gingiva), 2 (Mild inflammat- ion, change in texture involves entire papillary/marginal gingiva), 3 (Moderate inflam- mation, redness, edema, and/or hypertrophy of marginal or papillary gingiva), and 4 (Severe inflammation, marked redness, edema, and/or hypertrophy, spontaneous bleeding and ulceration). The score of 2 papillary and 2 marginal units are totaled and then divided by 4 (surfaces) to yield the MGI score of a tooth. The MGI score per person is obtained by totaling all of the tooth scores and dividing by the number of teeth examined. Either full or partial mouth assessment can be performed. It perhaps most widely used index in clinical trials of therapeutic agents. This index can not identify the gingivitis in absence of periodontitis because it does not involve pocket probing.

4.1.4 Periodontal Index

After realizing the true paucity of valid index in early 1950's for measuring the prevalence of advanced periodontal diseases; the 'Periodontal Index'(PI) was developed to determine the presence/absence of gingival inflammation, severity of inflammation, periodontal pocket formation and disturbance of masticatory function (Russell, 1956). It not only assesses all the gingival tissues encircling the tooth but also scores the supporting tissues. Mouth mirror, light source and explorer are used to assess tissue. The scoring criteria are: 0 (Absence of inflammation), 1 (Mild inflammation, slight color change, change in texture only a portion of papillary or marginal gingiva but not in entire gingiva), 2 (Mild inflammation involves entire papillary or marginal gingival unit), 4 (when radiograph is advised), 6 (Moderate inflammation, redness, edema, and/or hypertrophy of marginal or papillary gingival unit with pocket formation), and 8 (Severe inflammation, redness, edema, spontaneous bleeding and ulceration with advanced destruction and impairment of function). In doubtful condition, lower score should be considered. The periodontal index score per person is obtained by totaling all of the tooth scores and dividing by the number of teeth examined. It is an index with true biologic gradient because it measures both reversible and irreversible aspects of periodontal disease. It underestimates true level of periodontal destruction and early bone loss. As the number of teeth decrease, the chances of scoring bias will increase.

- 0.0-0.2 (Group PI score) – Clinically normal Reversible stage
- 0.3-0.9 (″) – Simple gingivitis (″)
- 0.7-1.9 - (″) – Beginning of periodontal destruction – (″)
- 1.6-5.0 - (″) – Established periodontal destruction - Irreversible stages
- 3.8-8.0 - (″) – Terminal disease (″)

4.1.5 Gingivitis component of the Periodontal Disease Index

The periodontal disease index (PDI) was developed by Ramfjord SP in 1959, to which few criteria further added in 1967. The PDI is used to measure incidence and prevalence of periodontal disease. This index assesses gingivitis, gingival sulcus depth and plaque at all

interproximal, facial and lingual surfaces on six selected teeth (i.e., Ramford's teeth # 16,21,24,36,41,44) because these teeth have been tested as reliable indicators for various regions of the mouth (Ramfjord, 1959, 1967). The calculus component assesses the presence and extent of calculus on facial and lingual surfaces of indexed teeth. Plaque and calculus component of PDI are not a part of PDI score rather helpful in a total assessment of periodontal status. The selection of indexed teeth may be altered in longitudinal studies and clinical trials, where all teeth or quadrants of teeth or the teeth appropriate for the objective of the study can be chosen. It can be used in large survey because it is quick and easy. The PDI is useful for comprehensive assessment of periodontal status in cross-sectional surveys, longitudinal studies and clinical trials of therapeutic or preventive procedures. The gingivitis index scoring criteria are: 0 (Absence of signs of gingivitis), 1 (Mild to moderate gingivitis, not extending around the tooth), 2 (Mild to moderate gingivitis extending all around the tooth), 3 (Severe gingivitis, marked redness and edema, tendency to bleed, and ulceration). The gingivitis scores per tooth are totaled and then divided by the number of teeth examined to yield the gingivitis score per person. Same measurement method follows for plaque and calculus score. The plaque, gingival sulcus depth and calculus index component will be discussed in separate section.

4.2 Indices used to assess gingival bleeding

4.2.1 Gingival index used by the National Institute of Dental Research (NIDR)

It was developed to assess gingival inflammation (Miller et al., 1987). It has two components: 1) Bleeding index, and 2) Calculus index. The mesio-buccal interproximal and mid-buccal gingiva on all teeth except molars and mesio-buccal interproximal and mid-buccal gingiva on mesial root of the molars are assessed. The sites are randomly determined as one half of the upper arch and contra-lateral side of the lowr arch. NIDR probe marked on 2, 4, 6, 8, 10 and 12 mm with alternating yellow color band are inserted 2mm into the gingival crevice in mid-buccal gingiva and gently drawn in a horizontal direction along the inner wall of the crevice to mesio-buccal interproximal direction. The score for bleeding index is 0 (no bleeding) and 1(bleeding present). The score of bleeding sites are totaled and then divided by the number of sites examined to yield the gingival bleeding index score of a person. Superficial gingival crevice palpation and interproximal cleaning aids are more suitable to assess gingivitis than indices that utilizes apical probing (i.e., useful for diagnosing periodontitis).

4.2.2 National Institute of Dental & Craniofacial Research (NICDR) protocol

The NICDR protocol was first used in The Third National Health and Nutrition Examination Survey (NHANES),1988-94 (NHANES III, 1997). Gingival assessment is one of the components of NIDCR protocol for assessment of periodontium. Similar technique as NIDR, has been used to assess the prevalence of gingivitis in NIDCR protocol but was slightly modified by adding mesio-facial site to the NIDR examination protocol, resulting three sites per tooth. As per NHANES III survey, gingival bleeding was more prevalent with 13-17 years age group (63%), then gradually decreases with increasing age. Adolescents have higher prevalence of gingivitis than pre-puberty and adult may be due to increasing sex hormone level that alters the composition of subgingival microflora and facilitates colonization of increasing level of Prevotella intermedia and Prevotella nigrescence (Nakagawa et al., 1994). Prevalence of gingivitis in males of any age group is higher than

female. It suggests that plaque control in puberty gingivitis is more important than rising level of hormone.

Several other indices are used to assess gingival bleeding such as Sulcus Bleeding Index (Möhlemann & Major, 1958), Bleeding Point Index (Lenox & Kopczyk, 1973), Ainamo's Gingival Bleeding Index (Ainamo & Bay, 1975), Carter's Gingival Bleeding Index (Carter & Barnes, 1974) and Eastman Interdental Bleeding Index (Caton & Polson, 1985).

The association between rate of plaque formation and gingivitis was observed in the classical study of 'experimental gingivitis in man' (Löe et al., 1965), which demonstrated the cause and effect relationship between plaque and gingivitis. 12 individuals (9-dental students, 1-instructor and 2-technicians) were asked to stop from all sorts of oral hygiene measures. Dental plaque increased quickly and all subjects developed gingivitis within 10-21days. It indicated that when brushing was omitted, the formation of plaque and development of gingivitis were closely parallel. Mean GI score increased from 0.27 to 1.05 at the end of 'no brushing period'. Gingivitis was resolved in all subjects within 1 week of reinstitution of tooth brushing. This evidence demonstrated the reversible nature of gingivitis and also showed a concomitant decrease in plaque and gingivitis. They concluded that bacterial plaque was essential in the production of gingivitis. Bleeding on probing can occur as early as 2 days after gingivitis begins in healthy mouth. If plaque and calculus are removed, gingival bleeding and ulceration will heal after 7-10days. If plaque accumulates further; bleeding return back within 2 days. Bleeding on probing from multiple sites in a single examination or from a particular site in subsequent examination is a good indicator of current inflammation at all stages of periodontal disease.

4.3 Indices used to assess periodontal destruction

Alveolar bone destruction is an important criterion for assessing severity of periodontal disease by using crevice measurement, radiographic evaluation of bone loss, assessment of gingival recession and tooth mobility. Radiograph only reveals interdental bone level and is not useful for buccal and lingual assessment of bone level or attachment loss.

4.3.1 Gingival sulcus measurement component of Ramfjord's PDI

This technique has been introduced for determining the gingival sulcus/pocket depth with a calibrated periodontal probe involves measuring the distance from cemento-enamel junction (CEJ) to the free gingival margin (First measurement score) and the distance from free gingival margin to the bottom of the gingival sulcus/pocket (Second measurement score). The subtraction of first measurement score from the second score yields the clinical attachment loss (Ramfjord, 1967). A) If the gingival margin is on the enamel, then the above calculation reveals the level of attachment. B) If the gingival margin is on the cementum, then the first measurement should be recorded as minus score and the second measurement as plus score. Then the clinical attachment level is measured by subtracting the first measurement score from the second score (i.e., addition of two scores). Ramfjord's PDI is still considered as the "gold standard" method for determining the status of periodontium. The first measurement is useful in assessing gingival recession or gain after therapy. In cross-section study, only 6 indexed teeth should be assessed. In longitudinal study and in clinical trial, other teeth can be included according to the objective of the study.

PDI criteria for epidemiologic surveys:
- When CAL = 0, Gingivitis score represents the PDI score of that tooth
- When CAL ≤ 3 in any of the two measured areas of tooth, PDI score of tooth = 4 (Gingivitis score is disregarded)
- When CAL > 3 - ≤ 6 in any of the two measured areas of tooth, PDI score of tooth = 5
- (Gingivitis score is disregarded)
- When CAL > 6 in any of the measured areas of tooth, PDI score of tooth = 6 (Gingivitis score is disregarded)

4.3.2 Extent and Severity Index (ESI)

This index was developed because of a lack of satisfaction with previous indices as PI and PDI do not provide the information about the extent of the periodontal disease (Carlos et al., 1986). PI was based on the concept that periodontal disease was slow growing continuous process. Later on, periodontal disease was viewed as chronic process with intermittent periods of activity and remission that affects individual tooth and sites around teeth at different rates within same mouth. The NIDR probe has been used to estimate percentage of sites affected by attachment loss >1mm (Extent- E) and mean attachment loss (LA) >1mm (Severity- S). 14 sites at upper arch and 14 sites at contra lateral half of lower arch were measured (Mesio-buccal interproximal and mid-buccal of all teeth except molars and Mesio-buccal interproximal and mid-buccal of mesial root of molars). Extent and severity index (ESI) described the distribution of the disease. NIDR includes the method of measurement of ESI but severity component is modified to > 3 mm of attachment loss. In epidemiological studies, measurements are rounded off to the next digit, so >1 mm is written as 2 mm. The percentage of sites examined that have LA > 1 mm represents the extent score, whereas; average LA/site among the diseased sites represents the severity score. ESI (20, 3.0) means 20% of sites had diseased and within diseased site average LA was 3mm. ESI measured for full mouth assessment or as much as sites/tooth.

4.4 Indices used to assess plaque accumulation

4.4.1 Plaque component of PDI (PlI)

It is the first index attempted to assess the extent of plaque quantitatively covering the all four surface areas of Ramford's teeth (Ramfjord, 1959). Bismark brown disclosing agent was used. The PlI scoring criteria are: 0 (absence of plaque), 1 (plaque covering <1/3 of gingival half of facial or lingual surface of a tooth), 2 (plaque covering >1/3 to 2/3 of gingival half of facial and lingual of the tooth), 3 (>2/3 of gingival half of facial and lingual of the tooth). The PlI score per person is obtained by totaling all of the tooth scores and then dividing by the number of teeth examined. This index was modified by excluding interproximal area of tooth and restricting the scoring of plaque to the gingival half of the facial and lingual surface (Shick & Ash, 1961).

4.4.2 Oral Hygiene Index

The Oral Hygiene Index (OHI) is composed of the combined debris index and calculus index (Greene & Vermillion, 1960). The upper and the lower arches are divided separately into three segments (i.e., six sextants): i) the segment distal to the right canine, ii) segment distal to the left canine, and iii) the segment mesial to the right and left first premolars. Each segment is examined for debris or calculus. Debris includes plaque, materia alba and debris

itself. From each segment, buccal and lingual surface of one tooth is used for calculating the individual index for that particular segment. The criteria used for assigning scores to the tooth surfaces for the OHI are described in the OHI-simplified section.

4.4.3 Oral Hygiene Index-Simplified (OHI -S)

Green & Vermillion in 1964 simplified the OHI by including only six teeth surfaces rather than twelve that were representative of all anterior and posterior segments of the mouth. This modification was called Oral Hygiene Index-Simplified (OHI -S). The tooth used for the calculation must have the greatest area covered by either debris or calculus. The method for scoring calculus is the same as that applied to debris. It has two components: debris index - simplified (DI-S) and calculus index – simplified (CI-S). The mouth mirror, and shephard's crook or sickle type explorer are used to examine facial surfaces of teeth # 11,16,26,31 and lingual surfaces of teeth # 36,46, by running the instrument from distal gingival crevice to mesial gingival crevice of a particular surface (½ of tooth circumference) subgingivally. In the absence of selected molars, second or third molar and in absence of selected anterior teeth, the teeth # 21 or 41 is substituted. At least two surfaces must have been examined for an individual score to be calculated. The CI-S score per person is obtained by totaling all of the buccal and lingual calculus scores and then dividing by the number of surface examined. The CI-S scoring criteria are: 0 (no calculus present), 1 (supragingival calculus covering ≤1/3 of exposed tooth surface), 2 (supragingival calculus covering >1/3 but ≤ 2/3 of exposed tooth surface or presence of flecks of subgingival calculus or both), 3 (subgingival calculus covering >2/3 of exposed tooth surface or continuous heavy band of subgingival calculus around the crevice of teeth or both). The debris score of all the buccal and lingual surfaces are totaled and then divided by the number of surface examined to yield the DI-S score of a person. The DI-S scoring criteria are: 0 (no debris or stain present), 1 (soft debris covering ≤ 1/3 of tooth surface or extrinsic stain regardless of area covered), 2 (soft debris covering >1/3 but ≤ 2/3 of exposed tooth surface), 3 (>2/3 surface area is involved). The average individual or group debris and calculus scores are combined to obtain the Simplified Oral Hygiene Index. It has been used extensively throughout the world because the criteria are objective and provide high level of reproducibility. The high degree of correlation (r=0.82) between the PI and the OHI-S helps to calculate the unknown score with regression analysis. The OHI-S is used in epidemiologic surveys, longitudinal studies and to evaluate the level of cleanliness of personal oral hygiene measures. The OHI-S score 0-1.2 of a person indicates good oral hygiene, 1.3-3.0 indicates fair oral hygiene and 3.1-6.0 indicates poor oral hygiene.

4.4.4 Plaque Index (PI)

It is unique among the indices because it ignores coronal extent of plaque and assesses only the thickness of plaque at the gingival area of the tooth using mouth mirror, and sickle type explorer or periodontal probe (Silness & Loe, 1964). As it is developed as a component parallel to the GI (Löe and Silness, 1963), it examines the same scoring units of the teeth (disto-facial/facial/ mesio-facial /lingual). Plaque index does not exclude or substitute a tooth with gingival restoration and crown. The scoring criteria are: 0 (no plaque at gingival area), 1 (a film of plaque on gingival margin and/or adjacent tooth surface, recognized only by running a probe across tooth surface), 2 (moderately soft deposits at margin and/or adjacent tooth surface that can be seen by naked eye), 3 (abundant soft matter at margin and

adjoining surface). The assessment of plaque thickness is so subjective that to obtain accurate data, highly trained and experienced examiners are required.

4.4.5 Patient's Hygiene Performance Index

It is the first index to assess an individual's performance in removing debris after tooth brushing instruction. It records presence or absence of debris as 1 or 0 respectively using 6 surfaces of OHI-S teeth. It is more sensitive than OHI-S as it divides each tooth surface into 5areas: 3 longitudinal thirds and middle third horizontally into thirds. The scoring is done by using disclosing agent and is used for individual patient education.
Another plaque index was focused on gingival 1/3 of the tooth surface of the facial surfaces of all anterior teeth using basic fuschin disclosing agent (Quigley & Hein, 1962).

4.5 Indices used for calculus measurement

- Calculus component of Oral Hygiene Index-Simplified (Green & Vermillion, 1964).
- Calculus component of PDI (Ramfjord, 1959).
- NIDR Calculus Index- It measures the presence or absence of calculus on buccal and lingual surfaces of a tooth using NIDR probe (Miller et al., 1987). The scoring criteria are: 0 (absence of Calculus), 1 (supragingival calculus present), 2 (supra and subgingival calculus present). The calculus index score per person is obtained by totaling all of the teeth scores and then dividing by the number of teeth examined.

4.6 Indices for treatment needs

4.6.1 Gingival Plaque Index (GPI)

The Gingival plaque index (O'Leary et al., 1963) is a modification the PDI of Ramfjord to detect periodontitis at its initial stage so that treatment may be instituted promptly. It measures three components: Gingival status, periodontal status (crevice depth) and collectively materia alba, calculus and overhanging restoration (i.e., irritational index). For the assessment of gingival status, each arch was divided into anterior, left posterior, right posterior segments. Severest condition within each of the 6 segments determines the score of that segment, using the criteria: 0 (normal), 1 (mild to moderate inflammation partially encircled the tooth), 2 (mild/moderate inflammation completely encircled one/more tooth), 3 (marked inflammation, ulceration, spontaneous bleeding, recession and clefts). The gingival score per person is obtained by totaling all of the scores of the segments and then dividing by the number of segment examined.

4.6.2 Periodontal Treatment Need System (PTNS)

This index is considered the presence of gingivitis, plaque and calculus. It determines the presence/absence of periodontal pockets of ≥ 5 mm in each quadrant (Bellini & Gjermo, 1973).

4.6.3 Community Periodontal Index of Treatment Needs (CPITN)

Without knowing the response of the periodontal tissue to initial therapy, estimation of treatment needs may be subject to over/underestimation of what is clinically prudent.
World Health Organization appointed an expert committee to review the methods to assess periodontal status and treatment needs (Ainamo et al., 1982). The index that resulted after extensive field testing by the investigators from the World Health Organization (WHO) and

the International Dental Fedaration (FDI) was called Community Periodontal Index of Treatment Needs (CPITN). It is composed of the combined elements of GPI and PTNS to assess presence/absence of gingival bleeding on gentle probing, presence/absence of supra and/or subgingival calculus and subdivided the periodontal pocket into shallow and deep using WHO periodontal probe (i.e., 0.5mm ball tip and marking at 3.5mm, 5.5mm, 8.5mm and 11.5mm, black color coding between 3.5mm and 5.5mm). In epidemiological study, 10 index teeth are examined but only worst finding from index teeth is recorded per sextant resulting in six scores. It permits rapid examination to determine periodontal treatment needs. However, a great deal of useful information is lost when only the worst score per sextant is recorded. CPITN underestimates the number of pocket > 6 mm in older age group and overestimate the need for scaling in younger age group because the WHO probe has no marking below 3.5 mm (Gaengler et al., 1988).

CPITN score	Periodontal status	Treatment need
0	Healthy periodontium	No treatment
1	Bleeding observed by probing/spontaneous	Improvement of Oral hygiene
2	Calculus felt by probe; entire black area is visible	I+ professional scaling
3	Pocket depth 4-5mm; Gingival margin on the black band	I+ professional scaling
4	Pocket depth>6mm; Entire black band is invisible	I+II+ complex surgery

Table 3. Scoring criteria of community periodontal index of treatment needs (CPITN).

4.7 NIDCR protocol for periodontal disease assessment

It has three components: 1) gingival assessment (Described in the previous section -same as NIDR), 2) calculus assessment (at each site assessed for attachment loss, where calculus should be assessed using the scoring criteria of NIDR), and 3) assessment of periodontal destruction (NHANES III, 1997). The assessment of periodontal destruction includes: i) Loss of attachment, and ii) furcation involvement). The attachment loss is measured at facial and mesio-facial sites of teeth in two randomly selected quadrant using Ramfjord criteria by NIDR probe. Assessment of furcation involvement is done on eight selected teeth # 17,16,24,26,27,36,37,46,47, by using explorer no.17 for upper arch and cowhorn explorer no.3 for lower arch. Extent of furcation is assessed at mesial/facial/distal surface of maxillary molar, mesial/distal surface of maxillary premolar, buccal/lingual surface of mandibular molar. The criteria for scoring furcation involvement are: 0 (no furcation involvement), 1 (partially involved but not through and through involvement), 2 (complete through and through furcation involvement). For greater statistical reliability, combining two or more NHANES survey (2-year cycles) is strongly recommended.

4.8 Reliability of periodontal indices

The term reliability means the ability of an index to provide same results each time measuring a condition in the same subject repeatedly. Since, all measurements are subjected to error/bias (i.e., examination bias, observer bias, time bias) and variability (i.e., intra/inter-examiner), several indices should be used whenever possible. Because

incorporation of unreliable indices in the prevalence estimates (from survey) which reveals significant difference in the comparative study results, become questionable when longitudinal study is performed. None of the periodontal indices are universally accepted. Recording of pocket depth (CAL is disregarded) in CPITN index may lead to underestimating disease severity among older population when gingival recession is prevalent. Therefore, the CPITN is not a reliable epidemiological index for periodontal study (Baelum et al., 1995).

4.9 Prevalence of gingivitis

A gingivitis case is a person with atleast mild inflammation in at least one of the examined gingival units (i.e., anatomic structure of gingiva such as interdental papillae, marginal gingiva or attached gingiva). Plaque induced gingivitis may be found in existing but non progressing attachment loss or stable periodontitis patient. Prevalence and severity of gingivitis increases with age, beginning at 5yrs of age, reaching highest at puberty (80-90%) then decreases but relatively high throughout the life (Stamm, 1986). Recently prevalence of gingivitis reported was less (80-85%) in India (Bhayya et al., 2010) compared to the studies conducted previously (99%). When gender wise prevalence of gingivitis was considered, males were showed poorer periodontal status (84% vs.78.3%) than the females ($p<0.01$) and the reason behind this can be attributed to the habits and consciousness of the females in doing better oral hygiene practices (Mehta et al., 2010). Even allowing for the differences in measurement techniques between the surveys, there appears to have been an improvement in gingival health in recent decades, which might be due to improvement in socio-economic status and education. In the NHANES III Survey, 50% of adults were found to have gingivitis. Whereas, in Sri Lankan tea workers, among whom both oral hygiene and the gingival condition were poorer at all ages, found 89 % cases were progressed beyond gingivitis to periodontitis (Loe et al., 1986). It suggests that higher prevalence and severity of gingivitis in developing countries was associated with extensive plaque and calculus deposits, low socio-economic status and education as compared to the peoples of developed countries. It has frequently been found that some gingival sites make the transition from being periodontally healthy to gingivitis may be due to genetic variability, stress, female sex hormones and higher dosages of oral contraceptives. The interproximal areas of teeth are most severely affected by gingivitis followed by buccal and lingual surface respectively. The interproximal and buccal surfaces of upper arch are more affected by gingivitis than lower arch and the relationship is reversed in the lingual surfaces (Löe et al., 1965). For facial surfaces, the areas most severely affected by gingivitis, in descending order, were the maxillary first and second molars, the mandibular anteriors, maxillary anteriors, maxillary premolars, mandibular first and second molars, and the mandibular premolars. Gingivitis most severely affected in the lingual surfaces, in descending order, were the mandibular first and second molars, mandibular premolars, mandibular anteriors, maxillary first and second molars, maxillary premolars, and the maxillary anteriors.

4.10 Incidence of periodontitis

Incidence of periodontal disease is not only means the onset of new disease in previously disease free adults in strict sense, but also refers to the development of new sites of periodontal lesions in diseased mouth and progression of existing attachment loss (i.e., progression of disease in already diseased sites). Incidence of periodontitis varies according

to the case definition of the disease. The more severe the extent of attachment loss or bone loss that is taken as case definition, the lower will be the incidence of periodontitis (Oringer et al., 1998). Although some cross-sectional studies have confirmed in identifying age as a risk factor for progression of CAL (Papapanou & Wennstrom, 1990) but most of the longitudinal studies have shown progression of CAL is more closely related to the extent of baseline CAL than to age (Beck et al, 1990). A previous disease episode did not put a site at higher risk for a subsequent attack.

4.11 Prevalence of periodontitis

Among the basic clinical measures for periodontitis (bleeding on probing, presence of local factors, probing depth, bone loss), loss of clinical attachment (CAL), a measure of accumulated past disease at a site rather than current activity, remains a "gold standard" diagnostic method for periodontitis. The standard deviation of repeated CAL measurements of the same site by an experienced examiner with a manual probe is around 0.8 mm (Haffajee & Socransky, 1986). Accordingly, change in attachment level in a clinical study needs to be at least 2 mm (i.e., two to three times the standard deviation) to estimate the real change rather than measurement error. Therefore, CAL cut off limit of 1 mm, needs to be increased for the reasons of examiner reliability discussed above. Any prevalence information must be interpreted in light of the population studied and the periodontitis case definition (sites, extent) applied. The older belief was that susceptibility to periodontal diseases was virtually universal. Today, it is well documented that only 5% to 15% of any population suffers from severe generalized periodontitis, even though mild to moderate disease affects a majority of adults (Oliver et al., 1998). Periodontitis was regarded for years as primarily the outcome from bacterial infection. The concept has been changed and the host response is now seen as a key factor for development of periodontitis, which often modified by behavioral and environmental factors (Page et al., 1997). Body's immune system generate inflammatory response in an attempt to protect itself from pathogens but at the same time inflammatory mediators can lead to periodontal connective tissue and bone destruction. In India, prevalence of chronic periodontitis was increased steadily with age from 35% for 35-40 years age group to 85% for 80-90 years old (mean 21-30%), whereas prevalence of aggressive periodontitis was below 1% and the loss of attachment (3 mm or more) was 45-77% in 35-44 year age group and 55-96% in 65-74 year olds (Jacob, 2010). The general trend for loss of attachment observed was higher in rural than in urban Indians and was higher in males compared to females. The previous belief was that higher prevalence and severity of periodontitis existed among populations of developing nations than the developed nations, has not been confirmed by most studies (Baelum et al., 1997). Comparing the groups of Norwegian and Sri Lankan young adults, found strikingly similar rates of periodontal breakdown, despite the last group having much poorer oral hygiene conditions. Clear differences are only apparent in poorer oral hygiene and greater calculus accumulation in even a young age group in populations of developing countries (Loe et al., 1986). Thus, the prevalence and severity of the disease can be considered far more similar between different populations and are confined to small groups at high risk in each population. Prevalence of periodontitis is greater in patients with teeth present in one side of the arch than teeth present in the both side of the arch. Supra gingival calculus is most commonly found on the maxillary first molars followed by mandibular anteriors, and least on maxillary anteriors. Sub gingival calculus is commonly found, in the descending order, on the mandibular central and lateral incisors, maxillary first and second molars, maxillary

anteriors and least commonly found on mandibular premolars and third molars. Supra gingival and sub gingival calculus (i.e., combined measurement) are most commonly found on the mandibular central and lateral incisors followed by the maxillary first molars and least commonly found on mandibular premolars and third molars. In general, severity of periodontitis follows the distribution pattern of subgingival and combined measurement of calculus; thus, incisors and molars are more severely involved than canine and premolar areas (least involvement).

4.12 Risk factors affecting prevalence and severity of periodontal diseases

According to current understanding of the pathogenesis of periodontal disease, it is essential to look at factors that may play a role in the initiation and progression of the disease. The risk factors that cannot be modified (e.g., age, sex, genetics) is often reffered to as determinant. The term risk indicator describes possible correlates of disease identified in case-control studies, and risk factor is best applied to those correlates confirmed in longitudinal (cohort) study and implies a modifiable condition.

4.12.1 Determinants of periodontitis

Age: The prevalence and severity of CAL is invariably related directly to age in cross-sectional surveys (Miller et al., 1987). But the older assumption that periodontitis is a disease of aging is no longer tenable. It is uncommon for elderly people with reasonably good periodontal health to exhibit sudden bursts of periodontitis (Page, 1984). The most rapid disease progression is seen in relatively small number of elderly persons in whom the disease starts at younger age, and there is some evidence that these individuals have some genetic susceptibility to periodontitis. It can be hypothesized that prevalence of the disease increases with age may be due to cumulative periodontal breakdown over time as a result of prolonged exposure to other risk factors (i.e., drug, altered food habits, lack of dexterity to maintain oral hygiene) rather than age related intrinsic deficiency which increases susceptibility to periodontal diseases (Genco, 1986). Although certain physiological changes in the periodontium occur with age but these changes alone are not responsible for periodontal breakdown in the elderly (Van der Velden, 1984). In contrast, age is an important factor for assessment of prognosis. The lesser extent of loss of attachment in younger patients should be considered more detrimental than greater extent of attachment loss in older age groups because younger patients have to be faced longer periods of exposure to offending agents. **Sex**: In one study, after adjusting age, oral hygiene and socio-economic status, males were found to have significantly greater extent of attachment loss and alveolar bone loss compared to females (Grossi, 1995). In another study, it was observed that males had consistently 10% higher prevalence of attachment loss than females (Miller et al., 1987). Increased prevalence and severity of periodontal disease in males are more likely due to less positive attitudes toward oral health, and dental-visit than to any genetic cause. However, the relationship observed between sex and the disease is not considered as strong and consistent. **Socioeconomic Status (SES):** The possible relationship between periodontal disease and socio-economic status was found in many studies (Locker & Leake, 1993). Generally, those who are better educated, rich, and live in more desirable circumstances enjoy better health status than the less educated and poor people. Gingival condition and poor oral hygiene are clearly related to lower SES, but the direct relationship between periodontitis and SES has been poorly established. It was found that the prevalence of CAL

at all levels of severity was not closely related to household income (Miller et al., 1987). On the other hand, severe CAL (≥5 mm) in at least one site was closely correlated with educational levels due probably to better oral hygiene among the educated people (Miller et al., 1987). The racial/ethnic differences in periodontal status have been thought unlikely to represent true genetic differences. **Genetics:** The association between severe periodontitis and interleukin (IL)-1 specific genotype was found only in non-smokers (Kornman et al., 1997), suggested that the genetic factor was not as strong a risk factor as smoking. The strength of association between genetics and periodontitis is still being determined because IL-1 has been identified as a contributory cause of periodontitis among some patients only. However, a combination of IL-1 polymorphism and smoking may provide a good risk profile for patients (McDevitt et al., 2000); therefore, smoking–genetic interaction may be considered as a contributory factor in severity of periodontitis. Further research are needed before concluding remarks on genetic contribution in the initiation and progression of periodontitis, till then, restraining from smoking would be a higher priority than a search for genetic cause. **Race, Place, and History** of **previous periodontal diseases**: The advanced periodontal breakdown have been shown three times more prevalent in blacks than in the whites (Beck et al., 1990). Racial differences in education, socio-economic status and the distribution of genetic factors may also contribute to differences in the prevalence and severity of periodontal disease. Low prevalence of periodontitis-associated interleukin (IL)-1α/IL-1β composite genotype among Chinese suggesting their inherited resistance to develop severe and lower prevalence of periodontitis (Armitage et al., 2000). Prevalence and extent of periodontal disease is slightly more in rural areas but is not likely due to progression of current disease.

4.12.2 Risk factors

Plaque, Microbiota, and Oral Hygiene: In earlier studies, strong positive correlation has been found between poor oral hygiene and periodontal diseases (Greene & Vermillion, 1964). Bacteria that accumulate in dental plaque are primary causative agents of gingivitis. A study demonstrated the inverse relationship of meticulous oral hygiene practice (brushing frequency) with that of level of periodontal disease and tooth loss in patients with periodontal pocket (Merchant et al., 2002). But poor oral hygiene does not imply that all patients would be suffered from periodontitis rather the relationship is less straightforward. It has been shown that colonisation of virulent bacteria is necessary, but not sufficient to initiate the disease process. There is an interaction of bacterial factors with other favourable host and environmental factors which may dramatically modify the disease expression (Page et al., 1997). So poor oral hygiene is an important risk factor in susceptible persons and is of less important in individual with strong host resistance. Longitudinal data are available which dictates neglected dental care increases the prevalence and severity of periodontal disease. Frequent professional supragingival cleaning and good personal oral hygiene have been shown to have a beneficial effect on subgingival microbiota in shallow to moderately deep pockets (Westfelt, 1996). These finding form an evidence base for control of supragingival plaque as part of periodontal therapy. The resistance of host and other factors such as smoking and some systemic diseases are recently thought to overweigh the role of bacterial pathogens in the pathogenesis of periodontitis. Tissue destruction may be initiated and progressed by both direct and indirect effects of bacteria plus the effects of the altered host defence system. **Tobacco:** Smoking is clearly a risk factor for chronic periodontitis,

independent of oral hygiene, age, or other factors but its role in gingivitis is unclear (Ismail et al., 1983). The risk of periodontitis attributed to smoker in the order of 2.5 to 6.0 or even higher compared to its nonuse and risk increases with increasing in frequency of exposure (Bergstrom & Preber, 1994). Exactly how it acts in the causal chain is still unclear. It has been stated that 90% of persons with refractory chronic periodontitis are smokers (Johnson & Slach, 2001). The healing following periodontal treatment is slower in smokers may be due to inhibition of growth and attachment of fibroblasts in the periodontal ligament and in slower reduction of white blood cells at diseased sites after therapy (Christan et al., 2002). Earlier studies showed no difference in prevalence of periodontal pathogens subgingivally (Preber et al., 1992), but more recent evidence suggests that smoking appears to promote a favorable habitat for pathogenic species in shallow pockets (Haffajee & Socransky, 2002). Decreased bleeding on probing in smokers might be due to suppression the vascular reaction by nicotine and compromising host response to infection. In experimental plaque-induced gingivitis, despite the rate of plaque accumulation being equal in smokers and non-smokers, the increase in gingival vascularity in smokers was only half of that seen in the non-smokers (Bergstrom et al., 1988). This is a masking effect on the signs of inflammation and should be considered while gingival bleeding is assessed. Some studies have confirmed that smoking suppresses hemorrhagic response (Bergstrom & Bostrom, 2002). However, others have found no difference in the extent of BOP between smokers and non-smokers despite the smokers having deeper pockets (van der Weijden et al., 2001). Recent studies suggest that inflamed sites in smokers have reduced vascular density and angiogenesis compared to inflamed sites in nonsmokers, thus impairing inflammatory response and wound healing (Rezavandi et al., 2002). Therefore, further study is needed on how smoking affects gingival bleeding. Smoking inhibits granulocyte function (chemotaxis, phagocytosis) and interactions between smoking and the IL-1 genotype-positive alleles in the progression of CAL, have also been indentified (Meisel et al., 2002). Smoking aggravates all tissue-destructive diseases (periodontitis), by stimulating the production of TNF-α and various tissue degrading cytokines (Fredriksson et al., 2002). Smoking has also been shown to be a stronger risk factor for periodontitis than insulin-dependent diabetes mellitus (Moore et al., 1999). The evidence is clear that smoking is a major risk factor for periodontitis. **Systemic factors:** One of the strongest systemic factor for high prevalence and extent of periodontal disease is uncontrolled diabetes mellitus. Both the Insulin and non-insulin dependent diabetics appear to be equal risk for periodontal disease. Not only poor glycemic control is the significant risk factor for periodontal disease but it has also been suggested that effective periodontal therapy in adjunct to systemic antibiotics can have a positive effect on the control of diabetes. A substantial body of evidence suggested a bidirectional relationship between both types of diabetes and periodontal disease (Taylor, 2001). Other diseases such as HIV infection, osteoporosis, and cardiovascular disease also showed an association with periodontal diseases but exactly what relationship exists is still unknown (Nunn, 2003). **Stress:** The psychological factors (financial strain, death of relative, negative life event, examinee, military life etc.) have been proposed as risk factor for periodontal disease. Psychological factors are thought to adversely affect the host immune response and disrupt homeostasis by releasing indigenous catecholamine and steroid hormone. In other way, emotional status of poor coping individual may lead to negligence in performing oral hygiene practices, dry mouth, changes in diet habits, increased smoking and bruxism, which making the individual more susceptible to oral diseases (Monteiro da Silva et al., 1998).

Several studies evaluated the effects of emotional stressors on periodontal health and reported significant increases in mean plaque score, subgingival calculus, bleeding on probing, pocket depth, attachment loss, bone loss, and tooth loss (Moss et al., 1996). Aggressive periodontitis and necrotizing ulcerative gingivitis (NUG) are the periodontal conditions most frequently associated with psychological stress. Significantly elevated cortisol level was observed in urine with NUG patients and that returned to normal after recovery (Cohen-Cole et al., 1983). Those individuals with more psychological stress were less responsive to periodontal therapy (Axtelius et al., 1998). The exact pathological link between stress and periodontal destruction, however, has not yet been established but are probably related to impaired immune function and altered oral health behaviors. In view of the successful treatment of NUG even in presence of the stressful condition or continued smoking (Cohen-Cole et al., 1983), the association is not sufficient to assume a causal relationship between these two conditions. It strongly suggests that stress has a limited role as an etiologic factor for periodontal disease. **Nutrition**: There are no nutritional deficiencies that by themselves can cause gingivitis or periodontal pockets. Most of the information regarding association between nutrition and periodontal diseases are primarily based on animal studies and few human reports that involved severe nutritional deficiencies (Pitiphat et al., 2002). Minor nutritional imbalance failed to show any effect on periodontal health. Validation of nutrition as a risk factor for periodontal disease requires longitudinal study designs to assess the timing between the deficiency and the onset of the disease. However, it is a difficult task to set an experimental design in human because nutritional requirement and food habit changes as one progresses from birth to elderly as well as due to ethical reasons. **Obesity:** Obesity may be considered as an unique form of malnutrition. A significant association was observed between higher body mass index and periodontal disease (attachment loss) that might be mediated via insulin resistance (Grossi et al., 2000). Relative risk increases 1.3 times with each 5% increment of body fat, after adjusting age, gender, oral hygiene status and smoking history (Siato et al., 1998). However, more researches are needed before obesity can be considered as risk factor for periodontitis. **Tooth factors:** Various abnormalities of tooth anatomy (enamel projection, enamel pearl, external root grooves) have been shown to be associated with furcation involvement. In addition, abnormal positioning of tooth (crowding, extreme labial or lingual positioning, open contact, occlusal discrepancy), overhanging restoration margin, subgingival crown margin have been implicated as strong predictor of periodontal breakdown (Nunn, 2003).

4.13 Risk assessment in periodontology - A new perspective

Recent research has demonstrated that some individuals or groups of individuals experience more severe form of periodontal disease than others. Therefore, the rationale for the risk assessment in periodontology is to target appropriate levels of prevention and care for high risk individuals (Stamm et al., 1991). The aim is to identify the presence of some easily measured entity by which clinicians would predict the risk of future disease with high reliability. The current understanding of periodontal diseases has put a further fundamental step in risk assessment for the disease (Page & Beck, 1997). A risk factor must be a part of the causal chain and criteria of identifying a risk factor are to be met only in longitudinal studies, by which the disease outcome can be compared to the baseline measures. The clinical measures of plaque and calculus do not predict the future disease to any useful extent (Badersten et al., 1990). The subgingival presence of specific periopathogens has

shown a moderate degree of predictability. It is now recognized that host response, smoking and genetic predisposition (IL-1 genotype) have major role in this regard. The multiple predictors work better than any one single predictor (except smoking- universal predictor) for the risk assessment. However, enough advances in our knowledge about risk factors yet to be made to permit the development of a risk calculator to help assess a patient's risk of disease.

5. Aggressive periodontitis

The primary features of aggressive periodontitis include a history of rapid attachment and bone loss with familial aggregation. Secondary features include phagocyte abnormalities and a hyperresponsive macrophage phenotype. Localized aggressive periodontitis (LAgP) patients have interproximal attachment loss on at least two permanent first molars and incisors, with attachment loss on no more than two teeth other than first molars and incisors. Generalized aggressive periodontitis (GAgP) patients exhibit generalized interproximal attachment loss including at least three teeth that are not first molars and incisors (Armitage, 1999). The onset of these diseases is often circumpubertal. With time, the localized form appears to be self-limiting ('burn out' of the disease), or may progress to GAgP with increasing age (Gunsolley et al., 1995). The incidental attachment loss should be excluded before diagnosing a case of aggressive periodontitis, in which one or more teeth had greater than 3mm attachment loss, but were not met the criteria for AgP. Reported estimates of the prevalence of LAgP and GAgp in geographically diverse young populations were ranged from 0.1% to 15% (average < 1 %), and 0.03% to 0.59% (average 0.13%) respectively (Marazita et al., 1994). In NIDR survey of adolescent (14-17 years of age), it was estimated that 0.53% had LAgP and 0.13% had GAgP and 1.61% had incidental loss of attachment and the teeth most severely affected in descending order were first molar, second molar, incisors (Löe & Brown, 1991). They reported that males had slightly higher but statistically insignificant prevalence of LAgP and GAgP. In Afro-Americans, prevalence of LAgP in male was 2.9 times more than in female, whereas, among Whites, females were 2.5 times more prone to LAgP than the males (Löe & Brown, 1991). The findings of several studies have suggested the fairly equal distribution of the disease between genders (Saxby, 1984). When genders were examined among the races, then gender differences were much more evident. A study of aggressive periodontitis involving different ethnic groups estimated the prevalence of AgP in Afro-Americans was 0.8%, Whites 0.02% and Asians 0.2% (Saxén, 1980). In general, blacks are more susceptible to AgP than the Whites. The prevalence rate among gender is followed, in descending order, as black male, black female, white female and white male. The age group mostly affected by AgP is between puberty to 30 years of age. Not all patients infected with *Actinobacillus a0.ctinomycetemcomitans* (Aa) develop LAgP and not all patients with LAgP have detectable levels of Aa (Lang et al., 1999). To date, moreover, no single species is found in all cases of LAgP. A variety of functional neutrophil defects have been reported in 70-75% patients with LAgP. These include anomalies of chemotaxis, phagocytosis, bactericidal activity, superoxide production, FcγRIIIb (CD16) expression, leukotriene B4 generation, Ca^{2+} channel and second messenger activation, abnormally low number of chemoattractant receptors and an abnormally low amount of cell surface glycoprotein GP-110 (Van Dyke et al., 1990). Adherence receptors on neutrophils and monocytes, such as LFA-1 and Mac-1, are normal in LAgP patients. Neutrophilic chemotactic defect is genetic in origin which predisposes individual to LAgP and that is the cause why the disease run in family. Not all LAP patients have neutrophilic

chemotactic defect and not all neutrophilic chemotactic defect patients have LAgP (Van Dyke et al., 1990). Therefore, other unidentifiable host factor likely to be involved in the pathogenesis of AgP. GAgP, can begin at any age and often affects the entire dentition. Individuals with GAgP exhibit marked periodontal inflammation and have heavy accumulations of plaque and calculus. Neutrophils from patients with GAgP frequently exhibit similar functional defects as observed in LAgP. The antibody response, and the clinical manifestations of aggressive periodontitis are modified by patients' genetic background as well as environmental factors such as smoking (Califano et al., 1996). The two forms of aggressive periodontitis can be considered to be different diseases unlike chronic periodontitis and appear to be associated with somewhat different subgingival bacterial profiles, difference in the number of affected teeth or pattern of damage and have separate genetic risk factors (Armitage, 2010). The '1999 World Workshop on the Classification of Periodontal Diseases' recommended deletion of age-dependent terms such as adult and juvenile periodontitis (Armitage, 1999). Nevertheless, age is still an important characteristic that can be useful in differentiating between chronic and aggressive forms of periodontitis. The loss of attachment in aggressive periodontitis (approximately 1–2 mm/year) patients progressed three or four times faster than in cases of chronic periodontitis (Average 0.2 mm/year), which serves as an important characteristic to distinguish clinically both the form of the disease (Baer, 1971). The mechanisms and regulation of bone loss associated with all forms of chronic or aggressive periodontitis appear biochemically, immunologically and histologically similar with respect to the molecular mediators and pathological processes. However, there are differences in the speed at which bone loss occurs (Bartold et al., 2010). There are no striking differences in risk factors between aggressive and chronic periodontitis, although the associated gene defects may be different (Stabholz et al., 2010). The general pattern of normal random migration and impaired chemotaxis in aggressive but not in chronic forms of periodontitis, could be due to a reduction of GP110 and f-Met-Leu-Phe surface receptors on neutrophils. The mode of inheritance of aggressive periodontitis is probably autosomal dominant among the African-American and Caucasian (Marazita et al., 1994). A strong familial influence has been observed on the prevalence of both the chronic and aggressive periodontitis. In the Japanese population, a polymorphism of the Fc-γRIIIb (CD16) was described in patients with both forms of periodontitis (Loos et al., 2003). Therefore, it can be suggested that no genetic risk factors or markers are able to distinguish between aggressive periodontitis and chronic periodontitis. The other environmental factors (smoking, oral hygiene, stress, obesity) have no uniqueness to either generalized aggressive periodontitis or chronic periodontitis. Oral hygiene, as assessed by plaque levels, is directly associated with disease severity in both entities, except in the localized form of aggressive periodontitis. Systemic diseases cannot be considered as risk factors for aggressive periodontitis. However, systemic diseases that can cause subtle perturbations in host susceptibility to infections (e.g. diabetes mellitus), can alter the clinical course of both chronic periodontitis and aggressive periodontitis.

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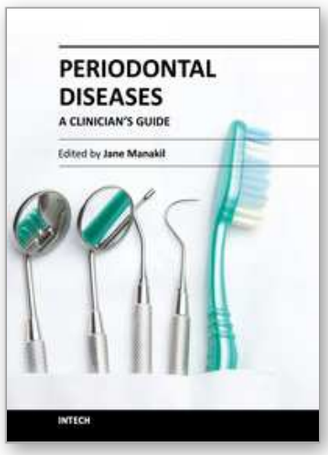

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"Periodontal diseases" is a web-based resource intended to reach the contemporary practitioners as well as educators and students in the field of periodontology. It is fully searchable and designed to enhance the learning experience. Within the book a description is presented of the current concepts presenting the complex interactions of microbial fingerprint, multiple genotypes, and host modulations. In addition, an overview is given of the clinical outcome of the disease's progression, as influenced by the epigenetic factors. Emerging concepts on periodontitis as a risk factor for various systemic diseases and as a bilateral modulating factor have been elucidated in detail as well.

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