Epidemiology of Dental Caries and Periodontal Disease in the "Third World"

Dental Fluorosis - Nature, Mechanisms and Dose-Response Relationship in Man

Course material for postgraduate training in
"Dentistry in third world countries"

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For too long it has been assumed that we know too little about dental diseases in developing countries. In reality there exists today a large data base important not for its size but for the fact that these data permit an advancement of our understanding of the disease processes involved. As such information emerging from these countries make important contributions to science and are not therefore to be considered as rareties or peculiarities particular to the problems of "developing nations".

Much of these data have highlighted issues central to the control of dental diseases and are therefore of central importance for those involved in primary health care.

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V. Baelum
Royal Dental College
Aarhus
Denmark

O. Fejerskov
Royal Dental College
Aarhus
Denmark

F. Manji
IDRC Regional Office
Nairobi
Kenya

THE DISTRIBUTION OF PERIODONTAL DESTRUCTION IN POPULATIONS IN NON-INDUSTRIALIZED COUNTRIES: EVIDENCE FOR THE EXISTENCE OF HIGH RISK GROUPS AND INDIVIDUALS

Vibeke Baelum[1], Firoze Manji[2] and Ole Fejerskov[3].

[1] Dept. of Periodontology and Oral Gerontology, Royal Dental College, Aarhus, Denmark

[2] International Development Research Center, Regional Office for Eastern and Southern Africa, Health Science Division, Nairobi, Kenya

[3] Dept. of Oral Anatomy, Dental Pathology and Operative Dentistry, Royal Dental College, Aarhus, Denmark

[1,3] WHO Collaborating Centre for Oral Health Planning and Research in Third World Countries

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INTRODUCTION

The concept of groups or individuals being at high-risk or of different susceptibility to periodontal destruction has recently attained considerable attention. For many years it has been generally held that in any population virtually every person would be equally susceptible to periodontal breakdown if the standard of oral hygiene was poor. Thus, it was concluded that the combined effect of age and oral hygiene standard could explain more than 90% of the variation in periodontal disease experience (Sherp, 1964). Likewise, Russell (1967a) found that the association between oral cleanliness and periodontal disease "is so strong as to leave very little variation in disease scores to be accounted for by any factor or factors independent of age or hygiene". According to this concept the predominant character of a high risk person would therefore be an unfavourable combination of old age and poor oral hygiene. However, more recent studies have indicated that risk factors, in addition to those of age and oral hygiene, are needed to explain the patterns of periodontal destruction observed in various populations (Hugoson & Jordan, 1982; Cutress, Powell & Ball, 1982; Beck et al., 1984; Löe et al., 1986; Baelum, Fejerskov & Karring, 1986; Baelum, Fejerskov & Manji, 1988a). Thus, the effect of age on the periodontal status as well as on the incidence of periodontitis may be neglible if a high standard of oral hygiene is maintained (Burt, Ismail & Eklund, 1985; Abdellatif & Burt, 1987). Moreover, only a small fraction of a population may

experience periodontitis of a severity which may lead to tooth loss, despite gingivitis being common (Hugoson & Jordan, 1982; Beck et al., 1984). Such observations have lead to the proposal of the existence of risk factors, other than oral hygiene per se, which may account for these distributions (Hugoson & Jordan, 1982; Beck et al., 1984). Furthermore, there is equivocal evidence for the role of oral hygiene in periodontal destruction in different forms of periodontitis, such as rapidly progressive periodontitis (Page et al., 1983) and juvenile periodontitis (Baer, 1971). In fact, one of the cardinal features of juvenile periodontitis is that the amount of destruction observed is incommensurate with the amounts of microbial plaque present (Page & Schroeder, 1982). Perhaps most striking are the findings that even in populations where the standard of oral hygiene is very poor and gingivitis extremely widespread it is nevertheless relatively uncommon to find destructive periodontal disease of a severity which may endanger the longevity of the dentition (Cutress et al., 1982; Baelum et al., 1986; Baelum et al., 1988a; Chen et al., 1989).

A further understanding of the patterns of distribution of destructive periodontal disease and the possible existence of high-risk groups to periodontal destruction is probably best obtained through studies of populations in non-industrialised countries. In such populations formal dental health care services are scarce and the use of various therapeutic measures known to interfere with the natural course of the disease processes, such as antibiotics (Douglass et al., 1983), is minimal. The present paper

considers the evidence for the existence of high-risk populations, groups, or individuals to periodontal destruction based on the data available from such populations in non-industrialised countries.

RISK ASSESSMENT AND PERIODONTAL DESTRUCTION: GENERAL CONSIDERATIONS

Risk is the probability of an event occurring within a specified period of time (Last, 1983). A high-risk individual is, therefore, an individual with a higher probability of an event occurring within a specified period of time. This higher probability is mediated through the acquisition of, or exposure to, one or more risk factors. A risk factor is biologically (or causally) related to the occurrence of the event (Kleinbaum, Kupper & Morgenstern, 1982). A risk factor thus contrasts with a risk indicator, or marker of risk, which may be highly associated with the event because it signals presence of - or exposure to - risk factors, but which in itself is not causally related to the occurrence of the event.

The above definitions have, albeit indirectly, touched upon the very key areas of interest for most of the current studies in the field of periodontal epidemiology. In common with a number of problems arising in medical epidemiology (e.g. diarrhoeal diseases) periodontal destruction is characterised by recurrent episodes. However, one of the distinguishing features of destructive periodontal disease is the possibility of simultaneous occurrence of breakdown

in a number of sites within any given individual, and, furthermore, the sites may experience varying extents of destruction.

This "multiple sites - varying extent" character of periodontal destruction clearly poses many analytical problems for risk-assessment studies (Imrey, 1986). The unit of measurement is the site whereas the unit of interest is the individual or even groups of individuals. The basic observations made at the site level are often condensed into summary figures for the individual or the group of individuals, resulting inevitably in loss of important information since the dimensions of extent and severity (dimensions that are not necessarily interdependent (Carlos, Wolfe & Kingman, 1986)) are not given any expression in aggregate statistics.

Two further problems arise for epidemiologic studies of destructive periodontal disease. First, it should be appreciated that in a hypothetical situation in which all individuals in a population or group are homogeneous with respect to all risk factors the response variable (i.e. periodontal destruction) will not be exactly the same because of random effects, i.e. there will be some individuals in the "tails" of the distribution. The existence of such tails, therefore, is not <u>a priori</u> evidence for the existence of differences in risk. Secondly, the problem is compounded by the fact that parameters such as loss of attachment or pocket depths are in reality continuous variables. Often attempts are made to simplify the issue of risk assessment by "arbitrarily" deciding upon

a cut-off point in order to dichotomize the response variable. Clearly, the cut-off point chosen will affect the conclusions to be drawn.

We shall not here deal with the issue of risk assessment in relation the individuals diagnosed as having prepubertal or juvenile periodontitis or as having severe generalised immune disorders. The mere presence of severe periodontal destruction at an early age makes prepubertal and juvenile periodontitis patients clearly distinguishable from their periodontally healthy counterparts (Page & Schroeder, 1982). The information on risk assessment that derives from the study of such special and severe forms of periodontal destruction will be dealt with elsewhere (Page, this meeting).

"Severe disease for age" as a measure of risk

The literature on the epidemiology of destructive periodontal disease in non-industrialised countries is dominated by cross-sectional studies. Longitudinal studies have seldomly been performed (Löe et al., 1978; 1986) and the data hitherto presented does not allow for an assessment of the variation in the changes of periodontal disease experience over time. The evidence for the existence of high-risk groups or individuals has, therefore, to be evaluated based on cross-sectional studies, usually comprising measures of periodontal status in relation to age. The most commonly acknowledged limitation for the interpretation of data from cross-sectional studies is the

influence of cohort effects (Last, 1983). However, what has rarely been recognised is the fact that the recording of, for example, loss of attachment at any given point of time does in itself not tell anything about the rate at which the condition has developed. Figure 1a illustrates one such situation which may arise from observing cross-sectional data. Let us consider two persons A and B of the same age who are observed to have the same amount of loss of attachment. From the point of view of observed disease experience for age we can conclude that both individuals had the same risk of developing that amount of periodontal breakdown. In drawing such a conclusion, however, we cannot make any assumptions that the pathway taken was the same in both individuals. Thus, the hatched line in Fig. 1a indicates that person B may have begun loosing periodontal attachment at a much later period in life but then experienced rapid breakdown such that, at the time of the cross-sectional study, both A and B were observed to have experienced the same amount of periodontal breakdown. The problem is further compounded by the fact that even had the pathways taken been known we would not be able to assume that in the future the breakdown in person B would continue to be more rapid than in person A. Figure 1b illustrates a similar scenario arising from observing cross-sectional data. In this figure the persons A and B have, at the time of the cross-sectional study, experienced the same amount of periodontal breakdown, but person B is considerably older than A. We may therefore conclude that A has experienced a higher risk of developing that amount of breakdown than has

B. As illustrated by the hatched line, however, the progression of breakdown may have been the same for both A and B, merely starting at a later time in B. Again, based on cross-sectional data no inferences can be made as to the possible pathways previously followed by different individuals nor can we make any assumptions about future pathways. Fig. 1c illustrates that the same arguments may be used for a situation in which A and B are of the same age but where A has experienced substantially more breakdown than B. Therefore, the observation of a communality in the degree of periodontal destruction in persons of the same age is not a priori evidence for a homogeneity of the exposure to risk factors. Conversely, of course, a heterogeneity of the degree of periodontal destruction is not a priori evidence for a heterogeneity of risk - an issue which we shall deal with later. From the figures 1a-c it may be seen that the measure "disease for age" is a measure of the combined effect of "duration" of the exposure to risk factors (represented in the figures by the x-axis intercepts) and the "virulence" or "quality" of the risk factors (represented by the slopes). "Disease for age" may thus be expressed as a product of "duration of exposure" and "virulence of exposure" although the relative magnitude of these components remains unknown. The measure "severe disease for age" cannot, therefore, be interpreted as a measure of prior, current, or future probabilities of changes in the degree of periodontal destruction within a specified (short) period of time.

Although the interpretation remaims uncertain, the "severe disease for age" measure is nevertheless the only measure of risk to periodontal destruction which may be evaluated from the cross-sectional epidemiological studies available. Assessment of the risk to periodontal destruction and the causes of this increased risk is therefore intimately linked to evaluation of the observed variations of past cumulative disease experience within as well as between populations.

Evaluation of variations in disease experience within and between populations

There is often a lack of clarity as to the importance of making distinctions between within population and between population variations in disease experience. When we compare the variation in disease experience between populations we are attempting to ascribe such variations to attributes of each population, that is, characters shared by each member of each population. However, when we seek to explain the variation in disease experience within a single population we are seeking attributes of individuals within that population. In most published epidemiological studies of destructive periodontal disease it is not possible to evaluate the variations within a population of the disease experience. Thus, the reporting of group mean values or the provision of one estimator of extent or severity of disease has been the most common way of presenting results. This renders the evaluation of the distribution of the periodontal scores within a population almost impossible.

Knowledge of the distributional characteristics of the periodontal disease scores is a cardinal requirement for evaluation of high risk groups or individuals within a population. Since only very few studies are available pertaining to non-industrialised countries which fullfil these criteria the evidence for the existence of high-risk groups and evaluation of their features has to be assessed based on only a few studies. When considering between population variation of disease experience as a measure of risk to periodontal destruction it is extremely important to bear in mind that sources of variation exist which cannot be attributed to variations in risk. These sources of variation comprise "errors" arising from use of different measurements, different interpretation of the criteria employed, differences between examiners etc. It is therefore mandatory to consider the possible effects of these "extraneous" sources of variations on the results obtained in epidemiological studies of destructive periodontal disease before conclusions are drawn with respect to the risk-status of different populations.

THE IMPACT OF DIFFERENT CRITERIA AND MEASUREMENTS USED FOR ASSESSMENT OF THE RISK OF PERIODONTAL DESTRUCTION.

The effect of changing the concepts of periodontal diseases.

The perception of periodontal disease has changed over the years. It is now widely agreed that destructive periodontal disease is "an inflammatory disease of the periodontium characterized by the presence of periodontal

pockets and active bone resorption with acute inflammation" (Page & Schroeder, 1982). This condition should be distinguished from gingivitis in epidemiological studies if usefull information about the prevalence, severity and distribution of destructive periodontal disease within and between populations is to be obtained (Page & Schroeder, 1982). However, this concept has only recently become widely accepted. For many years the term periodontal disease has been used as a generic name for any "inflammatory process affecting one or more of the supporting tissues of the teeth - the gingival tissue, the periodontal membrane, and the alveolar bone" (Scherp, 1964). Although the distinctions between gingivitis and destructive periodontal disease were appreciated these disease processes were considered to represent different stages of the same disease entity and a large number of the methods and criteria used for assessing periodontal disease in epidemiological studies have reflected these concepts.

The most predominant of such methods are the Russell Periodontal Index (PI) (Russell, 1956), the Periodontal Disease Index (PDI) (Ramfjord, 1959) and the Community Periodontal Index of Treatment Needs (CPITN) (Ainamo et al., 1982). The use of these recording systems comprize recording of, in a hierachical manner, gingival inflammation as well as either pocket depths (PI and CPITN) or loss of attachment (PDI). Whereas a full mouth recording is recommended for the use of the Russell PI (Russell, 1956) the PDI and the CPITN use partial recordings of selected teeth (Ramfjord, 1959; Ainamo et al., 1982).

Such recording systems do not, however, provide data of sufficient detail to enhance our understanding of the processes involved in the loss of periodontium (Ramfjord, 1974) and in the field of clinical trials of destructive periodontal disease measurements of loss of attachment have for many years been mandatory. This concept has gradually been adopted also for population based epidemiologic studies. Thus, in recent years a number of epidemiological studies have emerged in which the degree of periodontal destruction has been assessed using detailed measurements of loss of attachment (Löe et al., 1978, 1986; Cutress et al., 1982; Ånerud et al., 1983; Gadegaard & Fejerskov, 1983; Scheutz, Heidmann & Poulsen, 1983; Baelum et al., 1986, 1988a,b; Tirwomwe et al., 1988). Other methods of assessment which may be encountered in the literature include measurements of pocket depths (MacGregor & Sheiham, 1974; Ainamo & Ainamo, 1978; Speake & Malaki, 1982; Klausen & Fanöe, 1983; Lembariti, 1983; Sardo-Infirri, 1984; Matthesen et al., 1989) and radiographic assessment of bone destruction (Marshall Day & Shourie, 1949; Cutress et al., 1982; Budal, Henrikson & Nordenram, 1985). These assessment may be made on a full mouth basis or be restricted to selected teeth and surfaces.

Can assessments made using different methods be used for evaluating the risk-status of different populations? Comparing PI with CPITN

A comparison of the results obtained using the Russell

PI with those obtained using the CPITN in exactly the same individuals has shown that both the estimated prevalences as well as the distributional characteristics of the periodontal conditions differ depending on the method used (Cutress, Hunter & Hoskins, 1986). Using the Russell PI, 10% of the persons examined were classified as being "periodontitis" or "advanced disease" cases, whereas using the CPITN 24.2% were classified as having pockets (Cutress et al., 1986). These results are quite remarkable because both recording systems use pocket depth measurements as the basis for the diagnosis of "periodontitis" or "advanced disease". Furthermore, the CPITN findings are based on the assessment of 10 teeth only, in contrast to the full mouth recordings performed in the PI, and it should therefore be expected that the PI would find more disease than the CPITN. Equally important, however, whereas 28.5% of the individuals were classified as "healthy", according to the assessment using the CPITN, only 8.1% could be so classified using the PI (Cutress et al., 1986).

Comparing PI with PDI

The major difference between the criteria of the Russell PI and those of the Ramfjord PDI is that the PI includes pocket depth measurements whereas the PDI includes measurements of loss of attachment. Sheiham & Striffler (1970) found that 51.1% of the persons examined may belong to the "healthy" or the "gingivitis" categories, when assessment is made using the Russell PI, but demonstrate signs of destructive disease when assessed using the PDI.

These findings show, as might be expected, that assessment based on the recording of pockets underestimates the disease experience.

Comparing measurements of loss of attachment with pocket depth measurements

The findings of Sheiham & Striffler (1970) are further supported by the findings of Aucott & Ashley (1986) and Carlos, Brunelle & Wolfe (1987). Hence, Carlos et al., (1987) concluded that "the use of indices based upon pocket depth measurements alone may result in underestimates, and conceivably large underestimates, of the amount of destructive periodontal disease which has occurred". Moreover, the use of pocket depth measurements as indicative of periodontal destruction may yield distributions different from those observed using loss of attachment measurements. Thus, the presence of deepened periodontal pockets overestimates loss of attachment in the age groups until about 35 years whereas the absence of deepened pockets underestimates loss of attachment from the age of about 25 years (Baelum et al., 1988a).

Comparing full mouth recordings with recording of selected teeth or sites.

Ainamo & Ainamo (1982) reported a 33-68% underestimation of the prevalence of pockets when comparing the Ramfjord selection of index teeth with full mouth recordings and a 50% underestimation of the proportion of surfaces with deep

pockets. Similar findings have been reported by Downer (1972) and Ainamo & Ainamo (1985). The CPITN selection of teeth overestimates the proportion of sites with pockets by 50-100%, depending on the pocket depth considered (Ainamo & Ainamo, 1985; Aucott & Ashley, 1986) whereas the prevalence of deep pockets (≥ 6 mm) may be underestimated by 20% (Ainamo & Ainamo, 1985). The use of random half-mouth examinations seem to result in a 25% underestimation of the prevalence of pockets > 6 mm and a 10% overestimation of the mean number of teeth with such deep pockets whereas the proportion of teeth with deep pockets is accurately estimated (Hunt, 1987). When half mouth examinations are employed (Kingman et al., 1988) the prevalence of loss of attachment ≥ 4 mm and ≥ 7 mm may be underestimated by up to 35%, depending on the general disease level for the population examined, and the prevalence of pockets ≥ 4 mm may be underestimated by up to 21%. This underestimation will increase when less surfaces per tooth are assessed (Kingman et al., 1988). Depending on the population in question and the types of sites selected for examination, the average pocket depth and the average loss of attachment may be either underestimated or overestimated (Kingman et al., 1988).

Comparison of different examiners

The results obtained by different examiners using the same criteria may differ highly. Ufortunately, very little is known about between examiner variations when periodontal conditions are assessed using index systems. The data

provided by Russell (1956) do indicate differences between examiners amounting to as much as 30% of the maximum PI score assigned. These results were, however, obtained in children with very low PI scores, and the magnitude of between examiner variation in population groups with higher PI scores remains unknown. The mean pocket depth observed in the same individuals by different examiners may differ by up to 0.65 mm (Abbas et al., 1982; Badersten, Nilveus & Egelberg, 1984) and the mean loss of attachment observed may differ between examiners by about 0.50 mm (Badersteen et al., 1984). Although these between examiner differences have been estimated in clinical trial settings, the results corroborate the differences that may be calculated from the population based epidemiological data presented by Baelum et al., (1988a).

The importance of such between examiner differences is perhaps best illustrated by a comparison of the findings of three studies of the occurrence of chronic periodontitis among English schoolchildren using the same radiographic criteria for the presence of periodontitis. Two of the studies reported a prevalence of chronic periodontitis within the same order of magnitude, 51.5% and 44.0%, respectively, (Hull, Hillam & Beal, 1975; Davies, Downer & Lennon, 1978) whereas the third study reported a prevalence of less than 1% (Blankenstein, Murray & Lind, 1978). Although the individuals included in these studies were not the same, the question arises whether differences of this magnitude in a very young population were real and may

therefore illustrate the impact of the interpretation of criteria by different examiners upon the results obtained.

Conclusion

These large variations make it impossible to draw valid conlusions about the relative risk status of populations that have been examined using different criteria by different examiners. Therefore, the results of epidemiological studies of different populations should not be compared if the populations have been examined using different criteria. This argument may, moreover, be extended such that studies of the same population at different points of time cannot be used for making inferences about putative changes over time if the studies have been performed using different criteria. In the following, therefore, the evidence for the existence of high-risk groups to periodontal destruction in non-industrialized countries will be evaluated such that comparisons will only be undertaken between populations that have been examined using the same criteria.

EVIDENCE FOR THE EXISTENCE OF HIGH-RISK GROUPS TO DESTRUCTIVE PERIODONTAL DISEASE IN NON-INDUSTRIALIZED COUNTRIES

Studies using the Russell Periodontal Index (PI)

Most of our knowledge of the periodontal conditions in populations in non-industrialised countries stems from studies in which the Russell PI (Russell, 1956) has been

used. Table 1 summarises the results of a number of such studies. It is evident that the severity of periodontal disease, as assessed using the PI, apparently varies considerably between various populations in non-industrialised countries. Nevertheless, comparisons with results obtained in populations in industrialised countries have lead to the conclusion that the prevalence and severity of periodontal disease is much higher among populations in the non-industrialised part of the world. Thus, most populations in the industrialized countries have been considered to constitute a relatively healthy "lower extreme" whereas populations in Asia and Africa have been considered as high "extremes" with South American populations representing intermediate stages (Waerhaug, 1966). This apparently higher susceptibility to periodontal diseases has been interpreted as resulting mainly from poorer standards of oral hygiene (Waerhaug, 1966). However, a number of other factors such as nutritional status, anemia, genetic factors, general health status, vitamin deficiencies, tobacco consumption and betel chewing have been proposed to constitute significant additional risk factors which might explain the apparently higher prevalence and severity of periodontal disease observed in non-industrialised countries (Littleton, 1963; Emslie, 1966; Russell, Consolazio & White, 1961; Russell et al., 1965; Russell, 1962; 1963b; 1967a; Barros & Witkop, 1963a,b; Waerhaug, 1967; Enwonwu & Edozien, 1970; Johansen, 1970). The results of these studies have however, at the very best,

demonstrated only weak associations between the putative additional risk factors and periodontal disease status (Russell, 1967a). These "negative" findings have thus lent further support to the conclusion that age and oral cleanliness are by far the main determinants for periodontal disease (Russell, 1967a).

As previously indicated the implications of this conclusion is that age and oral hygiene standards are the only risk factors or determinants of periodontal status. However, some of the epidemiologic studies available, in which periodontal conditions have been assessed using the Russell PI, do nevertheless indicate that the effect on the periodontium of age and poor oral hygiene may differ between populations. Figs. 2a-g demonstrate the relationship between age, mean PI and the mean oral hygiene index value in seven different populations. It should be noted that the measure of oral hygiene in Figs. 2a-c is the OHI-S (Greene & Vermillion, 1964) whereas the OHI (Greene & Vermillion, 1960) has been used in Figs. 2d-g. The figures first of all illustrate that the age-specific severity of periodontal disease, as assessed using the Russell PI, does not unanimously vary with the degree of industralisation. Thus, the PI values observed in the British population studied by Sheiham (1969) (Fig. 2f) are very high. The most remarkable feature of these diagrams is, however, the relative positions of the lines demonstrating the age-specific PI values to the lines demonstrating the age-specific OHI values, respectively. While the U.S. population (Fig. 2a) (Johnson et al., 1965) and the Equador/Montana population

(Fig. 2b) (Greene, 1963) demonstrate similar levels of PI, the Equador/Montana population has considerably higher oral hygiene scores than the U.S. population. The oral hygiene scores are similar in the Equador/Montana and the Vietnamese population (Fig. 2c) (Russell et al., 1965) but the Vietnamese population demonstrates much higher PI scores. This might be indicative of a lower susceptibility to the effects of poor oral hygiene in the Equador/Montana population as compared to the U.S. population and the Vietnamese population. The Nigerian Yoruba population (Sheiham, 1967) (Fig. 2e) demonstate age-specific PI values similar to those of a British population (Sheiham, 1969) (Fig. 2f) and an Indian population (Ramachandran et al., 1973) (Fig. 2g), but a much poorer oral hygiene standard (Figs. 2e-g). Although inter-examiner differences may account for a large part of the between-population variation of these interrelationship, it is worthy of note that the data in Figs. 2d-f have been collected by the same examiner. Therefore, while the the higher age-specific PI values observed among Nigerian Yorubas as compared to Ibos might be attributed to a lower standard of oral hygiene among the Yorubas this does not explain why a British population has PI scores similar to those of Nigerian Yorubas when the standard of oral hygiene is much better. This observation would seem to indicate that the British population studied may be at higher risk to periodontal diseases than the Nigerian Yoruba population. Similarly, a comparison of the Nigerian Yoruba population (Sheiham, 1967) with the Indian

population (Ramachandran et al., 1973) might indicate that the Indian population constitutes a high-risk population.

Epidemiological studies performed using the Russell PI have indicated that in a number of populations in non-industrialised countries destructive periodontal disease is a major feature from very early age (Sheiham & Jeboda, 1981). These conclusions have been drawn based on the "translation" of PI scores into a periodontal diagnosis proposed by Russell (1956). According to this "translation" a PI score of 0.7-1.9 would indicate "beginning destructive periodontal disease" and PI scores between 1.6 and 5.0 would indicate "established destructive periodontal disease" (Russell 1967b). However, despite PI values that were "the highest recorded for any epidemiological survey using Russell's PI" (Sheiham, 1966) and indicated "established destructive periodontal disease" or worse among Nigerians from the age of 20 years, the number of teeth missing in all age groups surveyed was extremely small (Sheiham, 1967). Apparently therefore, the destructive disease indicated by the high PI values does not result in any major loss of teeth. The low level of tooth mortality was confirmed in a study of the patterns of pocketing among rural Yorubas (MacGregor & Sheiham, 1974) from which it appeared that the average number of teeth present in a random sample of 3717 Western Nigerians aged 10-60+ years was 28 teeth. Yet, the results were summarized as illustrating "the progressive nature of destructive periodontal disease".

Conclusion: Although the Russell Periodontal index system has been perceived as giving rather high weights to signs of

destructive periodontal disease (Russell, 1956) these conflicting results indicate that the Russell PI cannot distinguish between gingival inflammation and periodontal destruction. Therefore, the between population differences in susceptibility to periodontal diseases, as assessed using the PI, cannot be attributed to between population differences in susceptibility to destructive periodontal disease.

Studies using the CPITN

Table 2a,b provides an overview of the results of using the CPITN for assessment of prevalence and severity of periodontal disease in 35-44 year-olds and 15-19 year-olds, respectively, in a number of populations in non-industrialised countries. It appears that major variations between populations seem to exist. Hence, the prevalence of deepened pockets among 35-44 year olds seem to vary from 4% in Zaire and Zimbabwe to 100% in Burkina Faso, 99% in Bangladesh, and 84% in the Central African Republic (Table 2a). While these differences may indicate between population difference in the susceptibility to destructive periodontal disease they may also represent the combined effect of different examiners and different sampling methods used. Although the issue of susceptibility concerns variation in disease experience it is clear that true between population differences cannot be postulated unless the estimates of disease experience derive from a representative, random sample of the populations in question. The CPITN data in

Table 2a,b are derived from an often limited number of subjects and considerable differences in the sampling methods used have been noted (Pilot & Barmes, 1987; Pilot et al., 1986). Some indications of the possible magnitude of such factors may be seen from the data concerning 15-19 year olds in Thailand where the estimates for the prevalence of deepened pockets vary from 4% to 100% and the mean number of sextants with pockets vary between 0.1 and 3.9 (Table 2b). Although the possibility of within population differences of the risk to pocket formation cannot be excluded, these very large differences in a very young population may be accounted for by different interpretation of criteria and selected samples. Great care should therefore be exercised before between population differences of disease experience, as assessed through the CPITN, are attributed to different levels of risk to periodontal disease.

Only a few studies of the prevalence and severity of periodontal destruction, as assessed using the CPITN, have presented data on the distribution of the CPITN scores between individuals in the populations. In the Phillipines (Garcia & Cutress, 1986) less than 30% of the persons examined had 3 or more of their segments affected with 4-5 mm pockets and only very few persons had such pockets in all segments examined (Fig. 3). The number of pockets ≥ 6 mm per person shows a similarly skewed distribution for a Moroccan population (Sardo-Infirri, 1984).

Conclusion: In view of the "extraneous" sources of variation inherent in much of the data collected using the CPITN (Pilot & Barmes, 1987; Pilot et al., 1986) it remains

uncertain whether such data may provide evidence for between population variation of the susceptibility to both pocket formation and, in particular, to destructive periodontal disease. Information on the extent of pocket formation may be usefull for establishing the need for treatment of pockets but the extent of pocket formation is an uncertain estimator of the extent of destructive periodontal disease. The limited data available on the distribution of CPITN codes 3 and 4 within a population do indicate variations in disease experience in different individuals.

Studies using the PDI

Only very few studies have been performed using the Ramfjord PDI (Ramfjord, 1959) in populations in non-industrialised countries, possibly because the index has been regarded as being rather time consuming (Waerhaug, 1966). Among young Indians the PDI scores ranged from 1.53 among 15 year olds to 2.43 among 27-30 year olds (Ramfjord, 1961). Three percent of the 15 year olds, increasing to 92% of the 27-30 year olds, had experienced loss of periodontal support (Ramfjord, 1961). The PDI values reported for persons up to the age of 30 years in China (Wang et al., 1987) are similar to those reported by Ramfjord (1961) but the prevalence of loss of periodontal support seems lower among the Chinese population. The overall prevalence among 18-50 year Chinese was reported to be 44.4% which is much lower than the 92% reported for the Indian population aged 27-30 years (Ramfjord, 1961). However, this difference

probably arises from different criteria for the diagnosis of periodontal destruction. Whereas Ramfjord (1961) used the presence of a true pocket or recession as the diagnostic criterium, the diagnosis "periodontal destruction" in the Chinese study was made only if the PDI score for the individual exeeded 3 (Wang et al., 1987). The latter method is thus likely to underestimate the prevalence of periodontal destruction (Wang et al., 1987).

Conlusion: Because of the limited number of epidemiologic studies available in which the PDI has been used to assess periodontal destruction it is not possible to make inferences about risk differences between populations based on such studies.

Studies using pocket depth measurements.

Table 3 summarizes the results of studies of populations in non-industrialised countries in which destructive periodontal disease has been assessed through measurements of pocket depths. In some of the studies (Reddy et al., 1985; Baelum et al., 1986; Baelum et al., 1988a,b; Tirwomwe et al., 1988; Chen et al., 1989) measurements of pocket depths have been accompanied also by measurements of loss of attachment. However, the results dealing with the loss of attachment findings will be reviewed elsewhere. The majority of the studies listed in Table 3 seem to agree that the prevalence of pockets $\geq$ 4 mm increases with age. While a comparison of the above studies, in particular the studies pertaining to the younger age groups in the Kenyan and the Chinese populations (Baelum et al., 1988a,b; Chen et al.,

1989), might indicate major between population differences in the risk of pocket formation, this is not necessarily the case. Third molars are rarely congenitally absent, impacted or lost among young Kenyans (Manji, Baelum & Fejerskov, 1988) whereas over 60% of the third molars are missing for one of the above reasons in young Chinese (Luan et al., 1989). Since pockets ≥ 4 mm around third molars contribute greatly to the total occurrence of such pockets, in particular among young age groups (Baelum et al., 1988a), the differences of the prevalence of pockets between the Kenyan and the Chinese population may occur as a result of "third molar pocketing" rather than reflecting different levels of risk of pocket formation. The apparently lower prevalence of pocketing among Ugandans (Tirwomwe et al., 1988) is most likely a result of the examination of only selected teeth.

Some of the studies presented in Table 3 have considered the within population distribution of deepened periodontal pockets. In a Kenyan population the number of pockets per person may vary considerably such that 75% of the total number of pockets ≥ 4 mm may be accounted for by only 29% of the persons examined (Baelum et al., 1988a). Considering pockets as deep as 7 mm or more 75% of these are accounted for by 2-15% of the population, depending on age (Baelum et al., 1988a). Similarly skewed distributions of the number of pockets per person are seen among Tanzanians (Lembariti, 1983; Sardo-Infirri, 1984; Baelum et al., 1986), among Chinese (Baelum et al., 1988b; Chen et al., 1989), among

Ugandans (Tirwomwe et al., 1988), and among adult populations in Guinea-Bissau (Matthesen et al., 1989). Conclusion: We have already eluded to the difficulties involved in comparing data from different studies, especially where different examiners have been involved. Nevertheless, it is noteworthy that the prevalence of deepened pockets in these populations is remarkably similar. This would seem to indicate a limited variation between populations of the extent of pocket formation. A number of the studies clearly indicate a large between individual variation of the proclivity to form deep periodontal pockets. These differences may be attributed to variations within populations of the susceptibility to destructive periodontal disease. It remains to be established, however, whether a person with large number of deepened pockets is also a person who presents a large number of sites with extensive loss of periodontal attachment.

Studies using radiographic assessment of periodontal destruction

Due to the logistics involved, radiographic assessment of the degree of periodontal destruction has only rarely been performed in populations in non-industralised countries. Moreover, the different methods used in the assessment and presentation of results render between population comparisons difficult. Budal et al. (1985) assessed the degree of bone resorption in a Cameroon population using a scale from 0 to 10 such that score 1 would indicate less than 10% loss of alveolar bone, score 2 indicate 10-20% loss

etc. The mean bone loss score ranged from 1.3 in 13-19 year olds to 2.6 in 46-73 year olds (Budal et al., 1985). Using a similar methodology Marshall Day & Shourie (1949) in an Indian population reported a 100% prevalence of bone loss from the age of 17 years with an average bone resoption score ranging from .16 in 15-16 year olds to 4.13 in 45-49 years olds. The degree of bone resorption varied between individuals within each age group such that the maximum bone resorption ranged between 1.15 in 15-16 year olds and 7.70 in 41-44 year olds (Marshall Day & Shourie, 1949). In a Brazilian population aged 13-16 years Gjermo et al., (1984) reported a prevalence of bone loss of 27.6% but such that the majority of the affected persons had 2 or less sites demonstrating bone loss.

Conclusion: There seems to be some evidence, based on a few studies employing radiographic methods of assessment, for a skewed distribution of destructive periodontal disease among Brazilian and Indian populations.

Studies using clinical measurements of loss of attachment.

Measurement of loss of attachment probably provides the most meaningfull clinical assessment of the level of periodontal support (Ramfjord, 1974). The information deriving from cross-sectional or longitudinal epidemiological studies in which measurements of loss of attachment have been employed is of undisputable value for attempts to evaluate possible variations of the risk to destructive periodontal disease. However, the relative

scarcity of such epidemiologic studies is a limiting factor when dealing with possible between population differences, whereas within population variations of the risk to destructive periodontal disease are more easily assessed.

The extent and severity of loss of periodontal attachment has been assessed in Sri Lankans (Löe et al., 1978; 1986; Ånerud et al., 1983), in South Pacific island populations (Cutress et al., 1982), in Vietnamese refugees in Malaysia (Scheutz et al., 1983), in South African populations (Reddy et al., 1985), in Tanzanians (Gadegaard & Fejerskov, 1983; Baelum et al., 1986), in Kenyans (Baelum et al., 1988a), in Ugandans (Tirwomwe et al., 1988), and in Chinese (Baelum et al., 1988b; Chen et al., 1989). The methods of recording loss of attachment vary, such that in some studies only selected teeth or surfaces have been examined (Löe et al., 1978; 1986; Ånerud et al., 1983; Tirwomwe et al., 1988; Cutress et al., 1982; Gadegaard & Fejerskov, 1983; Scheutz et al., 1983; Reddy et al., 1985). In some of the studies the age spans covered (Ånerud et al., 1983; Löe et al., 1978; Tirwomwe et al., 1988) or the sample sizes employed (Reddy et al., 1985; Scheutz et al., 1983; Cutress et al., 1982; Baelum et al., 1986) may be relatively small. The most comprehensive studies currently available pertaining to populations in non-industrialised countries are those of Baelum et al. (1988a,b) and Chen et al. (1989) in which measurements of loss of attachment have been performed on four sites of all teeth present (including third molars) in random samples of adult Kenyan and Chinese populations comprising 1131 and 1744 persons, respectively.

The degree of detail with which measurements of loss of attachment has been performed in these studies (Baelum et al., 1988a,b; Chen et al., 1989) has made it possible for us to recalculate these data to conform with some of the methods and modes of data presentation employed in other studies, thus allowing for more accurate comparisons across populations. In these comparisons we have used the Kenyan population (Baelum et al., 1988) as the "standard" against which the destructive periodontal disease experience of other populations are assessed. In so doing we have taken the liberty of including data from a few major studies of loss of attachment in industrialised populations (Miller et al., 1987; Yonemamoto et al., 1988).

Figs. 4a-e show the results of such comparisons. From Fig. 4a it appears that the mean loss of attachment observed among adult Kenyans (Baelum et al., 1988a) is strikingly similar to that observed among adult Chinese (Baelum et al., 1988b, Chen et al., 1989) and among adult Tanzanians (Baelum et al., 1986).

When calculuting the mean loss of attachment in Kenyans including only those teeth and sites examined in Sri Lankan tea-laborers (Löe et al., 1978) and Sri Lankan students (Ånerud et al., 1983) it appears that from the age of 20 years the mean loss of attachment among Sri Lankan tea-laborers is much higher than among Kenyans (Fig. 4b). In particular from the age of about 25-30 years the difference between the two populations increases substantially (Fig. 4b). However, the Sri Lankan student population studied by

Ånerud et al. (1983) demonstrate a mean loss of attachment much more similar to that observed in the Kenyans (Fig. 4b). This is particularly interesting because the Sri Lankan students demonstated mean loss of attachment values similar to those of a young American and a young Norwegian population (Ånerud et al., 1983).

The median loss of attachment observed in buccal surfaces among Kenyans is considerably lower in all age groups than observed among the two South Pacific Island populations studied by Cutress et al. (1982) (Fig. 4c). This finding would indicate that both these two South Pacific island populations experience a loss of attachment which is also considerably higher than among the Sri Lankan population studied by Löe et al. (1978). Thus, the mean value of loss of attachment, in our experience, tends to be higher than the corresponding median value due to pronounced right skewness of the distributions (Baelum et al., 1986). The two South Pacific island populations studied by Cutress et al., (1982) would therefore seem to be the most severely affected populations as yet reported on.

The mean loss of attachment observed among Kenyan adults is quite similar to the levels observed in adult Japanese (Yonemamoto et al., 1988) (Fig. 4d) although young Japanese tend to have more loss of attachment than their Kenyan counterparts. Young U.S. adults (Miller et al., 1987) also have a higher mean loss of attachment than Kenyans of comparable age (Fig. 4e), whereas elderly Americans present less loss of attachment than elderly Kenyans (Fig. 4e) and elderly Japanese (Yonemamoto et al., 1988). It has been

suggested that these differences may be attributed to different methodologies rather than true population differences (Yonemamoto et al., 1988). The relatively high loss of attachment seen in both young Americans and in young Japanese as compared to the Kenyan population may be attributed to tooth brushing habits which have been suggested to influence attachment level among some young industrialised populations (Löe et al., 1978; Ånerud et al., 1983). Nevertheless, it remains a fact that the differences observed between the Kenyan and the Japanese/American populations of the levels of loss of attachment are much less that the differences previously demonstrated between the Kenyan and the Sri Lankan/South Pacific populations.

Rather few studies have examined the distribution of loss of periodontal attachment within the populations (Cutress et al., 1982; Löe et al., 1986; Baelum et al., 1986; Baelum et al., 1988a,b; Tirwomwe et al., 1988; Chen et al., 1989). For the South Pacific populations (Cutress et al., 1982) it was noted that "only a small proportion of individuals are likely to develop alveolar bone loss of sufficient severity to cause major dental breakdown and multiple tooth loss". This appears to contrast the generally high level of loss of attachment in these two populations but might indicate that the distribution of loss of attachment is extremely skewed. The very high levels of loss of attachment reported among the 15-29 year olds (Fig. 4c) seem to be completely accounted for by less than 10-40% of the persons. Thus, less than 10% of the 15-19 year olds and

less 40% of the 20-29 year olds were periodontitis cases, i.e. cases "where the inflammatory condition has caused the gingival attachment to move apically with associated loss of supporting bone" (Cutress et al., 1982).

Among Sri Lankan tea-laborers about 8% of all persons aged 15-46 years have been reported to belong to a "rapid disease progression" group. The basis for this judgement was the presence of at least two sites with loss of attachment ≥ 4 mm before the age of 21 years; or the loss of at least 8 teeth, or attachment loss ≥ 5 mm before the age of 30 years (Löe et al., 1986). About 11% of the individuals were designated "no disease progression" since they at no age presented loss of attachment > 2 mm on any mesial surface. The remaining 81%, falling in between these extremes, were designated a "moderately progressing" group (Löe et al., 1986). These results clearly illustrate a wide within population variation of the severity of loss of attachment.

Both Chinese and East African populations demonstrate markedly skewed distributions of loss of attachment in all age groups, provided that a loss of attachment ≥ 4 mm is used as the cut-off point (Baelum et al., 1986; Baelum et al., 1988a,b; Tirwomwe et al. 1988; Chen et al., 1989) as illustrated in Figs. 5a & b. Taking into account the slightly different age groupings used to characterise the rural Kenyan population (Fig. 5a) and the rural Chinese population (Fig. 5b), respectively, it appears that the features of these distributions are exactly the same. Thus, in all age groups in both populations the cumulative frequency distributions of the proportion of sites per

person that have loss of attachment ≥ 7 mm are pronouncedly skewed, such that less than 15% of the persons have more than 30% of their sites affected with such severe loss of attachment. Exactly the same principles apply to the cumulative frequency distributions of loss of attachment ≥ 4 mm, although the cumulative frequency distributions with increasing age tend to approach more or less a straight line (Figs. 5a,b). However, if loss of attachment of any size (≥ 1 mm) is included the cumulative frequency distribution curves will shift the direction of the skewness, indicating that it is rather unusual to have a large number of sites free of loss of attachment, in particular above the age of 35 years (Baelum et al., 1988a). The effect of these skewed distributions is that most of the marked breakdown observed is accounted for by a relative minority of persons.

The number of persons in the subfractions demonstrated above clearly depend on the threshold value chosen for the breakdown level as well as the age groups concerned. Table 4 shows the proportion of persons who account for 75% of the total number of sites with various levels of breakdown according to age in the Kenyan population (Baelum et al., 1988a). Thus, 75% of the total number of sites with loss of attachment ≥ 7 mm are accounted for by 1-31% of the total population, depending on age. Using loss of attachment ≥ 4 mm as the threshold value 75% of the breakdown is accounted for by 6-53% of the population.

Conclusion: It appears from the between population comparisons performed above that most of the studies in

which destructive periodontal disease has been assessed using measurements of loss of attachment indicate that the age-specific levels of loss of attachment are quite similar across populations. However, a population of Sri Lankan tea-laborers and two South Pacific island populations seem to constitute the exceptions from this rule. These populations may therefore be exposed to factors rendering them more susceptible to destructive periodontal disease than seen in other populations in both industrialised and non-industrialised countries.

All studies in which the within population variation of the severity of loss of attachment has been assessed point to the conclusion that there is a wide variation in the disease experience with only small proportions of the populations accounting for most of the advanced periodontal destruction observed.

Can high-risk groups to periodontal destruction be identified?

As shown above there seems to be good evidence for the proposal that, in any given age group, a small proportion of individuals experience both extensive and severe breakdown. However, the existence of a tail (skewness) in the distribution of destructive periodontal disease experience does not constitute a priori evidence for differences in risk within a population. As previously indicated, even if the intrinsic and extrinsic risk factors were the same for all individuals in the population a variation of the extent and severity of periodontal destruction would nevertheless

be observed. The question which remains to be answered is whether the dispersion of the distribution is greater than that which would be expected due to random effects alone. It seems to us rather important to explain why it is that the distribution of destructive periodontal disease experience in a number of diverse populations should so consistently have a similar and skewed shape. It is thus noteworthy that the observations of skewed distributions are in accordance with the early observations on the epidemiology of periodontal disease made by Russell (1956). Do these observations imply that the risk factors show similarly skewed distributions within a population or do they imply that such skewed distributions are intrinsic features of periodontal breakdown? These questions need to be addressed in epidemiological studies of destructive periodontal disease before definite conclusions as to existence of high-risk groups or individuals may be drawn. One way of approaching these issues might be to contrast these "tails" of the observed distributions of destructive periodontal disease for past exposure to hypothesised risk factors using a case-control design of the epidemiological study (Burt, 1988).

Plaque, calculus and gingival bleeding - risk factors?

Most of the studies presenting skewed distributions of the extent and severity of loss of attachment have implied that these skewed distributions occur in the presence of a pronounced homogeneity of the standards of oral hygiene and

levels of gingival inflammation. Thus, Cutress et al., (1982) noted that these periodontitis-prone subfractions existed ".. even where plaque is massive and gingivitis endemic..", and a similar uniformity of the oral hygiene and gingival inflammatory conditions has been noted also in other studies (Baelum et al., 1986; 1988a; Löe et al., 1986). Observations such as these have led to the proposal that high risk persons or susceptible persons are characterised by an unfavourable host reponse to dental plaque (Ainamo, 1989). Unfortunately, no analysis has so far been performed which may substantiate the proposal of a marked heterogeneity of the extent of periodontal destruction occurring in individuals who are remarkably homogeneous with respect to the oral hygiene standards and the levels of gingival inflammation.

Therefore, using the cross-sectional data obtained in a random sample of 1131 adult rural Kenyans we hypothesised that persons at "high-risk" to periodontal destruction show amounts of visible plaque deposits, subgingival calculus or gingival bleeding similar to those seen in persons of lower risk to periodontal destruction (Baelum et al., 1989). In this analysis risk was defined on the basis of "severe disease for age". Hence, for each of 25 two-year age cohorts 15-16 years, 17-18 years, etc., the loss of attachment observed in a site was considered "severe" or "present" when it exceeded the median value observed for that particular site in the relevant two-year age cohort. A person was considered "severely affected" when the proportion of sites with "severe" loss of attachment belonged to the upper third

of the distribution for the two-year age cohort to which the person belonged. "Moderately" and "mildly" affected persons were defined in a similar way as belonging to the middle or the lower thirds, respectively (Baelum et al., 1989). Table 5 shows that "severely" affected persons had more calculus, and more gingival bleeding than persons in the moderately affected group. Persons of "moderate" severity, in turn, had more plaque, more calculus and more gingival bleeding than the "mild" severity group. These differences were clearly reflected in the bivariate correlation coefficients for the association between the proportion of sites per person with "severe" loss of attachment and the proportion of sites per person with subgingival calculus or gingival bleeding, respectively, ($r=0.70$ and 0.53, respectively). The correlation coefficient for an association between "severe" loss of attachment and visible plaque was 0.20. These findings show that persons who are "severely" affected do generally have more gingival bleeding and more subgingival calculus than do persons who are less affected. This, in turn, questions the proposal that "severely" affected persons exist despite a homogeneity of the levels of calculus and gingival inflammation (Cutress et al., 1982; Löe et al., 1986; Baelum et al., 1986; 1988a). However, the results clearly demonstrated that no "threshold" values could be identified for the levels of plaque, calculus or gingival bleeding which could be of help for clinical identification of those persons defined as being "severely affected".

Some clinical studies have indicated that "high-risk" persons, using the "severe disease for age" definition, may be identified based on clinical criteria such as the bleeding/plaque ratio (Van der Velden, Winkel & Abbas, 1985). If so, these clinical criteria might be of help for identifying "high-risk" persons to destructive periodontal disease as well as for establising criteria that are universally applicable rather than derived from the observed distributions of destructive periodontal disease. In the Kenyan data (Baelum et al., 1988a) we hypothesized that "severe disease for age" persons would present a loss of attachment "response" to the presence of plaque, subgingival calculus or gingival bleeding in a site, which would be different from the "responses" seen in less affected persons. The associations between the presence in a site of visible plaque, or subgingival calculus or gingival bleeding, respectively, and the presence in the same site of "severe" loss of attachment were estimated for each individual in the sample using the Mantel-Haenszel odds ratio estimate for an association between disease and exposure (Breslow & Day, 1980). The distribution of these individual associations were examined for each of the three severity groups previously defined.

Figs. 6a-c show the distributions of the individual odds ratios on a natural logarithmic scale such that positive values indicate a positive association and negative values indicate a negative association. The assoication between "severe" loss of attachment and visible plaque is shown in Fig. 6a, and the association between "severe" loss of

attachment and subgingival calculus or gingival bleeding is shown in Fig. 6b and Fig. 6c, respectively, for the three severity groups defined. Figs. 6a-c demonstrate that the the individual associations were in each case sometimes strongly positive and sometimes strongly negative. The average association with visible plaque was close to 0, indicating no association between a "severe" attachment loss in a site and the presence of visible plaque in that site. However, the association with subgingival calculus and with gingival bleeding, respectively, was on average positive, indicating positive associations in the majority of persons (Figs. 6b&c). The associations between "severe" loss of attachment in a site and the presence of subgingival calculus or gingival bleeding in the same site were significantly higher among "severely" affected persons than among persons with less severe periodontal breakdown (Figs. 6b,c). The best separation between the groups was obtained when considering the association with gingival bleeding (Fig. 6c). It is, however, quite evident from Fig. 6c that the odds ratios observed were rather variable in all three risk-groups such that no threshold values existed for the association which may be used to distinguish persons according to their level of severity. Leaving aside the possible biological rationale for the absence or presence of the associations studied the results clearly demonstrate a considerable heterogeniety of the loss of attachment "responses" to plaque, calculus and gingival bleeding within the groups defined. It is thus apparent that these features

appear of limited value for the purpose of identifying risk-groups, at least based on cross-sectional studies. This conclusion, of course, rely entirely on the assumption that "severe disease for age" is an appropriate measure of risk to periodontal destruction, and as previously indicated this may not be the case.

CONCLUDING REMARKS

Although numerous microbial and host factors have been proposed and investigated (Genco & Slots, 1984; Genco et al., 1986; Slots et al., 1988; Wilton et al., 1988; Curtis et al., 1989) their role in increasing or decreasing the likelyhood of periodontal destruction remains poorly understood. While our understanding of the factors involved in the disease processes may improve through the development and use of more refined techniques for measurement and analysis it is also possible that such refined techniques and analytical methods may not necessarily improve our understanding of the disease process.

There is no doubt today that most forms of periodontal destruction have a multifactorial etiology (Slots et al., 1988). The consequence of this has sometimes led to statements such that while the "universal outcome will be periodontal destruction .. the contributing factors will be those peculiar to an individual" (Wilton et al., 1988). The logical outcome of such an approach would be that destructive periodontal disease will not be understood until every individual is thoroughly understood. The precise purpose of epidemiology is, however, to synthesize the

communalities and to bring order from the wealth of peculiarities.

Some studies have suggested that persons with widespread and severe loss of periodontal attachment may represent "completely different groups of diseases" relative to persons with less severe and widespread disease (Haffajee & Socransky, 1986; Socransky & Haffajee, 1986) based on computer simulations as well as clinical and microbiological findings. However, Cohen & Ralls (1988) have demonstrated that the disease patterns presented by Haffajee & Socransky (1986) may also reflect arbitrary stages in a continuous disease model. Severely affected persons and high risk groups may therefore merely represent a higher degree of "ageing" relative to an underlying process which is essentially the same for all persons (Cohen & Ralls, 1988). If such is the case, it is likely that attempts to identify specific etiological factors accounting for the existence of high-risk groups may be met with only limited success.

Finally, the extent to which random variation and measurement errors contribute to the observed distributions of loss of attachment remains unknown. The errors inherent in measurement of loss of attachment are relatively large causing major problems for attempts to identify attachment level changes in a site (Ralls & Cohen, 1986; Gunsolley & Best, 1988). The observation of a phenomenon of an episodic disease characterised by bursts and remissions (Socransky et al., 1984) has caused considerable interest since an understanding of the determinants of this behaviour could provide important new insights to the nature of destructive

periodontal disease. Thus, much focus has been placed on markers or predictors of these bursts (Haffajee, Socransky & Goodson, 1983; Badersten et al., 1985; Lang et al., 1986). Unfortunately, the interpretation of the data demonstrating bursts and remissions of destructive periodontal disease has been compounded by those who, quite correctly, have pointed out that the measurement error involved in and of itself could mistakenly be interpreted as evidence for the occurrence of bursts or remissions (Imrey, 1986; Ralls & Cohen, 1986; Gunsolley & Best, 1988). Much more importantly, however, it has been shown that bursts and remissions exceeding measurement error may occur as a function of the underlying disease process where the disease process is modelled as a stochastic process (Manji & Nagelkerke, 1989). This model thus lends credence to the claim for bursts and remissions by demonstrating that such burst and remissions can occur exceeding measurement errors. The model also demonstrates, however, that such bursts and remissions are unpredictable and markers or predictors of their occurrence will therefore not be found (Manji & Nagelkerke, 1989).

Whatever the mechanisms involved in destructive periodontal disease it remains at present unclear whether the within population variation in disease experience is a reflection of the "intrinsic" nature of the disease or whether attributable factors can be identified which account for the variations in disease experience.

TABLE 1 Results of epidemiologic studies of periodontal disease, as assessed using the Russell Periodontal index system, in populations in non-industrialised countries.

Age group / Population / Source	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	Comments (age)
Nigeria									
Akpabio 1966	1.1	1.7		1.8		2.0			
Sheiham 1967, Ibo	0.85	1.06	1.12	1.84	2.10	1.78	3.16	2.59*	*(50+)
Yoruba	1.25	1.66	1.86	2.61	3.39	3.22	4.24	4.40*	*(50+)
N H 1967	1.51	2.48		4.56		5.67		6.49*	*(50+)
	1.82	2.96		4.37		5.12		6.51*	*(50+)
	1.43	2.01		3.25		4.49		5.97*	*(50+)
Enwonwu & Edozien 1970	2.63	3.43		4.63		5.80		6.45*	*(50+)
Cameroon									
Bridal et al. 1985	1.2*	2.2		3.0*		4.4*			*(13-19) (30-45) (46-73)
Ethiopia									
Littleton 1983	0.61	0.86		1.25		1.86		2.54*	*(50+)
Olsson 1988	0.45*	0.63*		1.02			2.27*		*(13-14) (19-20) (45-54)
Kenya									
Akabio 1966	1.1	1.5		1.6		1.8			
Sudan									
Emslie 1966	1.1*	1.6*		2.40					*read from graph
Uganda									
Skougaard et al. 1970	1.3	2.1		2.3		2.5*			*(40+)
Kigezi	0.94	1.41							
Toro	1.49	2.19							
Acholi	1.41	2.40							
Mbale	1.89	2.55							
Ethnon us ell 1963b	0.32	0.54		1.75		3.02		3.95*	*(50+)

Table 1 (cont.)

Age group Population/ Source	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	Comments (age)
Iran									
Russell 1967a	1.30		1.94		3.00		3.37		
Thailand									
Russell 1963b	0.41		0.72		1.97		3.06	5.54*	*(50+)
Vietnam									
Russell 1963b	0.53		0.66		1.53		2.62	4.59*	*(50+)
Russell et al.									
1965 (all)	0.67	0.89	1.05	1.79	2.08	2.32	3.13	5.37*	*(60-69)
High	0.59		1.71		3.24		4.69	7.03*	
Interm.	0.51		0.90		2.25		2.73	4.46*	
Low	0.47		0.62		1.20		1.02	3.53*	
Indochina									
DiAngelis & Rojas									
1982	1.15*								*(12-19)
Polynesia									
McKegg									
1981	0.44		0.82		1.75*		3.00*		*(30-44) (45+)
Bolivia									
Palomino									
1978		1.13*			1.73*		2.44*		*(15-30)
		0.67			1.14		3.83		(31-40)
		0.98			1.42		3.12		(41+)
		1.10			1.69		2.70		
Chile									
Russell 1963a,b	0.34		0.67		1.13		2.09	3.51*	*(50+)
Barros & Witkop									
1963a	0.34	0.52	0.84	1.03	1.28	1.93	2.99*		*(45+)
Colombia									
Russell 1963a,b	0.46		0.60		1.58		2.28	3.45*	*(50+)
Equador									
Russell 1963a,b	0.38		0.41		0.89		1.43	2.34*	*(50+)

Table 1 (cont.)

Age group Population/ Source	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	Comments (age)
Venezuela									
Donnelly et al. 1977	1.32*		2.00*				2.64*		*(12-19) (20-39) (40+)
Ceylon (Sri Lanka)									
Maerhaug 1967	0.86*	1.54		3.26		4.40		5.38*	*(13-19) (50+)
India									
Gupta 1962	0.63*	1.27		3.00		4.11	4.55-4.75*		*(11-20) (51-70)
Basu & Dutta 1965	0.98*	1.29*	1.65*						*(12-17) (18-23) (24-30)
Ramachandran et al. 1973 urb.	1.0*	1.9		3.6		4.5		5.5*	*(11-20) (51+)
rur.	1.2*	2.2		3.6		4.9		4.1*	
Tewari et al. 1979	1.45*								*(16)
Johansen 1970		1.18*							*(18-32)
Greene 1960	1.00*	1.36*	1.45*	2.00*					*(18) (19-22) (23-26) (27-30)
Alaskan eskimos									
Russell et al. 1961	0.40	0.53	0.88	1.23	1.64	1.36	1.47*		*(45+)
Kristoffersen and Bang 1973	0.99	1.20		1.75		2.41*			*(40+)

TABLE 2a. Results of studies on the epidemiology of periodontal disease among populations in non-industrialised countries, as assessed using the CPITN. BLEEDING = gingival bleeding but absence of pockets ≥ 4 mm. POCKET1 = pocket depths of 4-5 mm; POCKET2 = pocket depths ≥ 6 mm. PREV = prevalence of code; MEAN = mean number of sextants with code.
If age is not stated the data concern 35-44 year olds. Unless otherwise is stated the data sources are Pilot et al. (1986), and Pilot & Barmes (1987).

Country/ Source	Age	BLEEDING PREV	BLEEDING MEAN	CALCULUS PREV	CALCULUS MEAN	POCKET1 PREV	POCKET1 MEAN	POCKET2 PREV	POCKET2 MEAN
Phillipines									
Garcia &	20-29	5	0.4	78	3.4	11	0.2	0	0
Cutress		2	0.4	66	3.9	29	0.5	1	0.0
1986	45+	0	0.2	39	3.4	56	1.2	4	0.1
Sardo-Infirri									
1984			1.2		4.0		0.4		0.0
Indonesia		3	0.2	65	3.3	16	0.3	4	0.1
		8	0.9	63	3.1	14	0.2	2	0.1
		0	1.0	36	3.5	53	1.3	10	0.2
Cook Islands		0	0.2	34	4.2	57	1.4	9	0.1
onga									
Sardo-Infirri			0.2		1.1		3.0		1.4
984									
western Samoa									
Sardo-Infirri			0.2		3.0		2.0		0.7
984									
Thailand		0	0.4	50	3.7	34	1.1	16	0.3
		0	0.0	47	3.8	47	1.6	11	0.2
rdo-Infirri									
1984			0.1		0.3		2.3		2.9
ina		2	0.1	51	4.6	36	0.7	9	0.2
rdo-Infirri									
1984			0.1		2.9		0.3		0.1
N pal		2	1.1	45	3.1	25	1.1	28	0.7
		1	0.0	43	3.6	25	1.6	30	0.7
Sardo-Infirri									
1 84			1.2		3.1		0.5		0.0
Bangladesh		0	0.2	0	0.4	34	3.1	65	2.1
S. rdo-Infirri									
1 34			0.1		0.3		3.2		2.0
Sri Lanka		1	0.2	55	3.3	27	0.7	10	0.2
Libya		0	0.1	13	3.1	53	1.9	34	0.6
Mo occo		4	1.5	46	1.1	28	0.9	14	0.3

		5	1.3	31	1.7	40	2.0	16	0.2
Burkina Faso		0	0.0	1	0.5	25	3.3	75	2.1
entral Afri-an Republic		1	0.4	14	2.0	52	2.3	32	0.8
ɔgo		3	0.3	35	0.9	21	0.6	6	0.1
Niger		20	1.9	53	2.4	26	0.8	0	0.0
ı ıgeria		5	0.8	52	3.5	35	1.1	3	0.1
Sardo-Infirri :)84			0.0		1.2		0.8		3.9
Kenya		4	0.3	31	2.6	49	2.3	14	0.2
T nzania		0	0.5	28	2.3	63	2.1	7	0.1
Lembariti	20-24		0.7		2.4		1.6		0.1
(983)	25-29		0.5		2.2		1.9		0.1
	30-44		0.5		2.5		2.0		0.1
	45+		0.2		1.8		2.7		0.2
Z-ire		1	0.3	93	4.9	4	0.1	0	0.0
Zımbabwe		0	0.0	87	1.4	3	0.1	1	0.0

TABLE 2b. Results of studies on the epidemiology of periodontal disease among populations in non-industrialised countries, as assessed using the CPITN. BLEEDING = gingival bleeding but absence of pockets ≥ 4 mm. POCKET1 = pocket depths of 4-5 mm; POCKET2 = pocket depths ≥ 6 mm. PREV = prevalence of code; MEAN = mean number of sextants with code.
Data concern 15-19 year olds. Unless otherwise is stated the data source is Pilot et al. (1987).

Code	BLEEDING		CALCULUS		POCKET1		POCKET2	
Country/ Source	PREV	MEAN	PREV	MEAN	PREV	MEAN	PREV	MEAN
Bhutan	14	1.1	75	1.8	6	0.1	0	0
Nepal	9	1.0	75	3.0	2	0.0	0	0
	4	2.3	88	3.2	6	0.1	1	0
China	4	0.4	74	3.2	17	0.3	0	0
Korea	16	0.4	33	0.5	2	0.0	0	0
	17	0.7	36	0.5	7	0.1	0	0
	13	0.8	41	0.8	12	0.2	1	0
	19	0.8	39	0.8	9	0.1	1	0
	19	0.7	46	1.3	11	0.2	1	0
Thailand	0	0.5	0	1.7	90	3.7	10	0.1
	2	1.5	94	4.1	4	0.1	0	0
	0	0.6	55	3.8	44	0.7	1	0
	1	0.1	83	4.6	15	0.3	1	0
Indonesia	1	0.2	76	3.6	19	0.4	0	0
Cook Islands	4	1.0	80	3.8	15	0.2	0	0
Phillipines Garcia & Cuenca 1986	7	0.7	76	2.5	4	0.1	0	0
Maldives	20	1.1	42	0.9	0	0.0	0	0
Seychelles	4	0.3	93	3.3	0	0.0	0	0
Bangladesh	14	1.5	27	1.9	55	1.5	0	0
	5	0.5	71	3.4	15	0.4	0	0
Libya	5	0.9	80	3.6	15	0.2	0	0
Niger	34	2.5	59	1.7	0	0.0	0	0
Nigeria	11	0.9	85	4.0	1	0.0	0	0
	3	0.9	31	3.4	56	1.3	10	0.1
Ethiopia	36	3.1	54	1.0	9	0.2	0	0
Somalia	43	1.8	14	0.8	0	0.0	0	0
Kenya	52	3.7	40	1.0	6	0.2	2	0

Tanzania	5	0.7	30	2.2	62	1.6	1	0
Malawi	3	0.1	36	1.4	0	0.0	0	0
Zaire	1	0.5	96	4.6	2	0.0	0	0
Zimbabwe	0	0.0	68	0.9	0	0.0	0	0
	0	0.1	80	1.3	0	0.0	0	0
Jamaica	9	1.6	20	1.7	34	0.9	37	0.6
Brazil Gjermo et a. (1983)	14*		23*		62*		1*	

*% segments

Table 3. The results of epidemiological studies in which destructive periodontal disease has been assessed using measurements of pocket depths.

Country/ Source	Age	Prevalence %	Severity	Remarks
Swaziland				
Klausen &	20-24	47.5%	1.0	WHO Basic Methods
Fanöe 1983	35-44	66.8%	2.1	Mean no. of segments affected
South Africa				
Reddy et al.	15-19	-	2.40	Mean pocket depth.
1985	20-39	-	2.38	Less than 9% have
	40+	-	2.73	pockets ≥ 4 mm
Tanzania				
Sardo-Infirri	35-44	5.2%		Pockets ≥ 6 mm
1984				3% have 1 pockets
				2% have 2-6 pockets
Lembariti	15-19	-	2.0-2.4	Mean no. of teeth with
et al. 1988	20-24	-	3.2-3.4	pockets > 3.5 mm. Rural
	25-29	-	3.5-4.7	more than urban. Overall
	30-44	-	4.3-4.5	% with pockets 3.5-5.5
	45+	-	4.0-6.7	mm=70%; 5.2% have ≥ 6 mm
Baelum et al.				
1986	30-34	45%	5%	Pockets ≥ 4 mm. Fraction
	35-39	79%	24%	with > 10 pockets ≥ 4 mm
	40-49	89%	36%	
	50+	84%	29%	
Nigeria				
MacGregor &	10-19	33.4%	4.8	Pockets ≥ 3 mm associa-
Sheiham 1974	20-29	58.0%	16.4	ted with apical migra-
	30-39	79.6%	34.3	tion of epithelium.
	40-49	85.8%	52.3	% teeth affected.
	50-59	100.0%	56.9	
	60+	75.0%	48.4	
Guinea-Bissau				
Matthesen et	30-39	93%	24%	Pockets ≥ 4 mm. Fraction
al. 1989	50-59	98%	49%	with > 20% sites with
				pockets ≥ 4 mm.
Kenya				
Baelum et al.	15-24	75%	3%	Pockets ≥ 4 mm. Fraction
1988a	25-34	87%	13%	with > 20% of their
	35-44	83%	20%	surfaces with pockets
	45-54	92%	34%	≥ 4 mm
	55-65	95%	40%	
Uganda				
				Pockets ≥ 4 mm.
Tirwomwe et	35-44	36.4%	3.6%	Fraction with ≥ 25% of
al. 1988	55+	57.9%	6.1%	sites with pockets ≥ 4mm
China				
				Pockets ≥ 4 mm.
Chen et al.	20-29	25-32%	1-8%	Urban < Rural. Fraction
1989	30-39	30-51%	3-11%	with ≥ 10% of their
	40-49	51-62%	15-20%	surfaces with pockets
	50-59	58-74%	13-25%	≥ 4 mm

Baelum et al. 1988b	60-64	74-84%	21-35%	Urban - Rural. Fraction with ≥ 10% of their surfaces with pockets ≥ 4 mm
	65-69	80-87%	27-43%	
	70+	70-82%	30-43%	
India				
Ainamo & Ainamo 1978		31%	1.5-2%	Mean age 21-23 years. % surfaces with pockets > 3 mm
Tuvalu South Pacific				
Speake & Malaki 1982	19	1.1%	0	WHO Basic Methods. Mean number of segments affected
	25-29	6.0%	0.2	
	35-44	29.8%	0.9	
	45-54	45.8%	1.5	
	55-64	33.7%	1.1	

TABLE 4. The proportion of persons in each age group accounting for 75% of the total number of sites with loss of attachment ≥ 1 mm, ≥ 4 mm, and ≥ 7 mm, respectively. Data from Baelum *et al.*, (1988a).

	Proportion of persons accounting for 75% of the total number of sites with loss of attachment		
Age (years)	≥ 1 mm	≥ 4 mm	≥ 7 mm
15-24	23%	6%	1%
25-34	38%	22%	4%
35-44	52%	32%	11%
45-54	60%	43%	21%
55-65	62%	53%	31%

TABLE 5. The average proportion of sites per person with "severe" loss of attachment (see text for definition), with visible plaque, with subgingival calculus, and with gingival bleeding. Given according to level of severity (see text for definition). Numbers in parenthesis denote the standard deviations.

Proportion of	Degree of severity of the individuals		
sites with	Low	Moderate	High
"Severe" loss of attachment	6.1 (6.6)	30.9 (17.9)	65.2 (31.0)
Visible plaque	44.8 (33.8)	52.7 (36.3)	53.2 (36.0)
Subgingival calculus	31.5 (30.4)	54.0 (32.4)	68.4 (34.6)
Gingival bleeding	47.8 (27.0)	61.2 (26.3)	75.0 (24.5)

LEGENDS

Fig. 1a. Two persons, A and B, of the same age are at the time of the cross-sectional study observed to have experienced the same amount of periodontal breakdown. However, the pathway followed by person B (hatched line) may have been different from that followed by person A.

Fig. 1b. Two persons, A and B, are at the time of the cross-sectional study observed to have experienced the same amount of periodontal breakdown, but person B is considerably older than person A. However, the disease may have started at a later time in person B (hatched line) and progressed at the same rate as in person A.

Fig. 1c. Two persons, A and B, of the same age are at the time of the cross-sectional study observed to have experienced different amount of periodontal destruction, such that B is much less affected than A. However, as indicated by the hatched line, the disease may have started in B at a later age, but progressed at the same rate as in A.

Figs. 2a-g. The age-specific relationships between Russell PI values and oral hygiene index values as observed in a number of populations. Note that the oral hygiene index used in Figs. 2a-c is the OHI-S, whereas the OHI is used in Figs. 2d-g.

Fig. 2a: A United States population (Johnson et al., 1965);

Fig. 2b: Equador and Montana populations (Greene 1963);

Fig. 2c: A Vietnamese population (Russell, 1965);

Fig. 2d: A Nigerian Ibo population (Sheiham, 1967);

Fig. 2e: A Nigerian Yoruba population (Sheiham, 1967);

Fig. 2f: A British population (Sheiham, 1969);

Fig. 2g: An Indian population (Ramachandran et al., 1973)

Fig. 3. The cumulative frequency distribution of persons according to the number of sextants per person with 4-5 mm pockets as observed in a Phillipine population. Data from Garcia & Cutress, (1986).

Figs. 4a-e. A comparison of the age-specific severity of loss of attachment as observed in various populations. Data originating in an adult rural Kenyan population (Baelum et al., 1988) have been used as the "standard" such that the calculations for the Kenyan population have been performed in the same way as in the populations with which comparisons are made.

Fig. 4a: The mean loss of attachment observed in adult Kenyans (Baelum et al., 1988a) compared with the mean loss of attachment observed in adult Chinese (Chen et al., 1989), and in adult Tanzanians (Baelum et al., 1986). Calculations based on the mesial, buccal, distal and lingual recordings of loss of attachment in all teeth present.

Fig. 4b: The mean loss of attachment observed among Sri Lankan tea-laborers (Löe et al., 1978), Sri Lankan students (Ånerud et al., 1983) and adult Kenyans. Calculations based

on the mesial and buccal recordings for all teeth present except third molars.

Fig. 4c: The median loss of attachment observed in buccal surfaces among two South Pacific Island populations (Cutress et al., 1982) and in adult Kenyans. Calculations based on buccal surfaces in all teeth present.

Fig. 4d: The mean loss of attachment observed among adult Japanese (Yonemamoto et al., 1988) and adult Kenyans. Japanese data are based on 6 sites in all teeth present whereas only 4 sites were available for the Kenyan population.

Fig. 4e: The mean loss of attachment observed among adult Americans (Miller et al., 1987) and adult Kenyans. The USA data are based on recordings of the mesial and buccal site in all teeth present, excluding third molars, in two randomly selected quadrants whereas the Kenyan data are based on all 4 quadrants.

Fig. 5a,b: The cumulative frequency distribution of individuals according to the proportion of sites per person with loss of attachment $\geq$ 4 mm, and $\geq$ 7 mm, respectively. Given for rural Kenyans (Fig. 5a), and for rural Chinese (Fig. 5b).

Figs. 6a-c: The cumulative frequency distributions of persons according to their odds ratio for an association between "severe" loss of attachment in a site (see text for definition) and the presence of visible plaque in the same

site (Fig. 6a); the presence of sub-gingival calculus in the same site (Fig. 6b); or the presence of gingival bleeding in the site (Fig. 6c).

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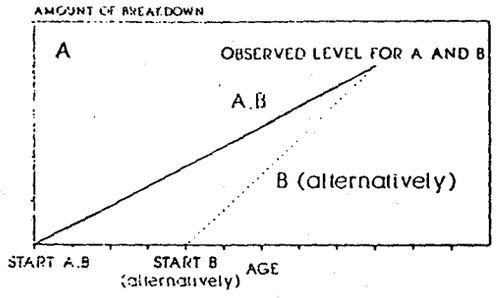
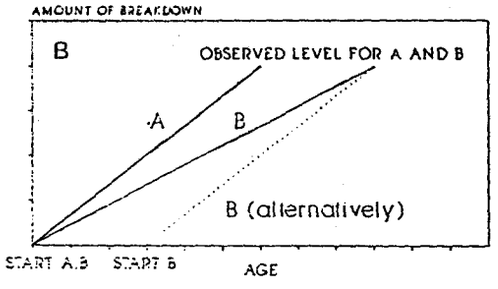

AMOUNT OF BREAKDOWN
A
OBSERVED LEVEL FOR A AND B
A.B
B (alternatively)
START A.B
START B (alternatively)
AGE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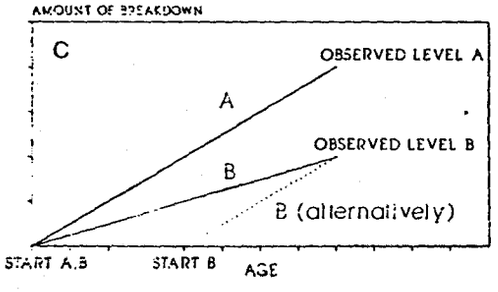
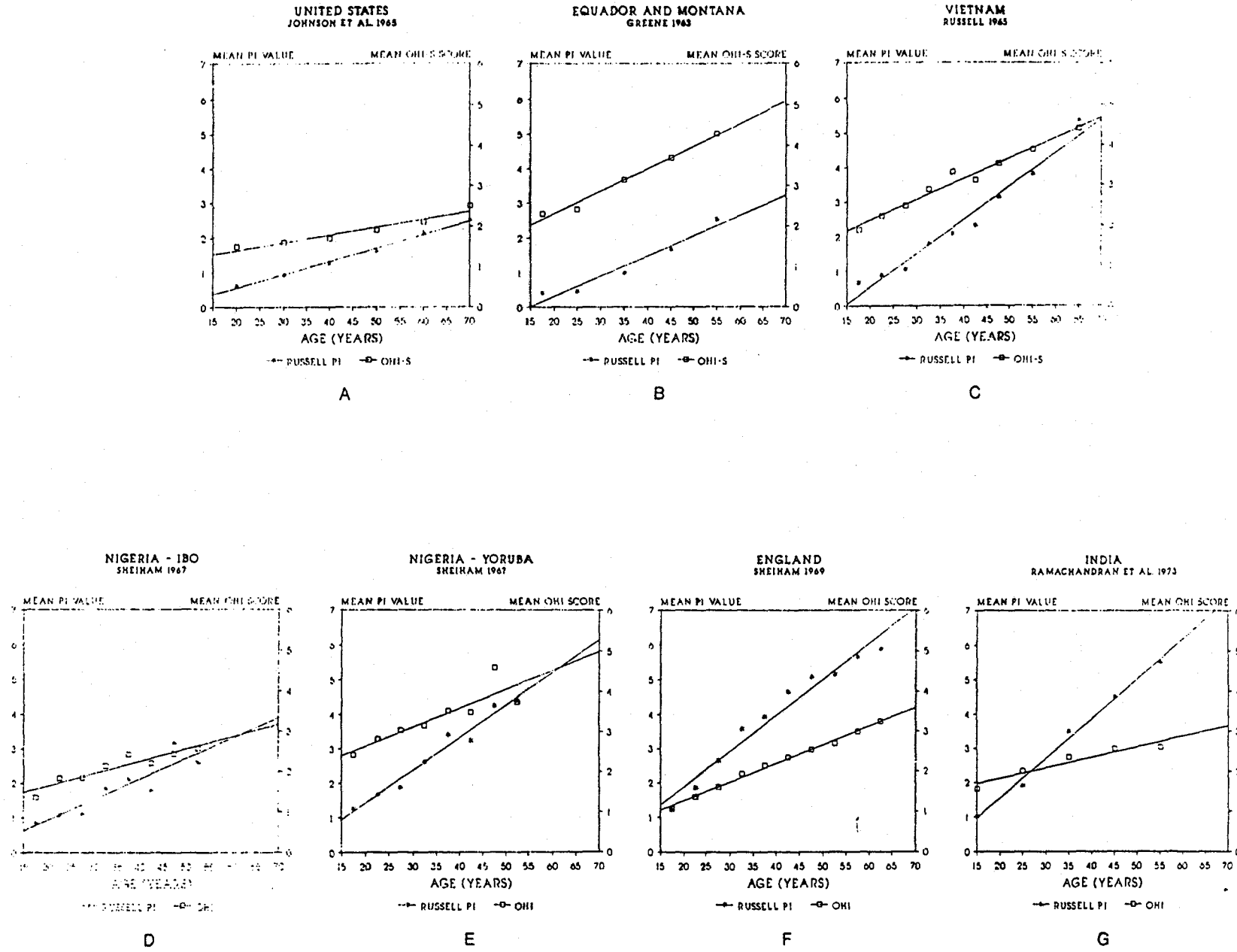
B
OBSERVED LEVEL FOR A AND B
A
B
B (alternatively)
START A.B
START B
AGE


AMOUNT OF BREAKDOWN
C
OBSERVED LEVEL A
A
OBSERVED LEVEL B
B
B (alternatively)
START A.B
START B
AGE

Figs. 1a-c


UNITED STATES
JOHNSON ET AL. 1965
EQUADOR AND MONTANA
GREENE 1963
VIETNAM
RUSSELL 1965
MEAN PI VALUE
MEAN OHI-S SCORE
AGE (YEARS)
RUSSELL PI
OHI-S
A
B
C
NIGERIA - IBO
SHEIHAM 1967
NIGERIA - YORUBA
SHEIHAM 1967
ENGLAND
SHEIHAM 1969
INDIA
RAMACHANDRAN ET AL. 1973
MEAN OHI SCORE
OHI
D
E
F
G

Figs. 2a-g.

1

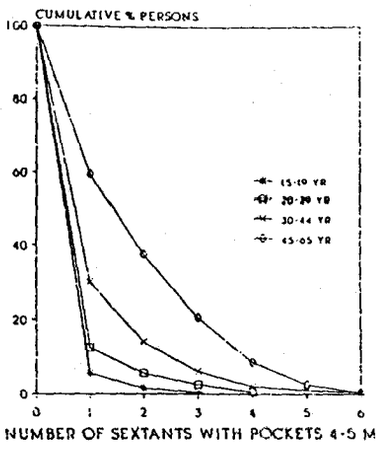
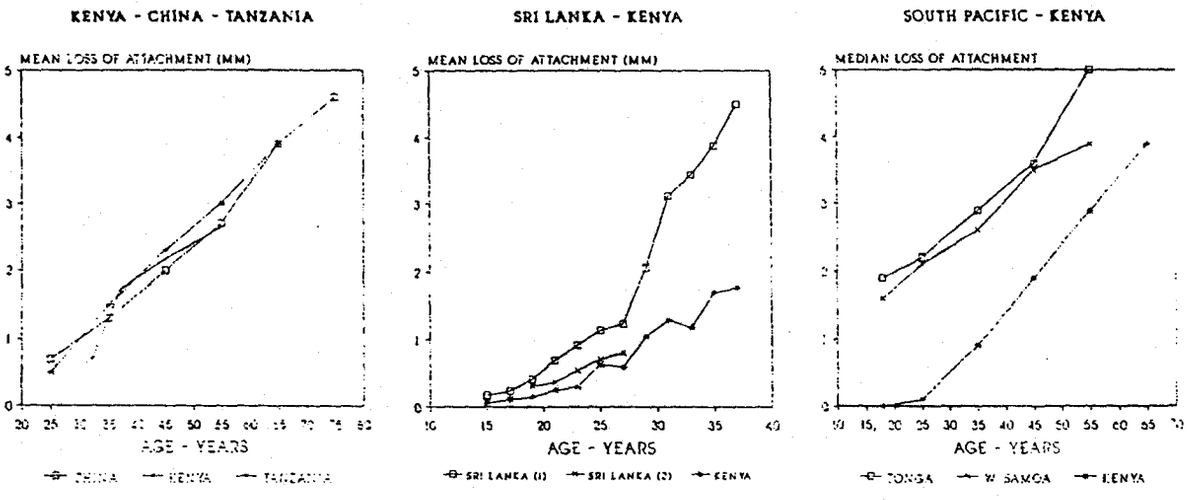
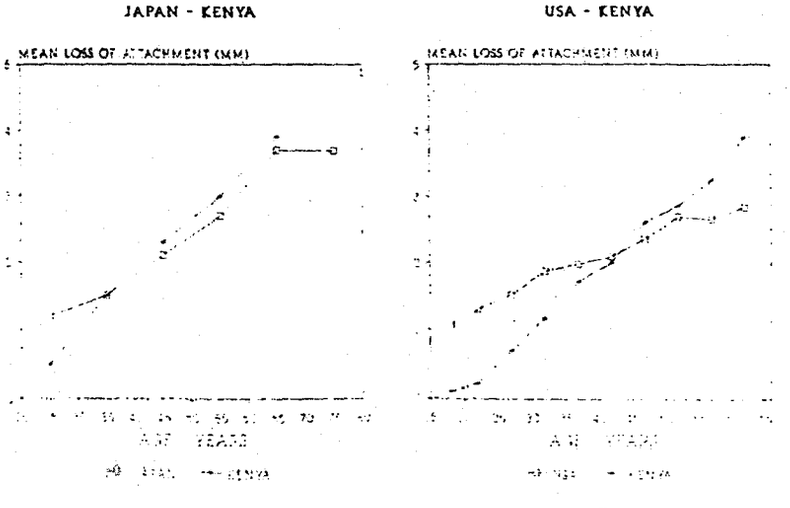


Fig 3.



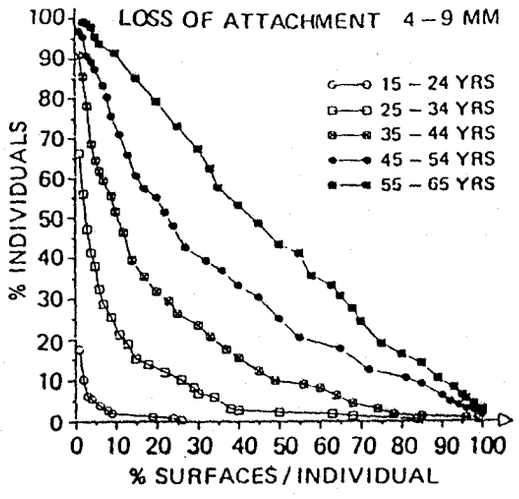
A B C

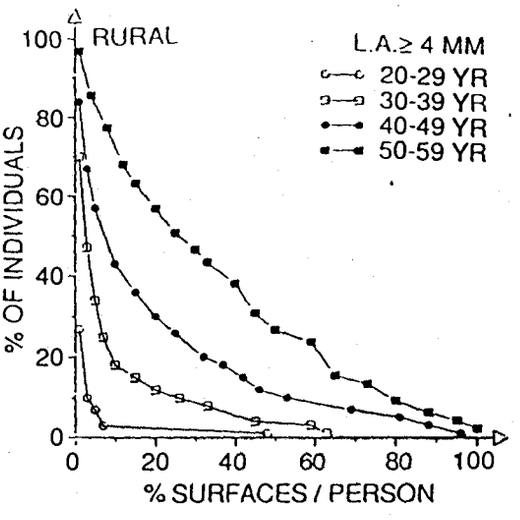
Fig 4a-c

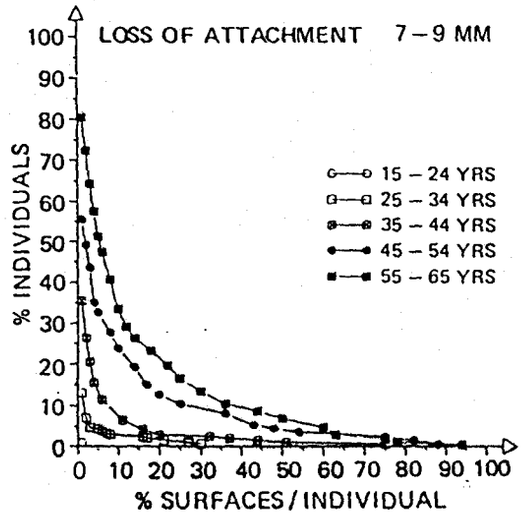


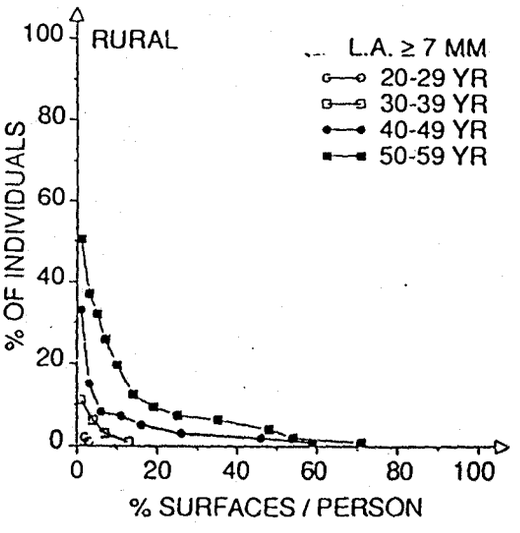
D E

Fig 4 d-e



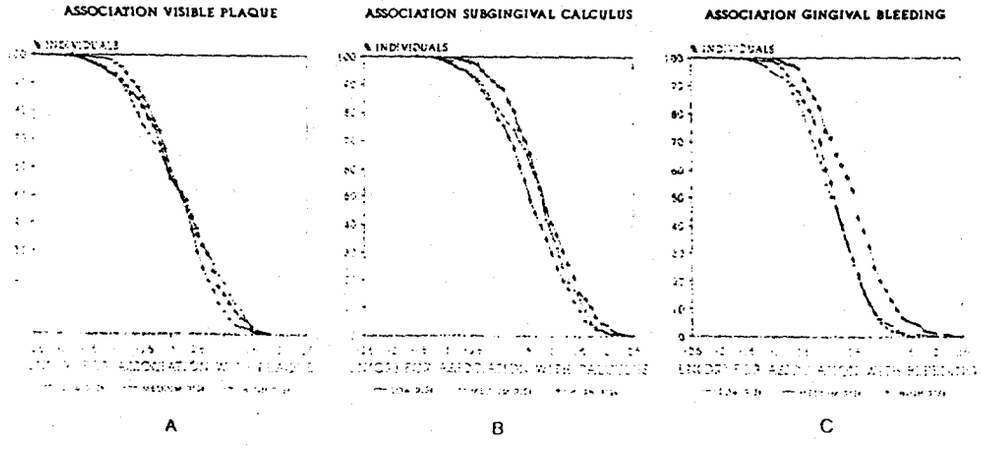






A B

Figs 5a & B



A B C

The Nature and Mechanisms of Dental Fluorosis in Man

O. FEJERSKOV, F. MANJI[1] and V. BAELUM[2]

Dept. of Oral Anatomy, Dental Pathology and Operative Dentistry, [2]Dept. of Periodontology and Oral Gerontology, and the WHO Collaborating Centre for Oral Health Planning and Research in Third World Countries, The Royal Dental College, Vennelyst Boulevard, DK-8000 Aarhus C, Denmark, and [1]Kenya Medical Research Institute, Medical Research Centre, PO Box 20752, Nairobi, Kenya.

Short title: DENTAL FLUOROSIS IN MAN

Any use of fluorides, whether systemic or topical, in caries prevention and treatment in children results in ingestion and absorption of fluoride into the blood circulation. The mineralization of teeth under formation may be affected so that dental fluorosis may occur. Dental fluorosis reflects an increasing porosity of the surface and subsurface enamel, causing the enamel to appear opaque. The clinical features represent a continuum of changes ranging from fine white opaque lines running across the tooth on all parts of the enamel to entirely chalky white teeth. In the latter cases the enamel may be so porous (or hypomineralized) that the outer enamel breaks apart posteruptively and the exposed porous subsurface enamel becomes discoloured. These changes can be classified clinically using the TF index to reflect, in an ordinal scale, the histopathological changes associated with dental fluorosis. Compared with Dean's and the TSIF index, we consider the TF index to be more precise. Recent studies on human enamel representing the entire spectrum of dental fluorosis have demonstrated a clear association between increasing TF-score and increasing fluoride content of the enamel. So far no useful data on dose (expressed in mg fluoride/kg b.w.) -response (dental fluorosis) relationships are available. In this paper we have therefore, reevaluated the original data by Dean et al. (1941; 1942), Richards et al. (1967) and Butler et al. (1985) from the USA applying the equation of Galaghan and Vermillion (1957) which permits the calculation of water intake as a function of temperature. By so doing, it can be demonstrated that there is a linear association between fluoride dose and dental fluorosis ($r^2 = 0.87$). Even with very low fluoride intake from water a certain level of dental fluorosis will be found in a population. When the linear dose-response curve is applied to previous data from the use of fluoride supplements, these data are in full accordance. This indicates that we have already useful data available which to some extent allows us to predict prevalence and severity of fluorosis in a child population

which is exposed to a known amount of fluoride. Because dental fluorosis may occur in some individuals and populations to a higher prevalence and degree than expected, and rare cases do occur who exhibit clinical changes similar to those of fluorosis - but with no known excessive fluoride background - it is concluded that it is important to intensify studies on factors which alone or in combination can make individuals more or less susceptible to the effect of fluoride.

Introduction.

Up until the last major international meeting on cariostatic mechanisms of fluoride held in Naples 1976 (Brown and König, 1977), there was almost consensus about the nature and mechanisms of dental fluorosis. The prevailing concept was that maximum caries reduction would only be obtained through systemic use of fluorides. Dental fluorosis was therefore considered an unavoidable "side effect" which was commonly thought to result from the toxic effect of fluoride on the secretory phase of enamel formation.

Studies on human dental fluorosis during the early 1970s (Fejerskov et al., 1974; Fejerskov et al., 1975) led us to conclude that a reevaluation of human dental fluorosis was necessary and that little was actually known about pathogenic mechanisms in dental fluorosis (Fejerskov et al., 1977). At the same time Myers (1978) in a review on dose-response relationships in dental fluorosis stated "while levels of 2 ppm of fluoride (in water) are associated only with the mildest form of dental fluorosis, a margin of safety of 2 can be regarded as impressively small". The statement reveals that the underlying assumptions about dose-response relationship was strongly related to the concept that an optimal dose of fluoride was well-defined. A proper understanding of the nature and mechanisms of dental fluorosis necessitates a thorough understanding of how fluoride affects amelogenesis.

The aim of this paper is therefore

- To present what is known about the clinical features of dental fluorosis in humans and to discuss appropriate classification systems.
- To discuss the histopathology, biochemistry and chemistry of human fluorotic enamel from the point of view of identifying possible pathogenic mechanisms.
- To reconsider established epidemiological data in order to elucidate the nature of the dose-response relationship, and thereby derive the means for predicting the toxicological effects to be expected when additional fluoride is

provided to a population.

Clinical features of dental fluorosis

Much of the apparent confusion about the clinical appearance of fluoride-induced enamel defects stems from the use of the term "mottled" enamel. Beyond doubt, fluoride is the one single factor most commonly responsible for causing enamel "mottling". However, other factors, although relatively rare, may cause opacities in the enamel (Small and Murray, 1978). The term "mottled" enamel covers, therefore, a broader clinical spectrum of lesions than does "dental fluorosis".

Dental fluorosis in humans has a very characteristic appearance both in terms of the single tooth surface and the distribution within the mouth of an individual (Dean, 1936; 1942; Thylstrup and Fejerskov, 1978; Fejerskov et al., 1988a; b). Therefore, dental fluorosis may usually be distinguished from any other enamel disturbance. Apart from one study on strontium in water supplies (Curzon and Spector, 1977) no other causative factor has so far been identified in man capable of inducing the spectrum of enamel changes which is characteristic of the changes induced by fluoride. Furthermore, there exists today an extremely large amount of epidemiological data demonstrating that the occurrence of such lesions is associated with excessive fluoride intake (Dean, 1934; 1942; Møller, 1965; Richards et al., 1967; Myers, 1978; Thylstrup and Fejerskov, 1978; Manji et al., 1986a;b; Larsen et al., 1986; Szpunar and Burt, 1988).

Fluorosis at the time of eruption. - Fluoride-induced enamel changes as they appear at the time of eruption range from thin white opaque lines corresponding to the perikymata running across the tooth surface, to an entirely chalky white enamel (Thylstrup and Fejerskov, 1978; Fejerskov et al., 1988b). Such clinical features reflect that fluoride given in low concentrations over the long period of tooth development results in various degrees of enamel porosity

(or hypomineralization). In its mildest forms, the porosity is to be found in the outermost enamel, only, but the entire tooth surface is involved. With increasing severity both the depth of enamel involvment and degree of porosity of the enamel increases. Assuming a relatively constant exposure level (most commonly water-borne fluoride) all surfaces of a given tooth will be equally affected (Thylstrup and Fejerskov, 1978).

Within mouth distribution of fluorosis. - The changes are symmetrically distributed within the oral cavity, but the severity varies among the different types of teeth (Dean, 1934; 1942; Møller, 1965; Thylstrup and Fejerskov, 1978; Larsen et al., 1985; 1986; Manji et al., 1986a). The degree of severity appears in principle to reflect the stage in life at which the various tooth types are formed and mineralized (Larsen et al., 1987) irrespective of whether the child is born and reared in a low (< 0.2) or a higher (1-2 ppm) water fluoride area (Fig. 1). The teeth which form and mineralize early in life are those that are least affected, whereas the later in life the teeth mineralize the more severely will they be affected. In accordance with this concept, exposure to very low fluoride levels will result in only a few teeth exhibiting subsurface porosities to a degree that will be clinically manifest, although the underlying pattern will nevertheless remain the same. In areas of high fluoride exposure the primary dentition can also be involved, and in principle the same within-mouth distribution is found (Thylstrup, 1978; Larsen et al., 1988).

Given that dental fluorosis is characterized by increased-porosity of the enamel it will be appreciated that the oral environment may influence the clinical appearance of the enamel once the tooth has erupted into the mouth. However, the degree of posteruptive modification of the enamel will to some extent also be determined by the degree of hypomineralization at the time of eruption.

Posteruptive changes in fluorosis. - In the milder forms

of dental fluorosis, mechanical attrition, e.g. resulting from the use of abrasive tooth paste, will over time cause an apparent "remission" of the fluorotic lesions most likely due to surface enamel removal. The upper incisors are particularly susceptible to such environmental modifications. Often the incisal part of the central incisors may be exposed to air when there is insufficient lip closure. Consequently, the incisal part will become dried out for long periods and any porosities will therefore be discerned. In addition, the incisal edges/cuspal tips are not overlying dentin, so any change in pore volume in these areas will reflect itself as clinically different from that of the remaining parts of the teeth. This may give the impression that the incisal area is more affected than the remainder of the surface, which in reality is equally porous. This will not be apparent clinically unless the surface has been dried properly. The incisal edges may also be subjected to attrition and, depending on the degree of enamel porosity, this leads to exposure of the underlying more porous enamel. There will then be a strong tendency for stains to be taken up by the thus exposed enamel corresponding to the position of the upper lip across the surface (Fejerskov et al., 1988b). Generally, the uptake of stain will depend on the degree of porosity, but will also be influenced by the nature of the individuals' dietary habits. Thus, discoloration in itself is not an appropriate measure of severity. However, as discoloration occurs most often in the upper incisors, and as these are the teeth most easily seen by lay persons, such features have been mistakenly considered as constituting intrinsic and principle characteristics of dental fluorosis.

In more severe forms of dental fluorosis, the tooth erupts into the oral cavity entirely chalky white. The degree of porosity (hypomineralization) of such teeth results in a diminished physical strength of the enamel, and parts of the superficial enamel may break away. This type of damage ranges from small scattered round defects (pits) to

and Yasaki (1976). The latter set up differential diagnostic criteria between fluoride and non-fluoride-induced enamel opacities and when these differential diagnostic characteristics are taken into account, much less confusion about early diagnosis of dental fluorosis will prevail (Fejerskov et al., 1988b).

Classification systems of dental fluorosis

In order to describe the prevalence and severity of dental fluorosis within a population or within an individual, it is necessary to have a classification system which is sensitive, precise and valid (in the sense that it measures what it is supposed to measure). However, there are diverging opinions in this context. One line of thinking emphasizes the need for a classification system which specifically measures fluoride-induced enamel changes in order to reflect increasing severity of the lesions. Typical examples are those of Dean (1934), and Thylstrup and Fejerskov (1978). Opposing this concept, is the view that no diagnosis of enamel defects should be made on the basis of a presumed etiology (Jackson, 1961; Al-Alousi et al., 1975; FDI, 1982). According to this concept all defects in enamel are recorded based solely on descriptive criteria. If it is considered important to record the entire spectrum of enamel defects irrespective of causative factors, such an approach may be useful. However, when such systems are used, the authors usually spend most of their discussions trying to decide retrospectively which types of lesions might have been induced by fluoride. Of importance here is the fact that in such studies the very lesion types ("diffuse white opacities") which would be expected to be associated with fluoride intake are indeed so! (Cutress et al., 1985). Therefore, in contrast to others (eg. Clarkson and O'Mullane, 1989), we consider such classifications to be of no value, as was also recently stressed by Horowitz (1986).

The continuum of clinical changes which characterize dental fluorosis can best be classified using the TF-index to

reflect, in an ordinal scale, the histopathological changes associated with fluoride-induced enamel changes. In a recent review (Fejerskov et al., 1988b), it has been emphasized that the TF-index represents logical improvements and extentions of Deans's original approach. Dean's classification has certain limitations when dealing with both the early signs as well as the severe forms of dental fluorosis (Thylstrup and Fejerskov, 1978). The first difficulty lies in its weak definitions of the various categories in the milder forms. This is perhaps best illustrated by comparing the photographic illustrations in a recent WHO publication (WHO, 1987) with the features of dental fluorosis presented by Fejerskov et al. (1988b).

Based on existing knowledge at the time, Dean included staining in his diagnostic criteria. As previously indicated, however, such posteruptive modification of the enamel "is not a diagnostic sign" (Eklund et al., 1987). The second problem in Dean's index relates to his "severe" category. Considerable confusion about the mechanisms by which fluoride affects human enamel has arisen from imprecise reading of the criteria provided by Dean. Dean et al. (1935) were clear that true hypoplasias were not a feature of human dental fluorosis despite their use of the same word to describe the pits. It is therefore very unfortunate that an illustration of Dean's severe category, in the above-mentioned WHO publication includes a classic true hypoplasia without commenting on this important diagnostic problem.

Apart from being more precise, sensitive and easier to use than Dean's index, the TF-index has the advantage that it also allows for comparisons to be made between the two indices (Wenzel and Thylstrup, 1982; Granath et al., 1985; Burger et al., 1987). This is particularly important if, in the future, it is felt necessary to make comparisons with the findings of earlier studies in which Dean's index has been used.

Recently, Horowitz et al. (1984) published what was stated to be a new classification system. However, when

comparing it with both Dean's and the TF-index, it is apparent that this method mixes the basic principles of the two indices. In their method plaque is not removed, nor are the tooth surfaces dried. As apparent when comparing the illustrations provided in their two publications (Driscoll et al., 1983; Horowitz et al., 1984) Dean's category "questionable" has been excluded. Otherwise their scores 1, 2 and 3 are illustration-wise identical to Dean's categories "very mild", "mild" and "moderate". As such it is hampered by the same weakness as Dean's index in being imprecise in its definitions of the various categories in the milder forms. According to the proposal by Horowitz et al. (1984), it seems that all enamel defects included in Dean's category "questionable" should be considered "sound" or normal. However, Driscoll and Horowitz (personal communications this meeting 1989) have informed us that they include Dean's category "questionable" in their category 1. According to Dean and Elvove (1937) "the quantitative aspects of a survey would probably be entirely lost, or judged erroneously, if the "questionable" cases were thrown into one grade or another ..". Thus in the same population in which the same individuals have been examined using both Dean's and Horowitz et al.'s indices, it would be expected that the prevalence of dental fluorosis using Horowitz' method should be the same or lower. It is intriguing that quite the reverse was the case in the data presented (Driscoll et al., 1983; Horowitz et al., 1984). The most important limitation is that Horowitz et al. (1984) proposed that staining should be taken as a diagnostic criterion so important that if present, it supercedes all the lower scores (i.e. 1, 2 and 3). This may explain the apparent contradiction mentioned above. That staining should be given such weight in the light of current knowledge about the posteruptive changes in fluorotic enamel is surprising. The risk with this approach is that it might overestimate severity in populations exhibiting staining but with otherwise relatively mild enamel changes, and would thus be inappropriate for use

under field conditions in a variety of different populations.

Histopathology and chemistry of human fluorotic enamel.

During the past two decades new information has been gathered about the light and electron microscopical changes in human enamel exhibiting various degrees of dental fluorosis (Kérébel et al., 1973; Fejerskov et al., 1974; 1975; Kérébel and Daculsi, 1976; Sundström et al., 1978; Sundström and Myhrberg, 1978; Thylstrup, 1978; Thylstrup et al., 1978). The overall findings support the concept that fluoride affects the forming enamel to cause porosity of the enamel. The degree and extent of porosity depends on the tissue fluid concentration of fluoride during tooth development. The structural arrangement of the crystals appears normal, but the width of the intercrystalline spaces increases - hence the pores. Likewise the arcade-shaped gaps which partly surround the enamel rods during normal enamel development (Fejerskov and Thylstrup, 1987) become widened (Fejerskov et al., 1974).

The pits found in more severe cases of dental fluorosis are a result of posteruptive breakdown of the surface enamel, the hypomineralized lesion being located deep to a well mineralized surface zone which is very fragile to mechanical stress (Thylstrup and Fejerskov, 1979; Fejerskov et al., 1983). This is consistent with the clinical finding that pits develop after tooth eruption (Thylstrup, 1983; Baelum et al., 1986). This observation is very important in deriving hypotheses about pathogenic mechanisms in dental fluorosis. Thus, if the old concept is perpetuated that dental fluorosis comprises hypoplastic enamel (Horowitz, 1986; WHO, 1987) it lends support to the concept that such "hypoplasias" are a direct result of a toxic damage to the secretory ameloblasts by fluoride. Already Dean (1934) and Dean et al. (1935) were stressing the fact that typical hypoplasias should not be included in his classification system, and true hypoplasias have not been found to be asso-

ciated with previous fluoride exposure (Cutress et al., 1985).

Based on our light and electron microscopical findings (Fejerskov et al., 1974) we therefore proposed that the hypomineralized regions of fluorosed enamel might be a result of an arrest of enamel maturation. During normal enamel development a substantial crystal growth takes place in the inner enamel following the gradual removal of proteins, while outer enamel is still being secreted. At the time of cessation of enamel matrix secretion at any given point in the enamel, the underlying enamel will be at a stage of development where there is a gradual decrease in mineral content from the E/D junction towards the outer enamel. At this stage the outer enamel is very hypomineralized (as compared with normal mature enamel) except for a very thin outer layer of surface enamel which even at this stage contains more mineral than does the subsurface enamel (Fejerskov and Thylstrup, 1987). From then on until eruption of the tooth, the "amelogenins" and water will have to be removed concomitant with crystal growth to allow the enamel to become fully mineralized (or mature). If this maturation process is interfered with by fluoride the result would from a histopathological point of view be identical with what characterizes dental fluorosis. In support of this hypothesis was the findings that human fluorosed enamel, when compared with normal mature enamel, had a similar total protein content, but the fluorosed enamel retained a relatively high proportion of immature matrix proteins (Eastoe and Fejerskov, 1984).

Recently the fluoride content has been examined throughout the enamel in teeth representing the complete range of macroscopically defined degrees of severity of dental fluorosis classified according to the TF-index (Richards et al., 1989). These data show that with increasing severity of fluorosis the fluoride concentration increases not only in the superficial enamel, but throughout the whole tissue. These findings are contradictory to previous observations by

Olsen and Johansen (1978) who did not find any association between fluoride concentrations in the outer 100 μm of enamel and the surface appearance of the teeth. The authors classified their teeth according to the modification of Dean's index proposed by Møller (1965). As previously stressed, discoloration is misleadingly included in this classification and this may have led to confusion as to the true degree of severity.

Because of the porous nature of fluorotic enamel, it would be expected that posteruptive uptake of fluoride might take place to a substantial degree. This was reflected in a rather great variation in fluoride concentration in severely affected teeth (TF scores 7-9) where substantial destruction of the surface enamel was recorded. The observation of increased fluoride concentrations at all enamel depths in teeth exhibiting a TF score 4 as compared with lower TF categories indicates, however, that the fluoride concentrations represent fluoride incorporated into the enamel prior to eruption. That this assumption holds true has recently been demonstrated in a study of unerupted human fluorotic teeth (Richards et al., 1988).

How fluoride may interfere with the complex processes of enamel formation and maturation cannot be resolved easily from human studies. Human clinical studies have demonstrated that it is possible to develop dental fluorosis when children have been exposed to fluoride supplements during the period when the teeth are undergoing enamel maturation (Ishii and Nakagaki, 1984; Larsen et al. 1985). It can be speculated that fluoride may affect the maturation ameloblasts by influencing their ability to remove protein and water from maturing enamel and/or may interfere with the ameloblast's capacity to produce proteolytic enzymes necessary to initiate amelogenin breakdown. It is surprising that a limited fluoride intake such as that obtained from a Danish fluoride supplement program (Thylstrup et al., 1979) should be sufficient to result in tissue fluid concentrations of fluoride of a magnitude which may have any

effect on enzymatic processes in vivo. Studies on rats indicate, however, that plasma fluoride concentrations as low as 1.5 μmol/L may be sufficient to induce enamel changes (Angmar-Månsson and Whitford, 1984) so it seems highly relevant to reevaluate the dose-response relationship in humans before hypotheses on pathogenesis of dental fluorosis are further developed.

Dose-response relationship in humans

There has been some concern expressed in recent years that in North America and Europe the prevalence of dental fluorosis may have increased slightly (Szpunar and Burt, 1987; Leverett, 1986). Such concern has arisen principally in the light of the increasing use of a variety of fluoride regimes in these populations. In North America, in particular, where rather large populations are provided with artificially fluoridated waters, the addition of a variety of topical (and systemic) agents might be expected to influence both the prevalence and severity of dental fluorosis (Whitford et al., 1987).

At the same time, an increasing number of reports have appeared from countries in which no organized fluoride prophylactic programmes have been implemented, indicating higher prevalences and severity of dental fluorosis in low fluoride areas than would be expected (Møller et al., 1970; Olsson, 1978; Glass, 1984; Subbareddy and Tewari, 1985; Manji et al., 1986a; b; c; Brouwer et al., 1988; Evans, 1988; Grobler et al., 1988)

The principle difficulty in interpreting recent and past data on dental fluorosis is that there have been misapprehensions about the nature of the dose-response relationship. The result of a long lasting low fluoride exposure in humans is first recordable in children after the age of 6, and precise estimates of the most severe effects of fluoride can only be obtained when the premolars and the second molars have erupted. This means that the dose responsible for such changes has to be estimated from a past

cumulative history of about 10-12 years during which period rapidly growing children will have been exposed to considerable changes in dietary patterns and practices (from weaning, bottlefeeding etc.). The relative contribution of food-borne fluoride for the development of dental fluorosis is still debatable. As seen from the literature (Taves, 1983; Smith and Ekstrand, 1988) it is difficult to arrive at a firm agreement as to how much fluoride is actually ingested - and the bioavailability of fluoride in food is as yet uncertain. However, from epidemiological studies throughout the world there is a positive relationship between water-borne fluorides and the occurrence of dental fluorosis (Dean, 1942; Møller, 1965; Richards et al. 1967; Myers, 1978; Thylstrup and Fejerskov, 1978; Manji et al., 1986a; c; Larsen et al. 1987).

In order to understand the relationship between fluoride exposure (dose) and dental fluorosis (response) it is obvious that we should consider all signs of dental fluorosis, including its earliest manifestations.

It is a prevailing concept that temperature affects the prevalence and severity of dental fluorosis simply as a result of variations in daily consumption of water (Galaghan et al., 1957). Galaghan and Vermillion (1957) formulated an equation which permits the calculation of water-intake as a function of temperature (Table 1). Applying this formula to data for example from Dean's studies in Illinois and Ohio (Dean et al., 1941; 1942), it is possible to estimate the dosage of fluoride from water (mg F/kg body weight) associated with dental fluorosis in these communities at that time. From Fig. 2 it is apparent that with an intake from water equivalent to 0.02 mg F/kg b.w. a prevalence of about 40-50% was observed (about 15-25% excluding questionables). The community index values, Fci, at this intake would be between 0.3 and 0.4 (Fig. 3). Even if in small children fluoride from food might double this dose (Ophaug et al., 1985), these data from Dean suggest estimates that are considerably below that of Forsman (1977)

who stated that dental fluorosis is unlikely to occur at a dosage below 0.1 mg F/kg body weight.

In Fig. 4 we have included also data from Richards et al. (1967) adjusted for temperature variations and Butler et al. (1985). Two striking features about the data are apparent in this figure. First, irrespective of the source of the data it is clear that the slope of the regression line for the association between fluoride dose and dental fluorosis is such that even with very low fluoride intake from water a certain level of dental fluorosis will be found (r^2 = 0.87). Secondly, the dose-response relationship is linear. These data indicate that for every increase of dose of 0.01 mg F/kg b.w. an increase in Fci of 0.2 will be expected.

From the studies by Aasenden and Peebles (1974) it may be calculated that the median daily dose of fluoride from tablets would range between 0.042 to 0.070 mg F/kg b.w. with an average dose over this period of 0.056 (Baelum et al., 1987). This fluoride dosage resulted in an Fci value of 1.23 (based on the Fci weights recommended by Dean), a value which is consistent with the expected value for the Fci based on the epidemiological evidence in Fig. 4. Similarly, in a Swedish study on fluoride tablet supplementation (Granath et al., 1985) the median dosage of fluoride ranged from 0.024 to 0.042 mg F/kg b.w. resulting in an Fci value of about 0.4. Thus, it would appear that irrespective of whether fluoride is obtained from drinking water or from supplements, the expected outcome in terms of levels of dental fluorosis is approximately the same.

Currently, the recommendation in the USA for fluoride supplements in for example a < 0.3 ppm fluoride area (ADA, 1982) is that 0.25 mg of fluoride should be provided as supplements from birth to 2 years of age and thereafter 0.5 mg should be provided until the age of 3, thereafter 1 mg/day. In those receiving such supplements we would expect, based on Fig. 4, that the Fci values would be approximately 1.0, with a prevalence of about 70-80%.

However, such values will of course only be found provid-

ed that the dose-regime is strictly followed and that there is a 100 % compliance. The data provided by Holm and Andersson (1982) clearly indicate that the outcome in terms of prevalence of dental fluorosis is highly dependent on both time of initiation of fluoride supplementation as well as on the degree of compliance.

In the above calculations we have purposely used data only from studies in which Dean's original classification system has been used. As we have indicated in Figs. 3 and 4 the dose-response relationship in the three data sets suggests a linear relationship and imply therefore that there exists no "critical" value below which the effect of fluoride on dental enamel will not be manifest.

In presentation of Dean et al.'s data in order to estimate dose-response relationships Hodge (1950) used a logarithmic scale for the x-axis (dose). The effect of such transformation of the data in Fig. 4 is presented in Fig. 5. The conclusions one would draw from this logarithmic transformation would be, that the relationship is curvilinear such that below a certain level of fluoride intake the toxic effects of fluoride would be minimal and in any case below about an Fci value of 0.4-0.6 - ironically the level which Dean suggested to be of "borderline public health significance". The question arises, however, whether it is appropriate to use a log-transformation where the observed relationship is quite clearly already linear.

Taking the above considerations into account, it is to be expected that in populations in which additional fluoride has been prescribed an increase in prevalence and severity of dental fluorosis will occur (Szpunar and Burt, 1987; Leverett et al., 1988). Furthermore, water fluoride levels are seldom stable. Larsen et al. (1988) have recently shown that there may be considerable variations with time in the levels of fluoride in any given drinking water supply, even in artificially fluoridated water supplies.

There is no doubt that there may be a number of factors which influence the susceptibility of individuals and

populations to dental fluorosis, and which may account for higher than expected levels of dental fluorosis being found. Recently, Manji et al. (1986c) have presented epidemiological data indicating a positive association between fluoride, altitude and dental fluorosis in Kenya. The biological explanation for this is intriguing. While the association may reflect that individuals living at differing altitudes simply have different water consumption and dietary habits, it could also reflect that altitude may make individuals more susceptible to low fluoride exposure. Several other factors may be important to consider when unexpectedly high prevalence and severity of dental fluorosis is reported. There is no doubt that the bioavailability of ingested fluoride plays a considerable role. Thus, frequency and composition of meals will highly influence bioavailability of concomitantly ingested fluoride. A recent example is the demonstration that fluoride ingested from toothpaste is bioavailable to almost the full extent if ingested on an empty stomach whereas if taken in relation to a meal the bioavailability is reduced to about 50 % (Spak and Ekstrand, 1988). When comparing the frequencies and quantity of food intake, during the day, in children in industrialized countries with those of children in developing countries, it is apparent that the latter spend long periods of the day with relatively insubstantial amounts of food in the stomach (Manji, 1988). This alone seems to us to be of importance when trying to explain apparent variations in susceptibility to fluorides. It is possible that malnourishment may increase the susceptibility of populations to dental fluorosis, but this hypothesis has yet to be substantiated.

Finally, but probably equally important, the way the body handles fluoride in hot climates, where for example urinary excretion may be much less than seen in temperate climates, may play a role. Furthermore, little is known about the relative role for fluoride susceptibility in man of acid-base variations in body fluids.

Conclusion

The dose-response relationship has been estimated from data collected using a system in which the ordinality is not consistent with the actual biological effects of fluoride on developing dental enamel. In order to obtain more precise estimates on the dose-response relationships, large scale studies in different parts of the world should be conducted using the TF-index along the lines recently proposed by us (Fejerskov et al., 1988b).

Even when using available data from the USA, however, the dose-response curve presented shows a linear relationship and indicates that it may no longer be appropriate to seek for a definite lower border below which no individual in a population would not exhibit some signs of fluoride-induced changes. However, it is important to appreciate that these changes are essential to identify only when the biological effects of fluoride on mineralizing tissues are to be interpreted. Unless the existence of such changes is appreciated, we will not be able to improve our knowledge of how fluoride affects mineralization of teeth and bone in humans. At what level the changes should be considered of public health concern will strongly depend on the type of society we are dealing with.

Finally, it is necessary to establish better studies in humans to identify other factors which alone or in combination are able to influence the susceptibility of the individual to fluoride. Likewise attempts should be made to identify other factors which alone or in combination may be able to produce slight changes in porosity in the enamel similar in surface appearance and distribution within the mouth to those caused by fluoride.

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Legends

Fig. 1a, b - Percentage of teeth with dental fluorosis recorded according to Thylstrup and Fejerskov (1978) in an area of Denmark with about 0.1 ppm F in drinking water (a) and in an area with 1.5-2.0 ppm F (b). From Larsen et al. (1985), Larsen et al. (1986). The numbers along the x-axes refer to tooth numbers with + indicating maxillary and - = mandibular teeth.

Fig. 2 - The relationship between estimated daily dose of fluoride from drinking water and resulting prevalence of dental fluorosis in the populations examined by Dean et al. (1941; 1942). The category "questionable" is included.

Fig. 3 - The relationship between estimated daily dose of fluoride from drinking waters and the community index (F_{ci}) of dental fluorosis as obtained from Dean et al's original data (1941; 1942).

Fig. 4 - The community index of dental fluorosis (F_{ci}) plotted against daily dose of fluoride from drinking waters as estimated from data originating from Dean et al. (1941,; 1942), Richards et al. (1967) and Butler et al. (1985). The dose-response relationship is linear and data indicate an increase in F_{ci} of 0.2 for every increase of dose of 0.01 mg F/kg b.w.

Fig. 5 - The same data presented in Fig. 5, but the x-axis has been transformed on a logarithmic scale.

Fig. 1a

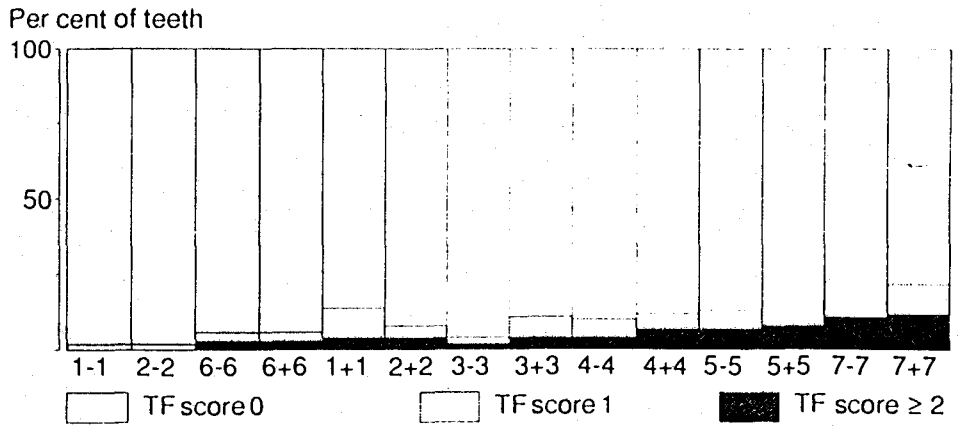
Per cent of teeth
100
50
1-1 2-2 6-6 6+6 1+1 2+2 3-3 3+3 4-4 4+4 5-5 5+5 7-7 7+7
TF score 0
TF score 1
TF score ≥ 2

Fig. 1b

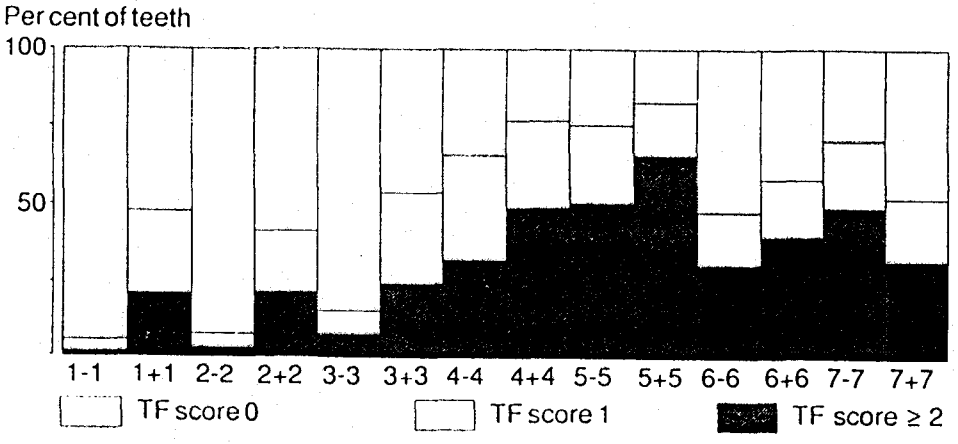
Per cent of teeth
100
50
1-1 1+1 2-2 2+2 3-3 3+3 4-4 4+4 5-5 5+5 6-6 6+6 7-7 7+7
TF score 0
TF score 1
TF score ≥ 2

Fig. 2

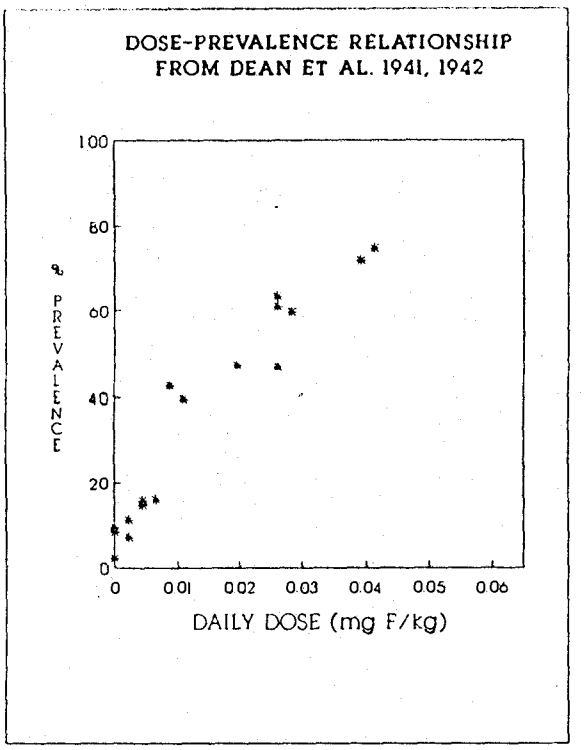

Fig. 3

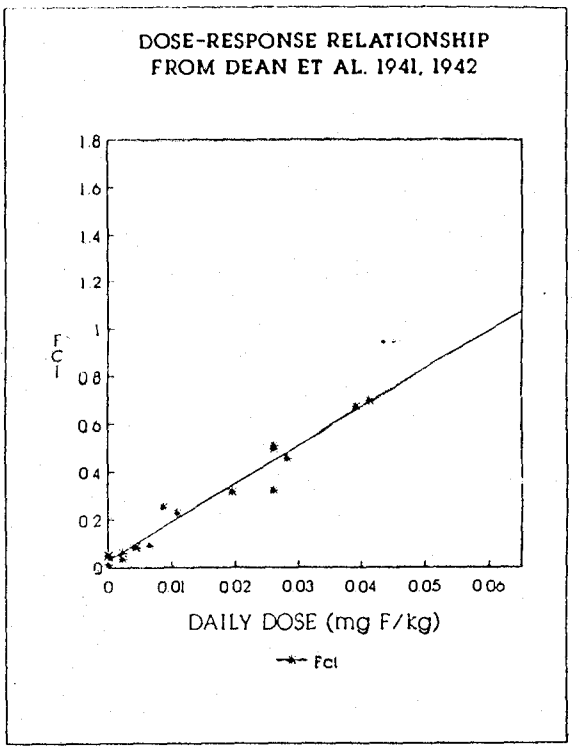

Fig. 4

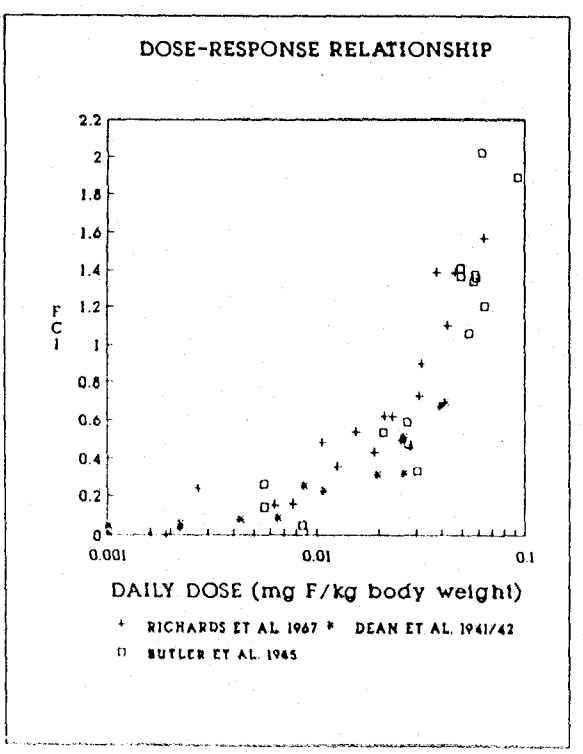

Fig. 5

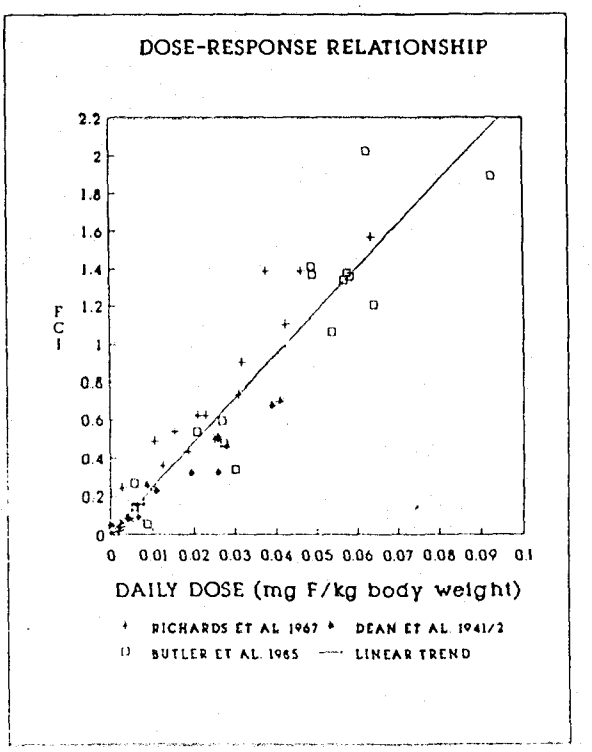

TABLE 1

FORMULA USED FOR COMPUTING
THE DAILY DOSE OF FLUORIDE FROM DRINKING WATERS

DOSE (mg F/kg body weight) = WATER F CONC. (mg F/L) x WATER INTAKE (L/kg body weight)

Galagan and Vermillion provided a formula from which the WATER INTAKE could be computed based on knowledge of the MEAN MAXIMUM AIR TEMPERATURE:

WATER INTAKE (fl.oz./lb body weight) = -0.038 + 0.0062 x MEAN MAXIMUM TEMPERATURE ($^{\circ}$ Fahrenheit)

Using the following relationships:

1 fl.oz (USA) = 0.0295735 litres
1 lb. = 0.4535924 kg

the above formula may be transformed:

WATER INTAKE (L/kg body weight) = -0.0024775 + 0.00040423 x MEAN MAXIMUM TEMPERATURE ($^{\circ}$ Fahrenheit)

Therefore, the equation

DOSE (mg F/kg body weight) = WATER F CONC. (mg/L) x WATER INTAKE (L/kg body weight)

is equivalent to

)SE = WATER F CONC. x (-0.0024775 + 0.00040423 x MEAN MAX TEMPERATURE)
;/L) (mg/L) ($^{\circ}$ Fahrenheit)

THE EPIDEMIOLOGICAL FEATURES OF DENTAL CARIES IN AFRICAN AND CHINESE POPULATIONS: IMPLICATIONS FOR RISK ASSESSMENT

Manji, Firoze[2], Fejerskov, Ole[1], Baelum, Vibeke[1], Luan, Wen-Min[3], Chen, Xia[3].

[1]Dept. of Oral Anatomy, Dental Pathology and Operative Dentistry, the WHO Collaborating Centre for Oral Health Planning and Research in Third World Countries, Royal Dental College, Vennelyst Boulevard, 8000 Aarhus C, Denmark, [2]Kenya Medical Research Institute, Medical Research Centre, PO Box 20752, Nairobi, Kenya, [3]Beijing Hospital, Beijing, China.

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INTRODUCTION

Risk is defined as the probability of an event occurring within a stated period of time (Last, 1983). Thus far, attempts to identify individuals or groups at risk to dental caries have met with limited success. One of the principle difficulties in measuring risk in dental caries arises from the very nature of the disease itself, being characterized essentially as a process with an imprecise beginning and an indefinite and often unpredictable outcome (Manji, Fejerskov & Nagelkerke, 1989b).

The majority of studies on risk assessment in dental caries have been conducted in populations of industrialized countries. In such populations the nature of the disease process, and the role of risk factors in this process, may often be masked by interference through formal dental care and through the administration or availability of numerous therapeutic measures. There may, therefore be considerable advantages to considering the nature of the disease in populations little exposed to such extensive interventions, and which have little tradition of thorough oral hygiene practices.

In this contribution we consider the epidemiological data on dental caries in populations of Africa and China - continents comprising a major part of the world's population - in order to assess whether it may be justified to postulate the existence of high or low risk "groups or individuals" from the available evidence. We show that the evidence for "dramatic" increases in the prevalence of dental caries - which might be interpreted as indicative of such populations being at "high risk" - is in reality equivocal. We argue that methods used for the diagnosis of dental caries are of central importance in risk assessment, and failure to bear this in mind has led to many of the

characteristic features of dental caries in such populations to be misinterpreted as indicative of differences in susceptibility to dental caries. There is now considerable evidence to demonstrate that caries in adult populations should be considered to be ubiquitous, but with a generally slow rate of progression. The observation that there are between individual differences in disease experience is an invariable feature of epidemiological data. However, we suggest that before such variations can be attributed to variations in risk, due attention should be paid to the fact that a considerable proportion of the observed variation in disease experience can be attributed to random effects.

TRENDS IN DENTAL CARIES AS A MEASURE OF CHANGING RISK

It is widely held that dental caries in developing countries is increasing (Barmes, 1979, Barmes, 1982, Heloe & Haugejorden, 1981, Sheiham, 1984a, 1984b) at a rate which is "frightening" because, "for the first time ever, the average 12-year-old in underdeveloped countries, where 80% of the world's children live, had a higher dental caries score .. than those in industrialized countries" (Sheiham, 1984a). Given such a scenario, we would expect that during the last two decades there has been a rapid accumulation of risk factors in developing countries which, in one way or another, has increased the likelihood of such populations developing caries. Although several attempts have been made to identify the nature of such risk factors (Enwonwu, 1981, Sreebny, 1982, Sheiham, 1984a, Narendran, 1983, Manji, 1986) it may be enlightening to assess the evidence in Africa and China for such trends in dental caries experience.

The principle evidence for this view comes from the data accumulated by the WHO Global Epidemiology Bank (Barmes, 1979, Barmes & Sardo Infirri, 1977, Heloe & Haugejorden, 1981, Barmes 1982). The main difficulty with using these

data for assessing the extent and direction of trends is that in most cases rather limited information is usually available about the characteristics of the specific study populations to which the data refer. Strict comparison of the results of different studies conducted at different times in the same country, or of the results of studies conducted at approximately the same period in different countries, is inevitably difficult, and only very tentative conclusions about trends can be drawn. For example: the data bank provides for Kenya in 1952 an estimate of the mean DMFT score of 12-year-olds of 0.1. This figure has been compared with that of a mean score of 1.7 in 1973 as evidence of increases in caries experience (Barmes, 1979). However, the 1952 study (attributed to Mackay & Martin, 1952) was conducted in a small constal village (Msambweni), whereas the data for the 1973 study (Gad, 1973) originated from a survey of 71 children in the highlands of Kenya, an economically more developed region of the country. To what extent the differences in mean DMFT scores were therefore a reflection of changes in caries experience with time, or merely a reflection of mere regional variations in caries experience within the country, is unclear.

Assessment of the trends in dental caries experience from the published literature is also not without its difficulties. Table 1 summarizes the findings of studies conducted in African countries in which persons aged approximately 12 years had been examined. These data originate from studies conducted for a variety of reasons, with different aims and objectives, and only a few have been designed with the specific intention of obtaining national estimates. Consequently, the external validity of the findings of most such studies is restricted, i.e. the extent to which inferences from the data can be made is restricted to the special sections of the population from which the sample was obtained, and may not necessarily reflect

national trends in a country at the time of the study.

The above is important because there are usually considerable variations in caries experience of different social groups, or of people in different regions of a given country. Thus for example, Akpabio, Gardiner & Adenyika (1982) reported that in Nigeria the mean DMFT score of 12-year-olds ranged from 0.9 in the low socio-economic groups to 2.8 in the high. Furthermore, they found marked regional disparites in caries experience, with a DMFT score of 0.7 in the Cross River State and 3.2 in Lagos (the capital city). Similarly, Tirwomwe et al. (1988) found a considerable range in the mean DMFT in different parts of the country: for example, in 35-44-year-olds, they found a DMFT of 1.74 in Mbale, while in Kabale a high of 5.82 was obtained. What was particularly interesting in this study was that the caries experience of all age groups from the capital city (Kampala) was found to be lower than in many of the rural regions of the country. This pattern was similar to that observed in China where the caries experience of adult and elderly people living in rural areas was significantly greater than that of their urban counterparts (Chen et al., 1989). Such findings are in contrast to the usual pattern in which caries in urban areas tends to be higher than rural (Enwonwu, 1981). Thus where increases in levels of dental caries have occurred in a country, these may not necessarily have been the result of a rate of urbanization which is "taking place at an explosive pace in most developing countries" (Heloe and Haugejorden, 1981).

One cannot, therefore, automatically assume that "urbanization" or "socio-economic status" constitute "risk factors" for dental caries. These data demonstrate well the importance of distinguishing between <u>risk factors</u> which make some functional biological contribution to the causation of disease, and <u>confounders</u> which are factors which may (in a

given society or at a given time) be associated with the disease, but which do not play any biological or functional role in causality.

Comparison of the results of the various studies in Africa (Table 1) is also problematic because of the differences in diagnostic methods used. Some have used criteria in which precavitation lesions have been ignored (e.g. Akpabio et al., 1982, Frencken, Manji & Mosha, 1986, Manji, Mosha & Frencken, 1986, Manji, Fejerskov & Baelum, 1989a, Chironga & Manji, 1989, Hobdell 1981). Others have made positive diagnoses at an earlier stage in lesion progression ("sticky fissures") (e.g. Akpabio, 1970, Akpata, 1979, Emslie, 1966), while still others have classified each lesion on an ordinal scale indicative of the degree to which the lesion appears to have progressed (e.g. Gadegaard & Fejerskov, 1983, Frencken, 1988, Manji, 1988, Tirwomwe et al., 1988, Manji et al., 1989a). Much of the variation of the mean DMF scores obtained in the different studies on the continent could, therefore, be accounted for by differences in diagnostic methods used.

Rather few studies in Africa have been conducted with the specific purpose of obtaining national estimates (Nigeria: Akpabio et al.,1982, Uganda: Moller, Pindborg & Roed-Petersen, 1972, Jensen et al. 1973, Tirwomwe et al. 1988). Each of these surveys have demonstrated relatively low levels of caries (at least in terms of the occurrence of frank cavities) in both child and adult populations.

In Nigeria two fairly extensive surveys have been conducted separated by a period of about 10 years (Akpabio, 1970, Akpabio et al., 1982), although only the second was designed as a national survey. The findings indicated that there had been increases in caries experience, with the mean DMFT scores of 10-14-year-olds, for example, increasing from 0.85

in the late 1960s to 2.0 in the early 1980s (Table 1). Conclusions from these surveys ought to be drawn cautiously, however, because of the considerable differences in the population groups studied and in the diagnostic criteria used.

In Uganda two national surveys have also been conducted, the first in the late 1960s (Moller et al., 1972, Jensen et al., 1973) and the second in 1987 (Tirwomwe et al., 1988). Although different sampling methods and diagnostic criteria had been used in these surveys, a comparison of the findings from those regions included in both surveys (Table 2) indicate that there had been, over some 20 years, small increases in dental caries experience in all comparable age groups, although occurring unevenly throughout the country (Tirwomwe et al., 1988).

More definitive evidence about trends in dental caries experience comes from studies in which the same population groups or study areas have been examined on two separate occasions using similar diagnostic criteria. Olujugba & Lennon (1987) recently reported marked increases in the mean DMFT of 12-year-olds from 0.13 to 2.15 in schools in Nigeria over a relatively short period of time (1977 to 1983). In 5-year-olds, however, they found no increases in caries experience over the same period except in those from the higher socio-economic groups.

In South Africa, Cleaton-Jones et al. (1983) compared the data from a series of cross-sectional studies in which 2- to 5-year-olds had been examined using the same diagnostic criteria. They found a decline in caries experience of the primary dentition in white children between 1976 and 1981, whereas that of the non-whites increased over the same period. Richardson et al. (1984) reported, furthermore, that such changes in caries experience had not been paralleled by

changes in sugar consumption in these populations. Although each of the cross-sectional studies were conducted in the same study areas, it is impossible to say whether in the non-white areas similar populations had been examined on each occasion: the non-white areas tend to experience considerable turn-over in populations as a consequence of the "apartheid" migrant labour laws (Magubane, 1979). It is unclear to what extent such changes in populations may have accounted for the observed changes in the caries experience, and for the apparent lack of relationship between the caries experience and sugar consumption noted by Richardson et al. (1984).

In Swaziland, studies were conducted by Bindslev et al. (1988) in the same regions of the country as had been surveyed 10 years previously by Klausen & Fanoe (1983), using similar diagnostic criteria. Comparison of the findings of the two studies indicated a marked reduction in mean DMFS scores of 14-year-olds (Table 1).

In Kenya, no changes in the dmft scores of 6-year-olds, nor in the DMFT scores of 12- and 15-year-olds were observed in either rural or urban areas in surveys conducted in 1984 and 1986 (Manji, 1988), while in Tanzania Frencken (1988) found no changes in caries experience over two years in children aged 7 to 9 years. Cross-sectional studies of children in Tanzania conducted 4-years apart in Dar es Salaam (Mosha, 1986), and 10-years apart in Arusha (Mosha et al., 1988), also provided limited evidence of any increases in caries experience. More recently, Mosha (1989) reported significant reductions in the mean DMFS of 12-year-olds in Dar es Salaam between 1984 and 1986. Comparing her findings with those of Barmes (1970), Marseilles (1984) found marked reduction in caries experience of 12-year-olds in Zaire (Table 1).

From the available evidence it seems, therefore, that there

have been both increases and declines in caries experience in Africa. Where there have been increases in caries experience, these have been on a scale (except, perhaps, in the study by Olujugba & Lennon, 1987) that may not perhaps warrant the description of "frightening" or "dramatic" (Sheiham, 1984a, Heloe & Haugejorden, 1981). Ignoring for the moment those aspects which do not allow for strict comparisons, the data from Africa indicate that in Nigeria, Ghana, Ethiopia, South Africa and to some extent in Uganda, there is evidence to suggest that caries experience has increased with time. In contrast, in Kenya and Tanzania, in Swaziland and in Zimbabwe, the evidence suggests a rather limited increase or, in some cases, evidence of a decline in caries experience.

It should be borne in mind that the low levels of caries observed in much of Africa occurs in the context of there apparently having been a marked increase in the availability of sugar in developing countries: according to one study, sugar consumption had increased from an estimated 22.3 kg/head/year in 1965 to 27.4 kg/head/year in 1981, with African countries experiencing the largest proportional growth (Narendran, 1983). In Kenya, per capita sugar consumption increased nearly two-fold during twenty years, while published epidemiological data over the same period indicates a likely decline in dental caries experience of 12-year-olds (review: Manji, 1986).

Data from China are rather difficult to interpret especially as mean data for a very wide age range (6 to 17 years of age) are usually presented. for example, in Guangdong Province, Zheng et al. (1986) reported a mean caries experience in primary teeth ranging from 3.0 to 3.7, with a prevalence of 77%to 84%, whereas in the permanent dentition the mean DMFT ranged from 0.5 to 0.8?, with a prevalence of 25% to 37%. Similarly, among 12 to 14-year-olds in Wuhan,

Wang et al. (1986) reported a mean DMFT of about 0.5, with a prevalence of 27%. In general, it seems that caries experience of children is still low (Wei, Yang & Barmes, 1986). Cross-sectional data on caries in several thousand children in the Beijing area in studies some 30 years apart indicate, moreover, that the prevalence of caries has remained at about 30%.

From such a review, we might perhaps gain the impression that the general characterization of caries in African and Chinese populations is that of relatively low caries levels. Does this indicate that sizeable proportion of the populations are, in some hitherto unknown way, "resistant" or lack a "susceptibility" to dental caries? Can these data be used for identifying potential "risk groups and individuals"? Before such questions can be addressed, some attention has to be given to the issue of what it is we are measuring, and whether the results of epidemiological data do indeed indicate any unexpected degree of resistance or susceptibility in these populations.

THE DEVELOPMENT AND PROGRESSION OF CARIES: THE IMPORTANCE OF DIAGNOSTIC METHOD IN RISK ASSESSMENT

It is well known that in any study of association where disease is either imprecisely measured (or where a "proxy" variable is used), the degree of association between the parameters will be considerably weaker than when the "real" measure of the disease is used. This issue is of crucial importance in any discussion about risk in dental caries.

It has become increasingly customary in epidemiological surveys for a positive diagnosis of caries to be made only when frank cavitation is found. It has been argued that in such studies the inclusion of enamel lesions in diagnosis results in poor reproducibility between and within examiners

(Palmer, Anderson & Downer, 1984, WHO, 1987, review: Pitts & Fyffe, 1988), although the results of several studies have not supported this view (Backer-Dirks, 1964, Haugejorden & Slack, 1975, Espelid & Tveit, 1986, Manji, 1988, Manji et al., 1989a, Pitts & Fyffe, 1988). If the purpose of the survey is to estimate in the population the number of cavities needing restorations, then ignoring precavitation lesions may be quite satisfactory. If, however, such information is to be used for the purposes of assessing risk (either relative or absolute), then the method if diagnosing caries is clearly vital.

First of all, ignoring the early signs of disease will mean that the caries experience will be considerably underestimated. Table 3 summarizes the findings of three recent studies of adults and children in East Africa in which DMF scores and per cent prevalence are presented based on including and excluding enamel lesions. It is apparent that in each of these studies, and in every age group, the mean DMF scores appear to have been considerably reduced where enamel lesions are ignored. There is, similarly, a considerable difference between the apparent prevalence of caries when enamel lesions are included and when excluded, although the difference becomes less marked with increasing age. Where, for example, enamel lesions are excluded, a misleading impression is obtained that only 26.5% of 23-year-old city-dwellers have caries in their permanent teeth. However, where the enamel lesions are included, over 75% apparently have had some caries experience of their permanent teeth. Such differences in apparent prevalence are of the same order of magnitude as those estimated in studies of adults in Hong Kong of comparable age (Pitts & Fyffe, 1988).

The argument that the earlier clinical signs of caries should be included in the diagnosis of caries is, however,

not the same as proposing that diagnosis should be reduced merely to a yes-no classification at an earlier stage of lesion progression (for example, as used by Akpabio (1970), Akpata (1979), Cleaton-Jones et al. (1984a)). To do so would result also in a considerable loss of information. Perhaps the best demonstration of the need to have information on the different stages of lesion progression has been provided in the Dutch water fluoride studies (Groeneveld, 1985) in which no measurable effect of fluoride could be demonstrated in test and control groups when the total number of lesions of all types was considered. When enamel lesions were excluded, a marked difference between the groups was apparent. These studies demonstrated that by taking account of the different stages of lesion progression, fluoride could be shown to exert an effect by interfering with the progression rate of caries lesions, rather than by "preventing" caries.

Although the apparent prevalence of caries in a population will appear considerably larger when the earlier signs of caries are included in the diagnostic threshold, it should be recognized that the actual caries experience of the population will nevertheless be underestimated. Populations in developing countries are usually characterized by having generally poor oral hygiene, with most surfaces of the teeth covered with plaque throughout life (Baelum, Fejerskov & Karring, 1986, Baelum, Fejerskov & Manji, 1988a). It would be expected, therefore, that from a very early period after eruption, some degree of caries activity (de- and remineralization) will occur at different sites from time to time. This concept that has support from histological studies of human enamel (Silverstone & Fejerskov, 1988) and studies of the interface between plaque and dental hard tissues (Nyvad & Fejerskov, 1989). Moreover, it is supported by a theoretical model for caries lesion development which sought to simulate the effects of plaque on loss and gain of

mineral from dental hard tissues (Manji et al., 1989b). Many of these minute lesions may well remain "subclinical" throughout life, whereas others may progress to become clinically visible. In due course, some may also advance to cavitation and more extensive involvement of the dental tissues. The proportion of lesions progressing to any particular stage may be dependent on a variety of factors such as the quality of plaque, the dietary practices of the individual, and the availability of fluoride, as well as the particular anatomical features of the site. The outcome, however, is usually unpredictable in any given individual.

In this context, estimates of prevalence of caries based on the number of lesions which are clinically diagnosable in a population indicates only the prevalence of lesions which have progressed to such a stage, and not the actual caries experience of the individuals in that population. It is inaccurate, therefore, to speak of "caries-free" individuals in such populations, except perhaps when referring to the very young.

The implications of this concept are that caries in most third world populations should be considered as an ubiquitous disease. As is apparent from Table 3, this ubiquity is clinically apparent in many populations from an early age, and is overtly apparent in those over the age of 15 year. The fact that the majority of studies have reported relatively "low" DMF scores in African populations indicates, therefore, that the rate of progression of caries generally tends to be slow.

Failure to bear in mind the importance of diagnosis has led to a number of misconceptions about the distribution of caries in third world populations and, consequently, to misleading conclusions about the relative susceptibility of these populations to dental caries. We discuss here some

important examples which have received considerable attention in the literature recently:

"No association" between caries and mutans streptococci: Given the ubiquitous nature of dental caries in populations exhibiting poor oral hygiene, it should not be surprising to find that cariogenic micro-organisms, such as the mutans streptococci, have also been found to be virtually ubiquitous in populatons of third world countries (Kilian, Thylstrup & Fejerskov, 1979, Reichart & Gehring, 1984, Carlsson, Olsson & Bratthall, 1985, Ibrahim, Bratthal & Carlsson, 1985, Matee et al., 1985, Reichart et al., 1985, Brathall et al., 1986, Carlsson et al., 1987, Beighton et al., 1989, Frencken, 1988, Manji, 1988). Furthermore, the lack of an association (or, at best, a weak one) between these micro-organisms and caries observed in these studies is to be expected if caries is equally ubiquitous, and explains why these micro-organisms do not appear to be determinant (or predictors) of the disease despite the evidence of their potential aetiological role.

Primary teeth "more" affected than permanent: Several studies have drawn attention to the fact that in many third world populations caries experience of the primary dentition is usually greater than that found in the permanent dentition (Akpabio, 1970, Jensen et al. 1973, Akpabio et al., 1982, Manji, 1984). The phenomenon has been taken either as an indication of a greater susceptibility of primary teeth to caries, or as a demonstration of the rising levels of dental caries in the population. However, as is apparent from Table 3, a higher caries experience in the primary than in the permanent teeth is only apparent when enamel lesions are excluded from the diagnosis of caries. When such lesions are included, there is little evidence of a greater caries experience of the primary as compared to the permanent dentition. At first glance these data might be

taken to indicate that caries progresses more rapidly in the primary dentition than in the permanent. However, since the enamel of primary teeth is considerably thinner than that of the permanent, a lesion progressing at any given (average) rate would be expected to reach the dentine sooner in primary teeth than in permanent. Thus there appears to be little evidence to support the hypothesis either that the caries experience of primary teeth is greater than that of the permanent, or that primary teeth are any more susceptible to caries than permanent teeth.

Second molars "more" affected than first molars: The slow rate of lesion progression in these populations occurs, usually, in the context of a relatively abrasive diet with a high fibre content (Kusin et al., 1984, Manji, 1988). It would be expected, therefore, that with time the clinical evidence of some of the early caries lesions which have not progressed rapidly will be ground away by the forces of attrition and abrasion. That this does indeed occur is apparent from studies of adult populations in Kenya (Manji et al., 1989a). In a number of studies of African populations (Jensen et al., 1973, Westwater, 1977, Akpata & Jackson, 1978, Cleaton-Jones & Walker, 1980), and newly arrived Vietnamese refugees in Norway (Selikowitz, 1984), a higher prevalence of caries has been reported to occur in the second than first molars. Various hypotheses have been proposed to explain this phenomenon including ill-defined genetic factors (Akpata & Jackson, 1978), the degree of malnourishment during the period of calcification of the molars (Westwater, 1977), relative inaccessibility of the second molars to effective oral hygiene procedures (Enwonwu, 1981), and the post-eruptive period during which the fissures of molars are colonized by mutans streptococci (Loesche, 1986). In recent studies in Kenya (Manji, 1988) it was found, however, that this phenomenon was only observable in non-city dwellers, and only when the early precavitation

signs of caries was included in the diagnosis of caries, when the later were excluded, the more conventional "western" pattern (first molars being more affected than the second) was found. Given a slow rate of progression of caries lesions and the presence of a relatively abrasive diet, we would expect that a proportion of the enamel lesions in the first molars will eventually "disappear" (or, at least, clinically they will be less apparent) by the time the second molars have erupted and come into occlusion (Muya et al., 1984). Since the second molars will not have been subject to the same period of attrition and abrasion as the first, many of the enamel lesions in the second molars will inevitably be clinically more visible than those in the first. This would then give the misleading impression, especially if the diagnostic threshold includes pre-cavitation signs, that the second molars were in some way more "susceptible" to caries. In the context of a diet comprising softer (e.g. processed) and less abrasive foods, we would expect that the first molars would be more affected than the second since the early lesions in the first molars would not have been worn away to the same extent, a hypothesis that is consistent with the observations made among city-dwellers in Kenya (Manji, 1988).

Diet and dental caries: A number of studies conducted in South Africa have reported an apparent lack of association between caries and sugar consumption (Retief, Cleaton-Jones & Walker, 1973, Cleaton-Jones et al., 1978, Cleaton-Jones, Richardson & McInnes 1981, Cleaton-Jones et al., 1983, 1984a, 1984b, Richardson et al., 1978a, 1978b, 1984, Richardson, Sinwel & Cleaton-Jones 1981, Richardson & Cleaton-Jones, 1979, Walker et al., 1981). These findings have been interpreted as indicative of some genetically mediated "immunity" of African populations to caries (Richardson, 1982). Recent reviews of these studies have shown, however, that the published data from South Africa do

not themselves support such conclusions (Newbrun 1978, Hackett & Rugg-Gunn, 1982, Manji, 1988), in particular as frequency of consumption of sugars have almost always been found to be positively associated with caries in the South African studies (Walker et al., 1981, Cleaton-Jones et al., 1984a, 1984b). There are, moreover, considerable limitations in the method of eliciting dietary information employed in these studies (review: Manji, 1988), based as it is on a method originally designed for relating sugar consumption to the occurrence of _acne vulgaris_ and _seborrhoeic dermatitis_ in western populations (Bett, Morland & Yudkin, 1967). Of particular note is the fact that all of these studies have endeavoured to relate the consumption of sugar at a given time with the occurrence of caries at the same time. As Rugg-Gunn (1983) has pointed out, it seems unreasonable under such circumstances to expect an association to be found since the response to cariogenic challenge provided by the diet will only be measurable some years later. Recent studies in African populations in Kenya have demonstrated that sugar _per se_ is not associated with caries when considered as a single parameter on its own, however, where account is taken of all the foods consumed and the way in which sugars are consumed in the context of that diet, then a clear association is demonstrable (Manji, 1988). Furthermore, little association was demonstrable when all stages of lesion progression were considered, whereas when enamel lesions were excluded, a positive association was apparent. This may be of some importance in understanding the findings of the South African studies in view of the fact that the method of diagnosis of caries employed there have consistently been based on the inclusion of the earlier signs of caries, but without differentiating these from the later stages of the lesions. There seems, thus, little evidence to indicate that African populations are any more or less susceptible to the influence of diet than other populations. This view is supported by the findings of a

number of other studies on the relationship between diet and dental caries in other African populations (MacGregor, 1963, Enwonwu, 1974, Henshaw & Adenubi, 1975, Olsson, 1979, Gupta, 1985, Salako, 1985, Frencken et al., 1986)

To conclude this section, most of the apparent "peculiarities" of the epidemiological characteristics of caries in developing countries can be easily explained if the dynamics of the processes involved in the development and progression of caries lesions are borne in mind. The evidence suggests, therefore, that the factors affecting the development and progression of caries in such populations are in principle no different to those to be found in other human populations. This fact is important to establish since we would therefore expect that the effect of various therapeutic measures on caries lesions development and progression would be the same as in all other populations, and that those factors which influence the risk of developing caries are likely to be the same as those found in other populations.

DENTAL CARIES IN ADULT AND ELDERLY POPULATIONS

Much of our knowledge of the epidemiology of dental caries arises from studies of children. A wealth of information about the nature of the disease can, however, be gained from studies of caries in adult populations. Comprehensive data on the occurrence of dental caries in adult populations in most developing countries is rare (Sheiham, 1967, Akpabio, 1970, Gadegaard & Fejerskov, 1983, Baelum & Fejerskov, 1986, Baelum et al., 1988, Chen et al., 1989, Luan et al. 1989, Manji & Fejerskov, 1989). In the following, we shall focus principally on data originating from the baseline of longitudinal studies being conducted in Kenya and China (Baelum et al., 1988a, Manji, Baelum & Fejerskov, 1988a) and from China (Baelum et al., 1988b, Chen et al., 1989, Luan et

al., 1989) with a view to demonstrating the extent to which the pattern in caries is similar in populations as diverse as these.

In these studies, similar diagnostic criteria were used: Namely, those described by Gadegaard & Fejerskov (1983), and subsequently modified by Manji et al. (1989a). In essence, the method involved the classification of surfaces under the following: enamel caries, without suspected dentinal involvement; dentinal caries, without pulpal involvement; pulpal involvement; root surface involvement; filled; or missing.

In reading this section, the following ought to be borne in mind:

a) There are differences in the grouping of the 10-year cohorts used in the studies from Kenya as compared to those from China, and care should be exercised when comparing the data in the figures.

b) The data for the Kenyan studies originate from a population with almost no access to formal dental care (Manji et al., 1988). However, in China an infrastructure of paradental workers provide simple dental care (fillings, extractions and prostheses). Thus irrespective of the differences between the Kenyan and Chinese populations in caries experience, considerably more restored and extracted teeth would be expected in the Chinese populations.

Tooth mortality: Although it has commonly been held that periodontal diseases are the major causes of tooth loss in developing countries (WHO, 1978), evidence from both Africa (Baelum & Fejerskov, 1986) and China (Baelum et al., 1988b, Luan et al., 1989) demonstrates that it is caries which

accounts for most of tooth mortality. This is immediately apparent from a comparison of the data in Figures 1 and 2: in Kenyan rural, and Chinese urban and rural population, in every age group, a greater proportion of individuals had teeth indicated for extraction because of caries than were indicated for extraction because of periodontal disease (marked mobility of the teeth).

Even up to the age of 50-60 years of age, nearly half the populations remains with about one-half of the full complement of teeth in the rural population of Kenya, and in both rural and urban Chinese populations (Fig. 3). The survival of the dentition in Kenya is considerably greater, with more than 50% individuals having more than 26 teeth present. The prevalence of edentulousness was reported as 0.3% in Kenya (Manji et al., 1988). Survival of the dentition in the Chinese population was poorer than in Kenya: among 60 to 69 year-olds, 50% individuals had more than 20 teeth (rural), and 25 teeth (urban), present. Above the age of 70 years more than half of the teeth were lost in 50% of the Chinese population. Twentynine per cent of urban women in the Chinese study were reported edentulous after the age of 60 years (Baelum et al., 1988b). In all these populations, however, tooth-loss was considerably smaller than for populations of similar ages in industrialized countries (Johansen 1970, Bouma, Schaub & van de Poel, 1985).

Mean caries experience: The mean DFT of each age cohort is shown in Figure 4. The first most striking difference between the Kenyan and Chinese populations is in the amount of enamel lesions in the Kenyan population. Whereas the Chinese populations show evidence of a considerable amount of fillings, hardly any Kenyans had any filled teeth. When enamel lesions are ignored, the mean DFT in the Kenyan rural and Chinese urban populations is rather similar for

comparable age groups. For every age cohort, however, the rural Chinese exhibited a greater caries experience that the other populations. The mean number of teeth with pulpal lesions increased with age in all three populations, as did the mean number of teeth with root surface caries. In particular it is apparant how a substantial number of lesions in the two oldest Chinese age groups have penetrated into the pulp. It is noteworthy how similar root surface caries experience is in populations as diverse these.

Distribution of caries within populations: The cumulative frequence distribution of the number of teeth filled or decayed is shown in Figure 5, in which enamel lesions have been excluded, and in Figure 6 where they have been included. The virtual ubiquity of dental caries occurring as a result of including enamel lesions is apparent in particular in the Kenyan population. In each of the three populations it is apparent that much of the caries experience is accounted for by a relatively small proportion of individuals. In the Kenyan population there is almost no difference between the age cohorts in the proportion of individuals with more than 14 teeth affected, whereas there are a greater proportion of individuals with 14 or more teeth affected in the two oldest age cohorts of the Chinese populations. In all three populations the distribution of caries is skewed.

Intra oral distribution

In Figures 7 and 8 the intra oral distributions of caries lesions according to the various components recorded are presented for the different tooth groups and in the different age cohort. If ignoring the enamel lesions, the within mouth distribution is very similar in comparable age groups in Kenya (Fig. 7) and China (Fig. 8). Again it is very interesting in the oldest Chinese age groups to note how the percentage of remaining teeth with pulp involvement

is very high. In both populations the percentage of remaining teeth exhibiting root surface caries increase steadily with age.

Relationship between root-surface caries and gingival recession

One of the principle features of periodontal disseases in both African (Baelum et al., 1986, Baelum, Fejerskov & Manji 1988a) and Chinese populations (Baelum et al. 1988b) is that loss of attachment is usually not accompanied by pocket formation, i.e. gingival "recessions" appear to be a characteristic feature of the disease. In that context, one might expect that root-surface caries would be very common in populations. Figure 9 shows data from China indicating the intra-oral distribution of root-surface lesions according to the surface type affected. In this diagram is shown also the proportion of surfaces that were at risk to developing such lesions (i.e. recession of the marginal gingiva was present). The striking conclusion to be drawn is that there appears to be little relationship between the proportion of surfaces at risk to root-surface caries and the occurrence of such lesions. These data, taken together with the fact that the levels of root-surface caries are strikingly similar to those to be found in North American populations of comparable age (Miller et al. 1987), are surprising epidemiological findings.

From the above brief review of caries in adult populations in Africa and China, a number of important conclusions can be drawn. First, it is apparent that caries continues to progress throughout life, albeit with a slow rate of progression. Teeth which are likely to become affected by caries do so from an early age, but the disease nevertheless progresses, with an increasing severity of the lesions with increasing age. Despite this slow progression rate, caries accounts for the principle cause of tooth-loss. In

populations as diverse as those from Kenya and China, there is a striking similarity in the intra-oral distribution of caries, and in the distribution of root-surface lesions. When enamel lesions are considered in the diagnosis of caries, there is clear evidence of the ubiquity of the disease. The distribution of caries is, however, fairly skewed in all age groups, with the majority of lesions occurring in a minority of individuals. Can such data be interpreted as being evidence of the existence of "high risk groups or individuals"?

VARIATION IN DISEASE EXPERIENCE AS A REFLECTION OF VARIATION IN RISK?

When dealing with the question of risk for developing dental caries, we are usually interested in assessing the extent to which variations in disease experience reflect underlying variations in (often unseen) risk factors. What is frequently overlooked in such investigations is to assess the degree to which variations in response between individual is accounted for by mere random effects.

In the presentation of the (cumulative) frequency distribution of caries experience above (Figs. 5 and 6), it was apparent that, at any given age, there is a considerable between individual variation in caries experience. The fact that some individuals have much caries while others have little might suggest that there may be variations in risk. However, even where groups or individuals may be homogeneous with respect to the risk of developing caries (i.e. for every risk factor they have the same probability of developing caries), there will nevertheless be a considerable variation in caries experience due entirely to random effects. To what extent, therefore, are the variations in caries experience in these studies accountable

for by random effects? Or to put it another way, how much remains to be explained by variations in risk after the contribution of random effects have been accounted for?

Since caries develops as a function of time, and since each tooth-type in the mouth erupts at different times, it may be difficult to assess risk or random effects by considering a composite index such as the whole mouth DMFT. Let us take, as an example, the caries experience of the first permanent molars of the adults from the above study from Kenya. Table 4 summarizes the mean DMF of this tooth-type in each 5-year age cohort. A test for dispersion (Nagelkerke, Manji & Muttunga, 1989) performed on these data indicated that only in a one age group (i.e. in the 55 to 59-year-olds, and even here on the borderland of statistical significance) was the variation greater than would be expected from random variations. In all other cases, there was little evidence to suggest that the observed variations in caries experience were a reflection of differences in risk.

It should be noted that these data originate from a population that was relatively homogeneous with respect to their exposure to risk factors (Ginneken & Muller, 1984, Manji et al., 1988, Manji et al., 1989a), and thus the results are to some extent indicative also of the extent to which variations in intrinsic (host) risk factors contribute to the variations in susceptibility to caries.

CONCLUSIONS

In this paper we have shown that dental caries in populations of developing countries can be considered to be an ubiquitous phenomenon, indicating that no individuals can be considered to be resistant, immune, or non-susceptible. With few exceptions, there is little evidence that caries

has been increasing at a dramatic rate in either Africa or China. The evidence indicates that the factors affecting the development and progression of caries in such populations are in principle no different to those which would be expected to be found in any human population. Although there may be variations in caries experience of individuals within a given community, there remains little evidence to suggest that such variations can be attributed to the existence of "high or low risk groups or individuals" who are having one and the same "risk factor" in common.

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Legends

Figure 1

Cumulative frequency distributions showing the number of teeth per individual which are indicated for extraction in the various age groups in rural population examined in Kenya and in urban and rural populations examined in China. Notice in particular the substantial difference between the rural Chinese population and the 2 other populations.

Figure 2

Cumulative frequency distributions showing the number of teeth per individual indicated for extraction because of substantially increased mobility in various age groups in rural Kenyan populations and in urban and rural Chinese populations. As compared with the previous figure, it is apparent that much less teeth in all age groups are indicated for extraction for periodontal reasons than for caries reasons.

Figure 3

Cumulative frequency distributions showing the number of teeth present per individual in various age groups in Chinese and Kenyan populations. In the 2 older Chinese age groups, there is an apparent loss of teeth, but in the younger age groups, both in Kenya and in China, it is apparent that 50% of the individuals are having about 28 teeth or more present.

Figure 4

Mean decayed and filled teeth according to age groups in Kenyan rural and Chinese urban and rural populations. The apparent substantial difference in enamel caries lesions between the Kenyan and the Chinese populations are a result of different diagnostic methods used! The mean number of teeth with pulpal involvement are substantially higher in the 2 oldest Chinese age groups.

Figure 5

Cumulative frequency distributions illustrating the number of teeth affected by caries (excluded enamel lesions) in Kenyan and Chinese populations. Notice the marked skew distribution showing that only about 10% of the individuals in most of the age groups are responsible for a substantial number of carious teeth.

Figure 6

Cumulative frequency distributions of the number of carious teeth (including enamel lesions) in the Kenyan and Chinese populations. Notice in particular in the Kenyan population dental caries is ubiquitous even from young age.

Figure 7

The intra-oral distribution of dental caries according to the tooth types in the various age group in a rural Kenyan population.

Figure 8

The intra-oral distribution of dental caries according to various tooth types and age groups in urban and rural Chinese populations presented together. If the slight difference in age groups between the Kenyan and Chinese study populations are taken into account, it is apparent when comparing Figures 7 and 8 that up to about the age of 60 years the within mouth distribution appears rather similar in these diverse populations.

Figure 9

The within mouth distribution of root surface caries in Chinese adult populations presented according to age groups. The open bars indicate the percentage of surfaces according to the different tooth types exhibiting gingival recession (surfaces at risk) and the black bars indicate the percentage of surfaces exhibiting root surface caries lesions. It is apparent that with increasing age there is a dramatic increase in surfaces at risk, but very limited root surface caries experience.

TABLE 1

Caries experience (mean DMFT, unless otherwise stated) in the permanent dentition of young persons in Africa

Country and Study	Age group (yr)	Mean DMF	Remarks
Nigeria			
Sheiham (1967)	10-14	0.01-0.13	West and East
Akpabio (1970)	10-14	0.85	
Enwonwu (1974)	10-14	0.08-1.14	
Henshaw and Adenubi (1975)	10-19	3.1	Northern region
Akpata (1979)	15	1.58	Lagos
Akpabio et al. (1982)	12	2.0	National
Arain (1983)	15-17	2.2	Lagos
Ojofeitimi et al. (1984)	8-15	0.27-0.57	School-type: non-fee-paying
		1.23-1.13	fee-paying
Arain and Arole (1985)	15-17	2.0-3.5	
Olujigba and Lennon (1987)	12	0.13 in 1977 2.15 in 1983	
Ghana			
Brown (1945)	6-20	0.38	
Houpt et al. (1967)	12	1.25	
Houpt and Botchway (1969)	5-13	1.03	
Nornoo (1986)	12	1.83	
Guinea-Bissau			
Matthesen et al. (1989)	12	2.4 (incl. enamel lesions) 0.5 (excl. enamel lesions)	
Zaire			
Barmes (1970)	12	1.0	
Marseilles (1984)	12	0.3	

Ethiopia			
Littleton (1963)	10-14	0.18	
Olsson (1978)	13-14	1.54	0.2-0.3 ppm F
Olsson (1979a)	13-14	1.69	3.5 ppm F
	13-14	2.46	12.4 ppm F
Olsson (1979b)	13-14	3.34	0.2-0.7 ppm F (private schools)
Sudan			
Emslie (1966)	10-14	0.7	
Ibrahim et al. (1985)	12-14	1.7	
Ibrahim et al. (1986)	12	2.9 (urban)	
		3.2 (rural)	
Carlsson et al. (1987)	12	0.17 (rural)	
Uganda			
Akpabio (1970)	10-14	1.2	
Moller et al. (1972)	10-14	0.1-0.4	National
Jensen et al. (1973)	12	2.1-2.5	Kampala
Tirwomwe et al. (1988)	12		National
		1.88 incl. enamel lesions	
		0.45 excl. enamel lesions	
Tanzania			
Gadegaard and Fejerskov (1983)	10-14	1.7-2.5 (DMFS)	National
Mosha and Langebæk (1983)	12-14	0.8-0.9	Arusha/Moshi
Frencken et al. (1986)	12	0.67	Dar es Salaam
Mosha (1986)	14	5.3 (DMFS) in 1979	Dar es
		3.1 (DMFS) in 1983	Salaam
Manji et al. (1989c)	12	0.51	Rural
Kerusuo et al. (1986)	12	0.9	Dar es Salaam
Mosha et al. (1988)	11-13	0.64 in 1973	
		0.39-1.06 in 1984	Arusha
Frencken (1988)	11	0.3-0.7	Morogoro

Kenya			
Akpabio (1970)	10-14	1.47-2.22	
Gad (1973)	12	1.7	National
Bakshi (1977)	9-12	1.0	Nairobi
Manji (1983a)	12	0.3	Nairobi
Manji (1983b)	12	0.6-1.1	Nairobi
Manji (1984)	12	0.2	Nairobi
Frencken et al. (1986)	12	0.51	Nairobi
Manji et al. (1989c)	12	0.21	Rural
Manji (1988)	12	0.6	Nairobi
	12	0.2	Rural
Burundi			
Vecchiati (1988)	12	0.91-1.22	
Swaziland			
Klausen and Fanøe (1983)	14	9.17	(DMFS)
Bindslev et al. (1988) (unpublished)	14	5.15	(DMFS incl. enamel lesions)
Mozambique			
Hobdell and Cabral (1980)	12	0.8	
Hobdell (1981)	12	0.8	
Zimbabwe			
Ritchie (1975)	13	0.48	
Chironga and Manji (1989)	12	0.57	(urban)
		0.49	(rural)
Madagascar			
Petersen and Steengaard (1986)	12	2.4	

TABLE 2

Comparison of data from two national surveys conducted in Uganda. The data on caries shown from Tirwomwe et al. (1988) exclude data for enamel lesions in order to allow for comparisons between the two data sets. The names of the areas shown in parentheses are the names formerly used by Moller et al. (1972).

	Moller et al. (1972)			Tirwomwe et al. (1988)		
	deft	DMFT	DMFT	dmft	DMFT	DMFT
Area	5-9yr	10-14yr	30-39yr	6yr	12yr	35-44yr
Kabarole (Toro)	0.1	0.1	0.2	0.9	0.3	1.9
Mbale (Bugisu)	0.8	0.1	0.2	1.2	0.3	1.2
Kabale (Kigezi)	1.3	0.3	1.1	1.9	0.3	1.9

TABLE 3

The effect on apparent prevalence and on the mean DMF scores of excluding enamel lesions from caries diagnosis. In each of these studies the same diagnostic methods were used, namely those described by Gadegaard and Fejerskov (1983), and subsequently modified by Manji et al. (1989).

		Incl. enamel lesions		Excl. enamel lesions	
Study	Age yr	% Prevalence	Mean DMFT	% Prevalence	Mean DMFT
(1)	6 *	72.9	3.61	44.0	1.55
	9 *	76.3	3.09	46.9	1.25
	12	76.1	2.97	26.5	0.55
	15	81.1	4.46	26.7	0.67
(2)	15-24	93.0	6.83	48.8	1.27
	25-34	97.2	10.65	82.4	3.98
	35-44	98.2	11.89	76.4	5.78
	45-54	97.2	10.97	82.8	5.93
	55-65	97.3	13.21	92.9	9.19
(3)	6 *	51.3	1.52	35.0	0.77
	12	60.4	1.88	23.1	0.45
	35-44	68.1	3.93	52.4	2.04
	≥ 55	82.7	4.95	71.4	3.68

(1): Manji (1988): data for city-dwellers * dmft scores

(2): Manji et al (1989)

(3): Tirwomwe et al. (1988)

Table 4

Mean DMF scores of first permanent molars of Kenyans aged 15-65 years and the X^2 (with n degrees of freedom) for over-dispersion of the distribution. A significant test statistic is indicative of the probability that the observed distribution was more dispersed than that which would have been expected from random effects alone.

Age group	Mean DMF	s.d.	n	X^2	p
15-19 yr	0.34	0.69	130	57.41	>0.98
20-24 yr	0.66	0.93	126	89.59	>0.75
25-29 yr	0.90	1.06	105	90.60	>0.50
30-34 yr	1.17	1.15	111	104.65	>0.25
35-39 yr	0.99	1.10	115	106.82	>0.25
40-44 yr	1.31	1.22	105	105.22	>0.25
45-49 yr	1.08	1.16	109	106.18	>0.25
50-54 yr	1.15	1.26	106	112.73	>0.10
55-59 yr	1.47	1.32	109	125.09	>0.05
60-65 yr	1.81	1.36	115	123.23	>0.05

Data originate from Manji et al.(1989 a)

Fig. 1

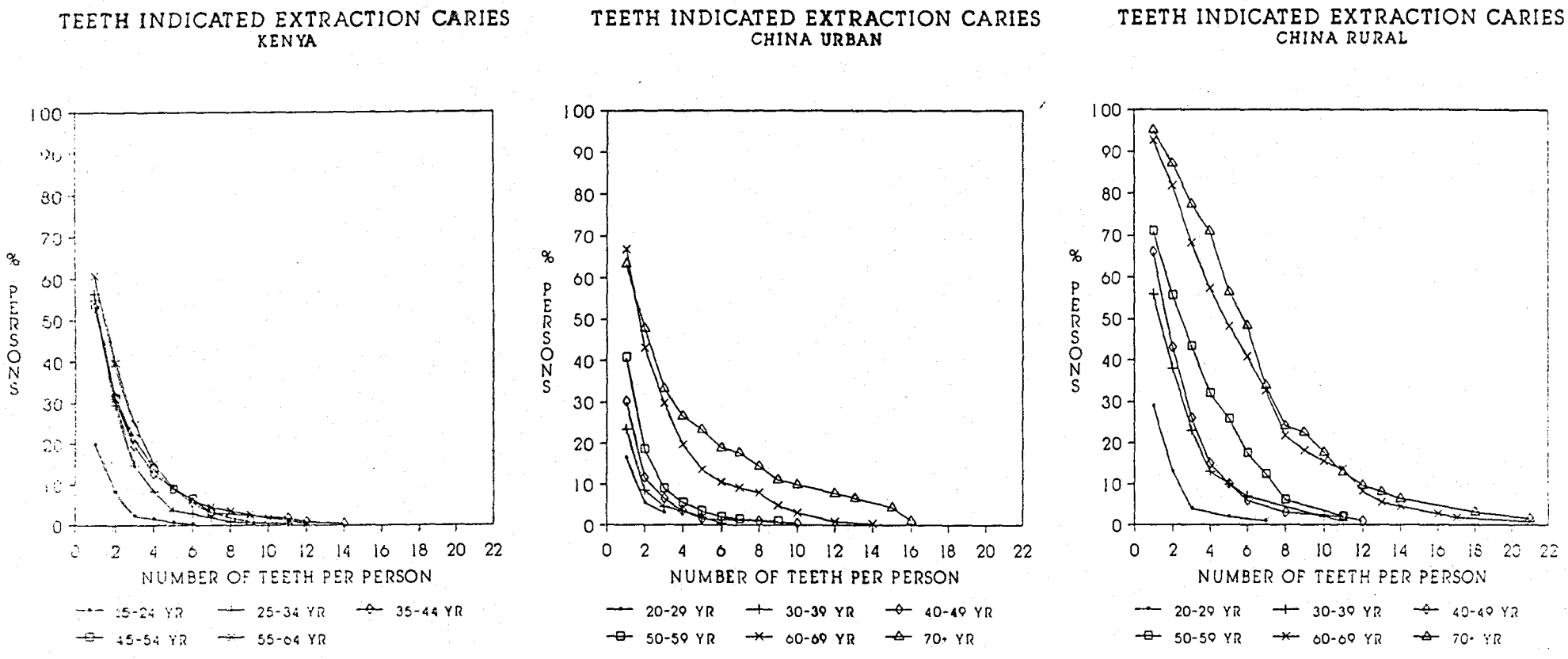
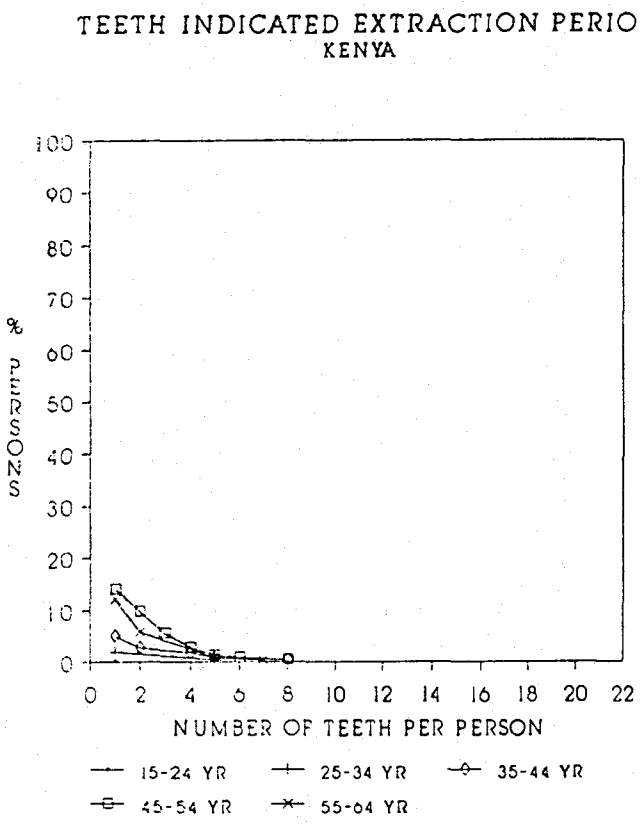
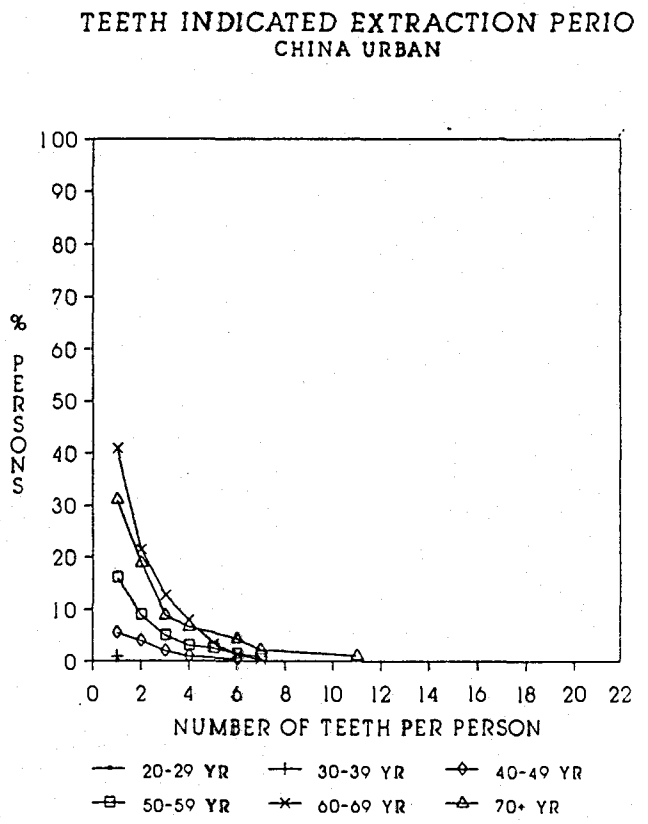

TEETH INDICATED EXTRACTION CARIES
KENYA
% PERSONS
NUMBER OF TEETH PER PERSON
15-24 YR
25-34 YR
35-44 YR
45-54 YR
55-64 YR
TEETH INDICATED EXTRACTION CARIES
CHINA URBAN
% PERSONS
NUMBER OF TEETH PER PERSON
20-29 YR
30-39 YR
40-49 YR
50-59 YR
60-69 YR
70+ YR
TEETH INDICATED EXTRACTION CARIES
CHINA RURAL
% PERSONS
NUMBER OF TEETH PER PERSON
20-29 YR
30-39 YR
40-49 YR
50-59 YR
60-69 YR
70+ YR

Fig. 2


TEETH INDICATED EXTRACTION PERIO
KENYA
% PERSONS
NUMBER OF TEETH PER PERSON
15-24 YR
25-34 YR
35-44 YR
45-54 YR
55-64 YR


TEETH INDICATED EXTRACTION PERIO
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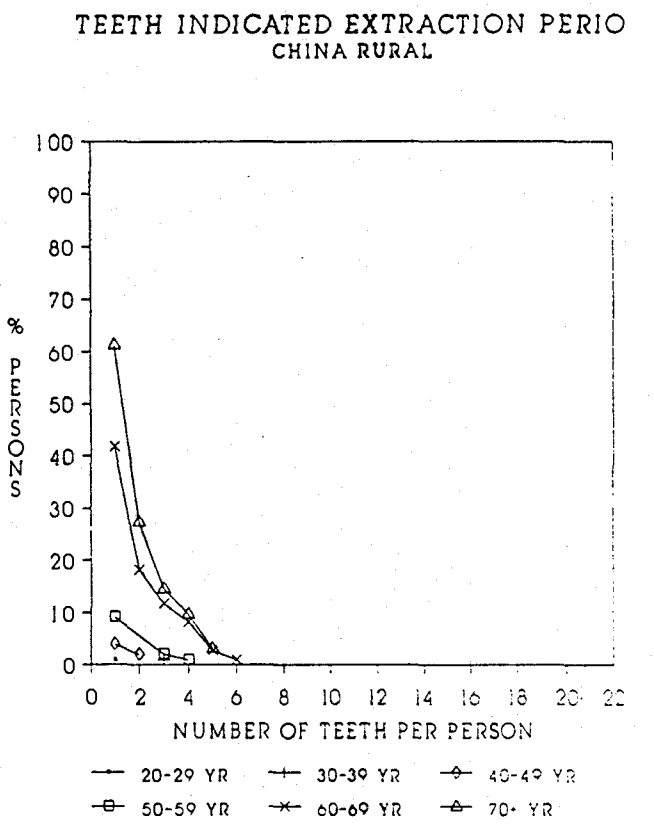
60-69 YR
70+ YR


TEETH INDICATED EXTRACTION PERIO
CHINA RURAL
% PERSONS
NUMBER OF TEETH PER PERSON
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30-39 YR
40-49 YR
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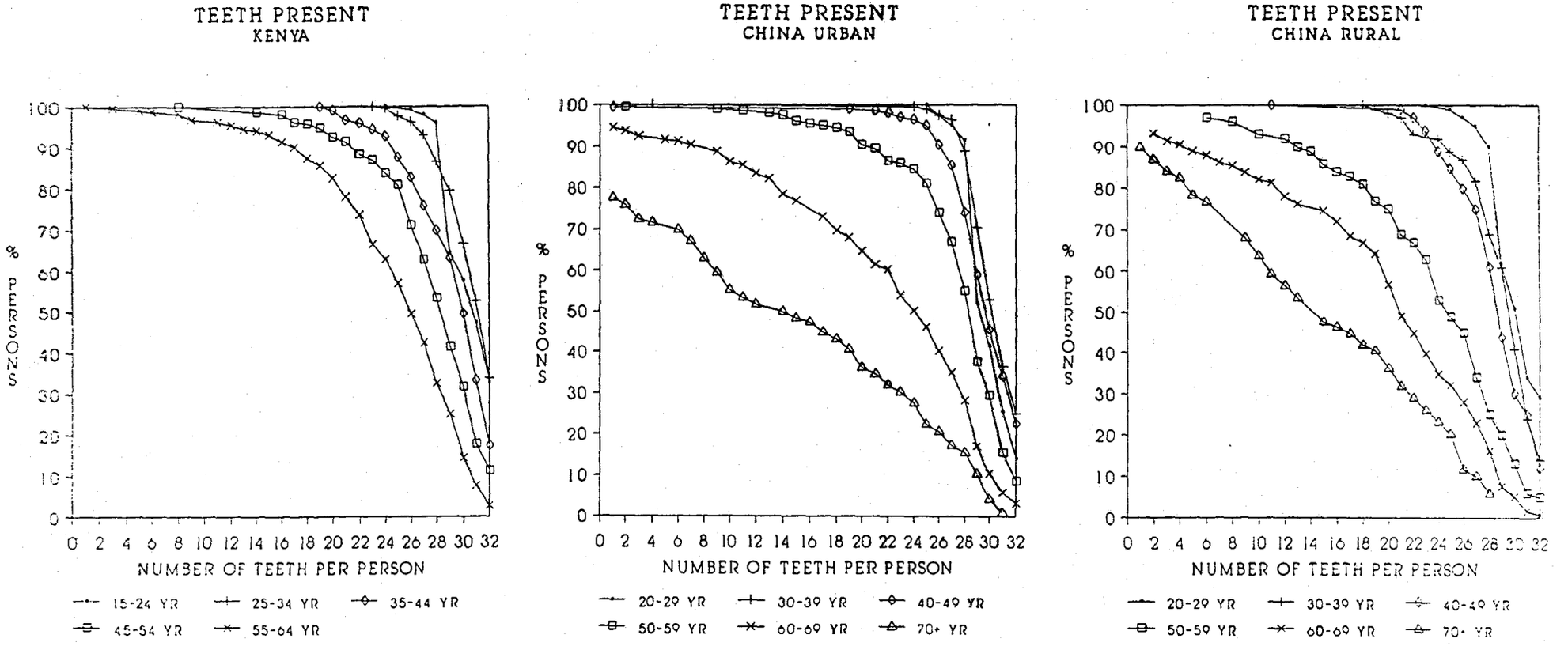
60-69 YR
70+ YR

Fig. 3


TEETH PRESENT
KENYA
% PERSONS
NUMBER OF TEETH PER PERSON
15-24 YR
25-34 YR
35-44 YR
45-54 YR
55-64 YR
TEETH PRESENT
CHINA URBAN
% PERSONS
NUMBER OF TEETH PER PERSON
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30-39 YR
40-49 YR
50-59 YR
60-69 YR
70+ YR
TEETH PRESENT
CHINA RURAL
% PERSONS
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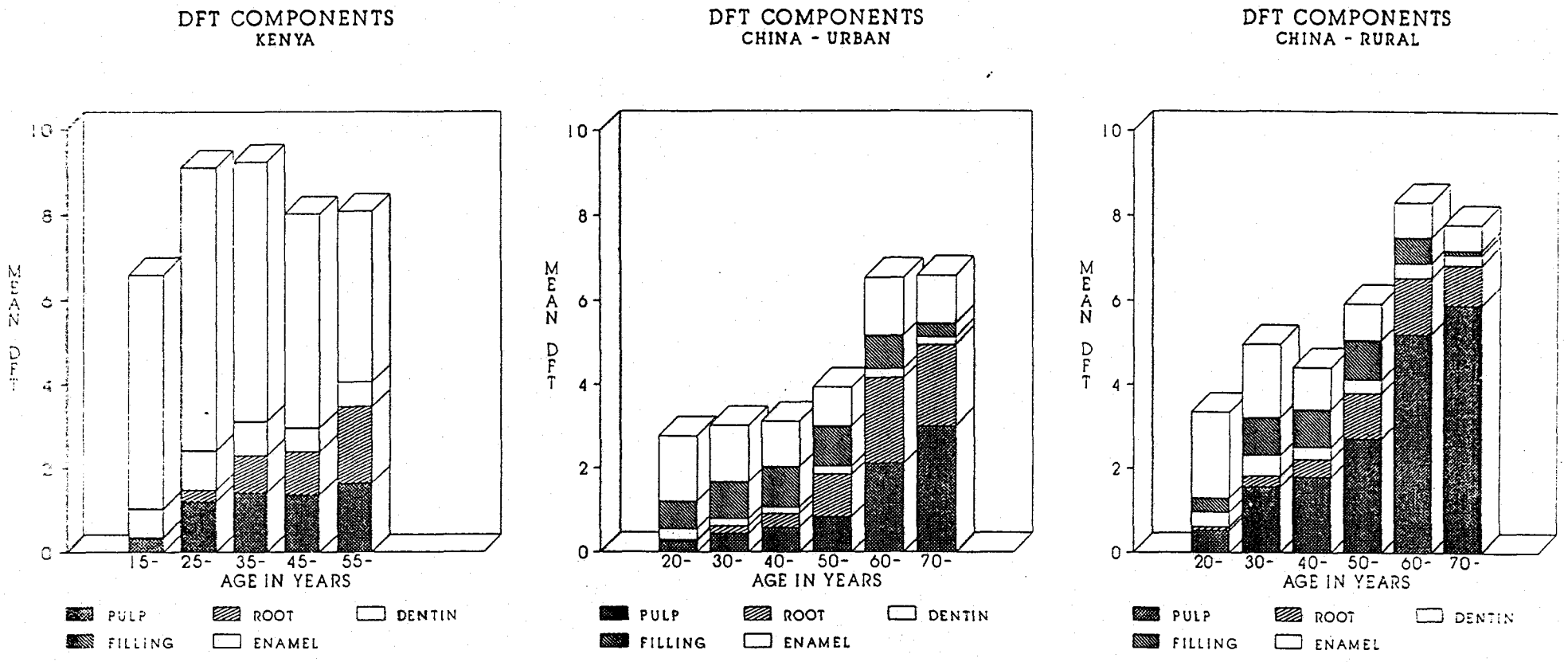
70+ YR

Fig. 4


DFT COMPONENTS
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AGE IN YEARS
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DFT COMPONENTS
CHINA - URBAN
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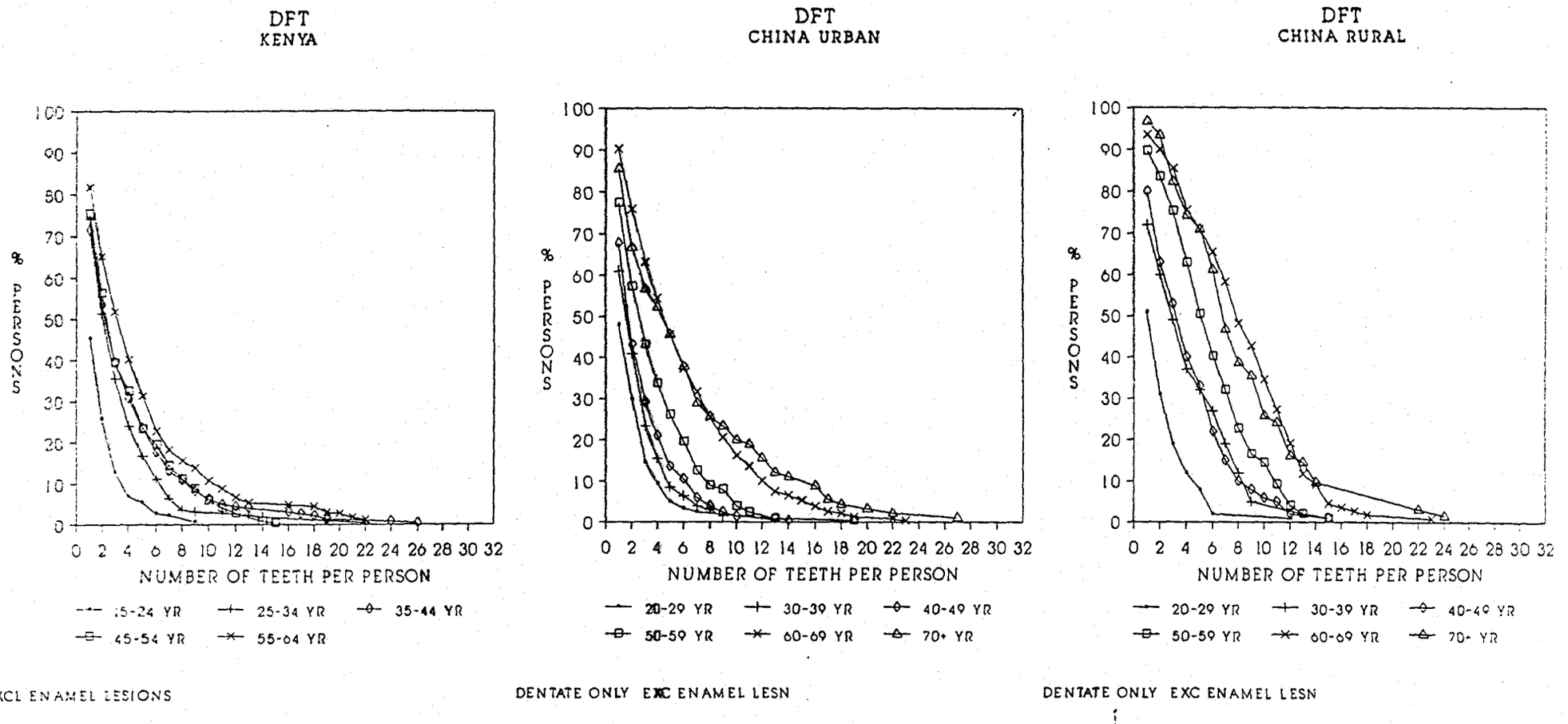
Fig. 5


DFT
KENYA
% PERSONS
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45-54 YR
55-64 YR
EXCL ENAMEL LESIONS
DFT
CHINA URBAN
% PERSONS
NUMBER OF TEETH PER PERSON
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50-59 YR
60-69 YR
70+ YR
DENTATE ONLY EXC ENAMEL LESN
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CHINA RURAL
% PERSONS
NUMBER OF TEETH PER PERSON
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60-69 YR
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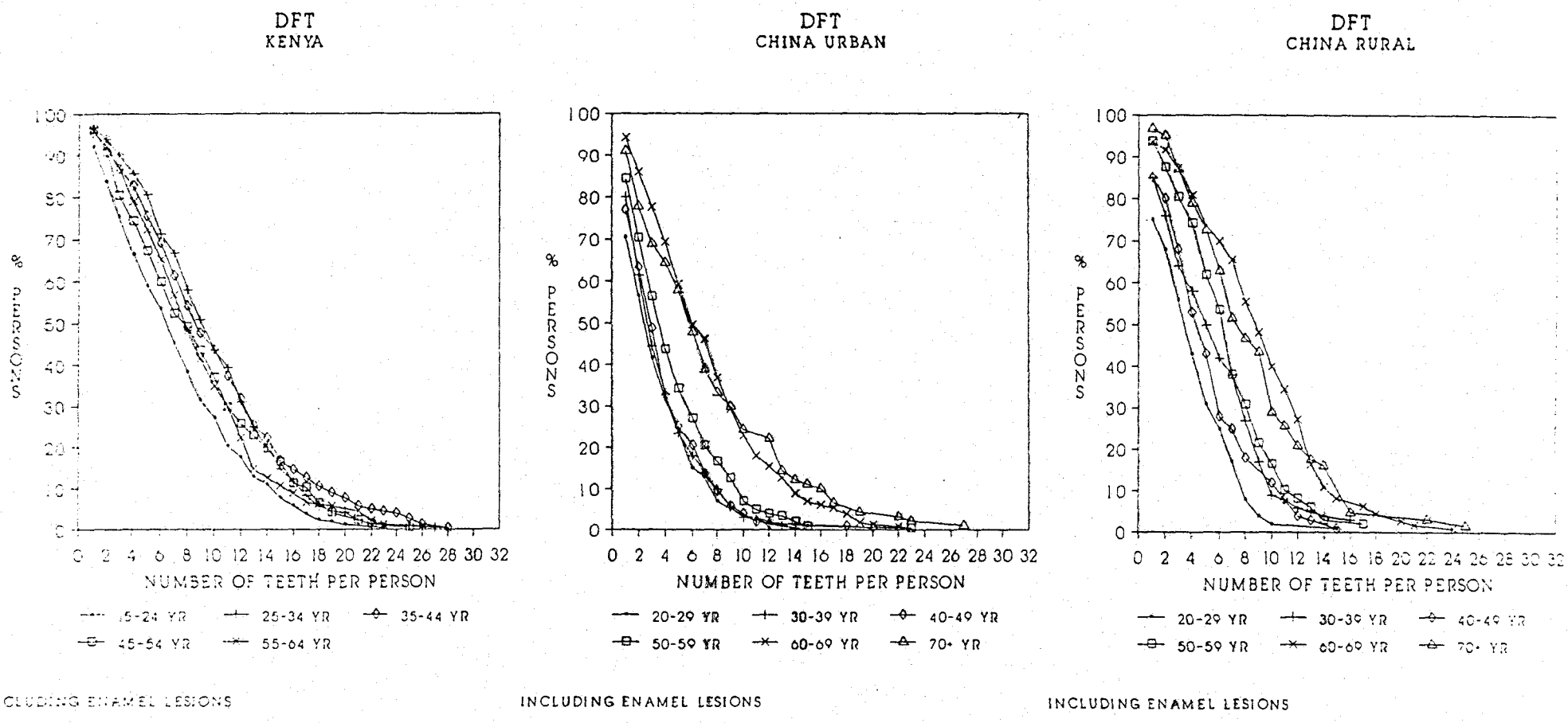
DENTATE ONLY EXC ENAMEL LESN

Fig. 6


DFT
KENYA
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NUMBER OF TEETH PER PERSON
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INCLUDING ENAMEL LESIONS
DFT
CHINA URBAN
% PERSONS
NUMBER OF TEETH PER PERSON
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50-59 YR
60-69 YR
70+ YR
INCLUDING ENAMEL LESIONS
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CHINA RURAL
% PERSONS
NUMBER OF TEETH PER PERSON
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INCLUDING ENAMEL LESIONS