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Inter-individual variation in the plaque formation rate of young individuals

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Abstract: *Objectives* The aim of the present study was to describe the inter-individual variation in the plaque formation rate of 38 young adults. *Methods* The plaque formation rate was estimated by employing a quantitative plaque percent index (P% index). A substantial inter-individual variation in the plaque formation rate was observed. The possible contributions of stimulated salivary flow rate, buffer capacity, diet and smoking to the variation in plaque formation were estimated by regression analysis. *Results* The control variables explained only 2.5% of the variance in the plaque formation rate. Neither associations nor the total model were significant. The present method of measuring plaque presented as a simple and time-effective procedure. *Conclusions* It is suggested that the observed variation in the plague formation rate between the young individuals can be regarded as a biological function which is possibly an inherent individual characteristic. Studies with larger sample sizes are required to confirm the findings of the present study.

Key words: adults; computer-assisted plaque estimation; diet; plaque formation rate

Introduction

Dental plaque is a known precursor to oral diseases such as gingivitis, periodontitis and caries (1, 2). Dental plaque or biofilm can be defined as the diverse community of micro-organisms found on the tooth surface (3). The plaque formation rate can be defined as the quantity of plaque that forms on clean tooth surfaces during a given amount of time (4). The amount of plaque that can be recorded in, for example, 24 h is an expression of an individual's capacity to form plaque. Plaque formation capacity can be of particular interest to epidemiologists as the

rate can be a marker for a biological function (5). If the plaque formation rate can be measured in a way that is appropriate for epidemiological usage, this biological function has the potential to explain variation among individuals.

The plaque formation rate index (PFRI) measurement (4) is based on an estimate of the freely accumulated plaque on buccal, lingual and approximal surfaces. The PFRI is a total quantitative score indicating the percentage of surfaces with plaque. On each surface the recording is a dichotomy: plaque present or plaque not present. The dichotomy of the recording may not be sufficiently precise to portray the genuine variability between individuals with regard to their plague formation capacity.

In clinical studies, dental plaque has been measured by ordinal and interval-scaled indices (6–11) while epidemiological studies mainly recorded plaque levels using an ordinal scale (12–17). Different methods of measuring plaque showed varying degrees of discriminating power of assessing individual oral hygiene (18). High intra-examiner reproducibility was reported when a few ordinal and interval scale indices were compared (19).

In the search for a quantitative way to measure plaque in epidemiological studies, one should aim for a practical and time-effective method. One method of digital imaging of plaque formation on tooth surfaces showed a high degree of reproducibility. It was also useful in detecting minor changes in plaque (2, 20).

The aim of the present study was to measure and describe the inter-individual variation in the plaque formation rate by means of digital recordings.

Study population and methodology

The study was approved by the ethics committee (Regional komité for medisinsk forskningsetikk – Helseregion Sør Ref.: S-01166) and financed by The University of Oslo. Permission from the school authority was obtained and forty 18 to 22-year-old professional (non-dental) female college students who were in good general health were invited to participate in the study. Prior to the study, all students received information regarding the study protocol. Informed consent was obtained from 38 individuals while two students were not interested in participating in the study.

Plaque levels were estimated employing a quantitative plaque percent index (P% index) whereby the area on a tooth that is covered with plaque is expressed as a percentage of the total tooth area (20). Measurements were done in the following way. Using a digital picture, the number of pixels was calculated for both the area of tooth covered with plaque and for the total tooth area. The individual total P% index was obtained by summing the indices of each tooth and then dividing this by the number of teeth present in the digital picture.

By means of the P% index, two clinical recordings were taken for each participant: baseline plaque, and plaque formation rate (the amount of plaque which accumulated during 24 h on cleaned teeth). Both plaque recordings were taken and evaluated by the same examiner (JA). At the baseline examination, instructions were given not to perform any tooth-brushing during the next 24 h.

A few factors (diet, smoking, stimulated salivary flow rate and salivary buffer capacity) were considered to be important in plaque formation. Information about a 24-h diet recall and smoking behaviour was collected by a questionnaire during the first examination and after the examination on the second day. Their possible contribution to the observed variation in plaque formation was estimated by regression analysis.

The 24-h diet report consisted of information about all food and drink consumed during the time between the two examinations. The smoking behaviour was estimated from self-reports (non-smokers, ex-smokers and current smokers).

Before the measurement of salivary flow rate was taken, saliva was pre-stimulated by means of chewing paraffin for 1 min without collecting saliva. Then, saliva was collected for 5 min. The salivary flow rate was defined as the amount of stimulated saliva produced per minute.

In the present study, a laboratory estimate of buffer capacity was obtained by titrating 1 ml of saliva with 0.1 M HCl solution until saliva reached a pH of 4.5. The laboratory estimate of buffer capacity was expressed as the volume of 0.1 M HCl required titrating 1 ml of saliva to a pH of 4.5. Higher amounts of HCl used for titration correspond to higher values of buffer capacity.

Digital estimations of plaque levels

Prior to taking each digital photograph, dental plaque was stained with plaque-staining tablets which stain 0 to 3-day-old plaque (RONDELLS RED; SDI Svenska Dental Instrument AB, Upplands–Vasby, Sweden). Then, participants were asked to thoroughly rinse their mouth. The digital images of stained plaque were taken twice, first for the baseline plaque recording and a second time for recording the plaque formation rate. At both clinical sessions, three images of buccal tooth surfaces were taken including premolars and front teeth: one of the labial area, one of the left buccal area and one of the right buccal area. Digital pictures included areas from right to left

premolars. The digital pictures of individuals who were lacking one or more premolars were taken to include first molars. The teeth were photographed with a Nikon camera using an artificial light from a dental unit. The camera was held perpendicular to the teeth being photographed. At the time of each clinical examination, the individual image was inspected and if necessary immediately retaken. After the image of baseline plaque levels was taken, all teeth were professionally cleaned. After a 24 h period, digital photographs were re-taken in order to obtain an estimate of the plaque formation rate.

For each participant, all photographs were digitized and images stored in an optical storage media. Subsequently images were analysed with the Adobe Photoshop program (Adobe Systems Ltd, Europe, Uxbridge, UK). The sequence of image evaluation is presented in Fig. 1. The total labial tooth surface area was measured manually, using the drawing facility to outline the tooth boundaries on each image. To measure the plaque area, the 'pen tool' and 'make path' were used. The steps in image estimation were as follows (Fig. 1). First, gingival edges were marked, then gingival areas were removed and the remainder of the image pasted into a new window, where the precision of teeth areas was adjusted if needed and then saved as a new TIFF file, retaining the original unaltered image. The 'auto-detect colour objects' option was used to automatically detect the tooth area, providing a measurement of the total area in pixels. Then all areas stained in red were marked and the number of pixels was recorded from the histogram option. The amount of plaque on the tooth was determined by summation of the number of plaque pixels. The number of plaque pixels was divided by the number of pixels corresponding to the total area examined and multiplied by 100. In this way, a 0% plaque score indicated an individual without plaque while a plaque score of 100% indicated an individual who had all buccal surfaces covered with plaque.

The intra-examiner reproducibility of the measurements was estimated by analysis of variance and by comparing 10 randomly chosen digital pictures. The coefficient for the intra-examiner agreement of 0.96 was considered to be satisfactory.

The SPSS 11.0 program (SPSS Inc., Chicago, IL, USA) was used for a subsequent statistical analysis. A linear multiple regression analysis was done in order to estimate the variation in the plaque formation rate while controlling for diet, smoking behaviour and two salivary factors: stimulated salivary flow rate and buffer capacity. The level of statistical significance was set at $P = 0.05$.

Results

In both the baseline plaque and plaque formation rate measurements, the presence of disclosable plaque was calculated as a percentage of the plaque covered area out of the total labial surface area that was examined. A substantial, statistically significant difference was observed between both plaque recordings (Table 1). Baseline plaque showed a slightly higher inter-individual variation than did plaque forming rate. Figure 2 illustrates inter-individual variation in the plaque

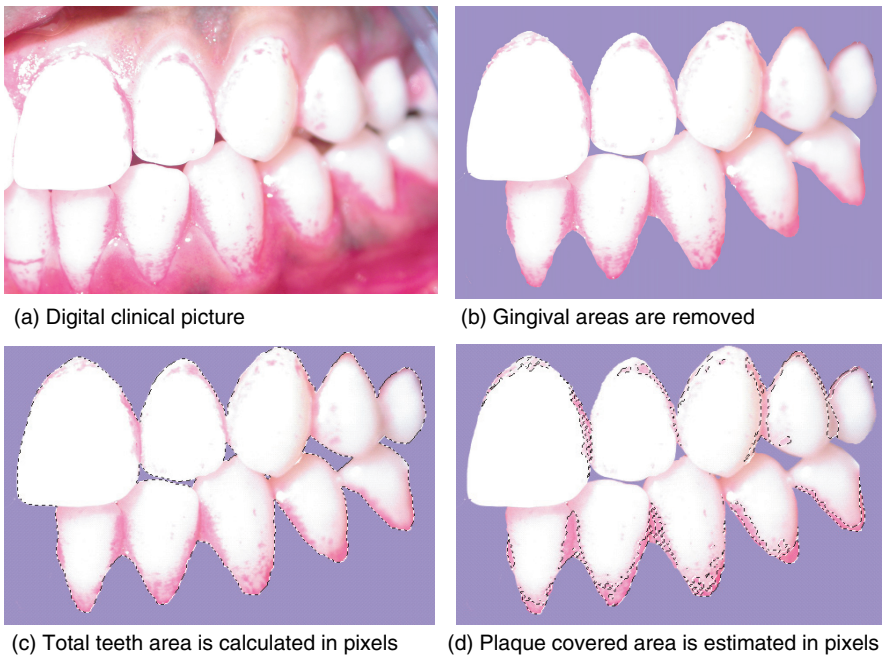

Fig. 1. Steps in digital estimation of plaque scores.

Table 1. **Variations in plaque levels and diet in young healthy college students (*N* = 38)**

	Mean ± SD	Min.	Max.
Baseline plaque levels (%)	14.8 ± 7.7	2.6	36.3
Plaque formation rate (%)	11.6 ± 3.8	3.9	20.7
Frequency of sugar-containing item consumption between the two examinations (24-h diet record)	1.9 ± 1.3	0	5

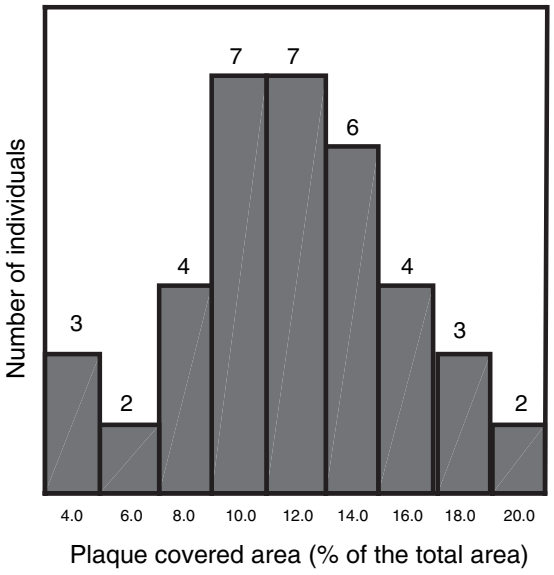

Fig. 2. Distribution of individuals according to their plaque formation rate.

formation rate. Individuals were normally distributed with regard to the plaque formation rate. The differences between smokers and non-smokers with respect to the plaque formation rate were not statistically significant.

The inter-individual variation with regard to diet is presented in Table 1. The evaluation of the 24-h questionnaire revealed that 12.9% did not consume any sugar-containing products while 62.3% consumed two or more sugar-containing items during the time between the two clinical examinations. With regard to self-reported smoking behaviour, 57.9% of individuals never smoked, 7% stopped smoking and there were 35.1% current smokers.

The effects of 'frequency of sugar-containing item consumption between the two examinations', 'smoking', 'stimulated salivary flow rate' and 'salivary buffer capacity' were examined by introducing them into the linear multiple regression model. Collinearity diagnostics of the regression model did not reveal any highly correlated factors (Table 2). Neither associations nor the regression model were significant. For the regression

Table 2. **Linear multiple regression analysis of the variation in the plaque formation rate, controlling for diet variables, smoking and saliva variables (*N* = 38)**

	Beta coefficient	*P*-value	Tolerance values
24-h diet record	0.471	0.069	0.808
Smoking	0.079	0.744	0.801
Stimulated salivary flow rate	−0.212	0.422	0.709
Salivary buffer capacity	−0.024	0.412	0.806

$R^2 = 0.025$; $P = 0.09$.

model, $R^2 = 0.025$, which means that diet, smoking, stimulated salivary flow and buffer capacity accounted for only 2.5% of the variation in the plaque formation rate.

Discussion

The present study showed that inter-individual variation in the dental plaque formation rate varied from 4% to 21%. A wider range of inter-individual variation (7–36%) was reported in the study by Soder et al. (21). The range of baseline plaque levels reported in the present study (14.8 ± 7.7%) was also narrower than the range (19.2 ± 9.4%) previously reported (22). Age and gender may be responsible for the wider range of variation reported by Soder et al. (21, 22) than in the present study: previous studies reported the variation in plaque among middle-aged males and females, while the present study included only young women.

Concomitantly, the present study and both aforementioned studies reported variations in plaque levels in adult samples. Perhaps the plaque formation rate as a biological function has an independent effect on caries onset and progression. Consequently, plaque formation and other important biological functions such as buffer capacity, sugar clearance and salivary flow may mediate the effect of more distant factors such as oral health behaviour, which may be specific to persons. Person-specific variables may affect caries independently of the other causal factors. Given constant distant variables; the impact of person-specific biological functions may be that caries occurrence varies between individuals because their biological functions vary.

There is an important difference between the method of recording plaque in the present study and that of Axelsson (4). This difference can be illustrated with an example. Figure 3 shows pictures of four different patterns of plaque distribution. Three methods of plaque measurement can be compared: (i) a dichotomous measurement where a surface is recorded as being with or without plaque; (ii) the presence or absence of plaque on six sites (distobuccal, buccal, mesiobuccal, mesiolingual,

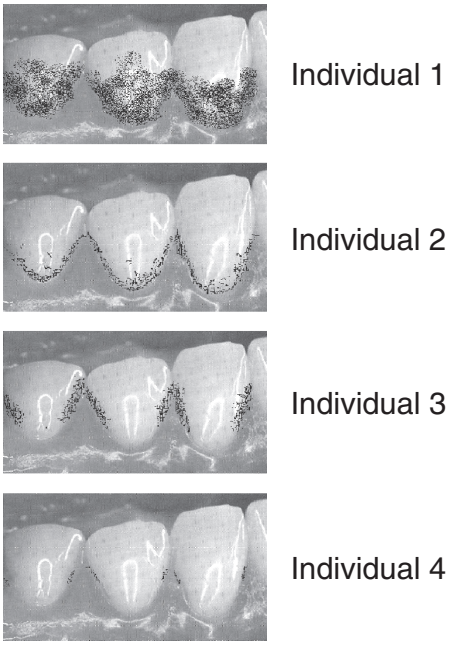

Fig. 3. A simulated example of individual variation in plaque scores.

lingual and distolingual); and (iii) the present method, where the area covered with plaque is measured. By means of the first method, all four pictures will be recorded as positive. By the second method, all six sites have plaque in pictures 1 and 2 while in pictures 3 and 4 there are fewer sites with plaque. By methods 1 and 2, the number of surfaces and sites with plaque are summed into a total plaque score. The measurements are recorded on an ordinal scale and subsequently summed into a measurement of an interval scale. However, using the third method, the quantification of the relative area covered with plaque is on a genuine interval scale: it provides four different scores and thus ensures that no inter-individual variation is lost. Method 3 appears to have an advantage over the other two methods as it seems to be the most precise measurement of the inter-individual variation. It is important to emphasize that any categorization or imprecision in measurement leads to information loss and possibly to increased measurement error. Moreover, categorization establishes arbitrary cut-off points for different degrees of the condition and that is always based on subjective decisions. In fact, disease or its risk factor measurements are nearly always quantitative rather than categorical or qualitative phenomena (23).

There are clear advantages to the method used in the present study: increase in precision, maintenance of complete information, avoidance of categorization, and reduction of measurement error. In addition, the method is simple to administer and saves patient time as only a small amount of time is required to take a few digital images. Images can be stored for later analyses, and the data can be retested for reliability and included in comparative studies (24).

Another important finding of the present study is that diet, smoking or salivary preventive factors, stimulated salivary flow rate and buffer capacity were not significantly related to the variation in the plaque formation rate. As well, some of the observed variation in the plaque formation rate may be due to factors that have not been accounted for.

A clear limitation of the present study is the small sample size. A guideline of more than 10 observations per included variable in multiple regression models has been suggested (25). Given that the regression model of the present study included only a limited number of observations, the findings should be interpreted with caution. In order to make more generalized conclusions, the findings of the present study should be repeated in studies involving larger sample sizes as well as wider age ranges.

Another limitation of the current study is that the present method was tested only for the estimation of the plaque formation rate on buccal surfaces, which are less prone to caries than occlusal and approximal surfaces. However, the digital estimation of occlusal and approximal surfaces is not feasible and thus plaque levels had to be estimated from other less relevant surfaces.

Conclusion

Healthy individuals differ in dental plaque formation rates and this variation between individuals seems to be irrespective of variations in preventive salivary factors, diet or smoking behaviour. Perhaps, the observed variation between the young individuals in the plaque formation capacity can be regarded as a biological function, which is possibly an inherent individual characteristic.

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References

1 Mascarenhas AK. Oral hygiene as a risk indicator of enamel and dentin caries. *Community Dent Oral Epidemiol* 1998; **26:** 331–339.
2 Sagel PA, Lapujade PG, Miller JM, Sunberg RJ. Objective quantification of plaque using digital image analysis. *Monogr Oral Sci* 2000; **17:** 130–143.

3 Allison D, Gilbert P, Lappin-Scott HM, Wilson M. *Community Structure and Co-operation in Biofilms. Society for General Microbiology Symposium 59*. Cambridge, Cambridge University Press, 2000, 1–368.
4 Axelsson P. A four-point scale for selection of caries patients, based on salivary *S. mutans* levels and plaque formation rate index. In: Johnson NW, ed. *Risk Markers for Oral Diseases. Caries*. Cambridge, Cambridge University Press, 1991: 159–171.
5 Hulka S, Margolin BH. Methodological issues in epidemiological studies using biologic markers. *Am J Epidemiol* 1992; **135:** 200–209.
6 Benson BJ, Henyon G, Grossman E, Mankodi S, Sharma NC. Development and verification of the proximal/marginal plaque index. *J Clin Dent* 1993; **4:** 14–20.
7 Charles CH, Sharma NC, Galustians HJ, Qaqish J, McGuire JA, Vincent JW. Comparative efficacy of an antiseptic mouthrinse and an antiplaque/antigingivitis dentifrice. A six-month clinical trial. *J Am Dent Assoc* 2001; **132:** 670–675.
8 Cronin MJ, Dembling WZ, Low MA, Jacobs DM, Weber DA. A comparative clinical investigation of a novel toothbrush designed to enhance plaque removal efficacy. *Am J Dent* 2000; **13(Spec No):** 21A–26A.
9 Marks RG, Magnusson I, Taylor M, Clouser B, Maruniak J, Clark WB. Evaluation of reliability and reproducibility of dental indices. *J Clin Periodontol* 1993; **20:** 54–58.
10 Nathoo S, Rustogi KN, Petrone ME et al. Comparative efficacy of the Colgate Actibrush battery-powered toothbrush vs. Oral-B Cross-Action toothbrush on established plaque and gingivitis: a 6-week clinical study. *Compend Contin Educ Dent Suppl* 2000; **31:** S19–S24.
11 Xu T, Barnes VM. The development of a new dental probe and a new plaque index. *J Clin Dent* 2003; **14:** 93–97.
12 Budtz-Jorgensen E, Mojon P, Rentsch A, Roehrich N, von der Muehll D, Baehni P. Caries prevalence and associated predisposing conditions in recently hospitalized elderly persons. *Acta Odontol Scand* 1996; **54:** 251–256.
13 Calsina G, Ramon JM, Echeverria JJ. Effects of smoking on periodontal tissues. *J Clin Periodontol* 2002; **29:** 771–776.
14 Demers M, Brodeur JM, Mouton C, Simard PL, Trahan L, Veilleux G. A multivariate model to predict caries increment in Montreal children aged 5 years. *Community Dent Health* 1992; **9:** 273–281.
15 Martens L, Vanobbergen J, Leroy R, Lesaffre E, Declerck D. Variables associated with oral hygiene levels in 7-year-olds in Belgium. *Community Dent Health* 2004; **21:** 4–10.
16 Mojon P, Favre P, Chung JP, Budtz-Jorgensen E. Examiner agreement on caries detection and plaque accumulation during dental surveys of elders. *Gerodontology* 1995; **12:** 49–55.
17 Wyatt CC. Elderly Canadians residing in long-term care hospitals. Part II. Dental caries status. *J Can Dent Assoc* 2002; **68:** 359–363.
18 Albino JE, Juliano DB, Slakter MJ. A comparison of six plaque scoring methods for assessing oral hygiene. *J Periodontol* 1978; **49:** 419–422.
19 Matthijs S, Sabzevar MM, Adriaens PA. Intra-examiner reproducibility of 4 dental plaque indices. *J Clin Periodontol* 2001; **28:** 250–254.
20 Soder PO, Jin LJ, Soder B. Computerized planimetric method for clinical plaque measurement. *Scand J Dent Res* 1993; **101:** 21–25.
21 Soder B, Johannsen A, Lagerlof F. Percent of plaque on individual tooth surfaces and differences in plaque area between adjacent teeth in healthy adults. *Int J Dent Hyg* 2003; **1:** 23–28.
22 Soder B, Jin LJ, Lundquist G, Soder P-O. A longitudinal investigation of the individual consistency of plaque levels in adults. *Acta Odontol Scand* 1995; **53:** 72–74.
23 Rose G. What needs to be prevented? In: Rose G, ed. *The Strategy of Preventive Medicine*. Oxford: Oxford University Press, 1992: 6–14.
24 Smith RN, Brook AH, Elcock C. The quantification of dental plaque using an image analysis system: reliability and validation. *J Clin Periodontol* 2001; **28:** 1158–1162.
25 Altman DG. Multiple regression. In: Altman DG, ed. *Practical Statistics for Medical Research*. London, Chapman & Hall, 1992: 336–351.