The dentogingival epithelial surface area revisited


Recent studies implicating periodontitis as a cause of systemic diseases have reported that the surface area of periodontal pockets exposed to bacterial biofilm ranges from 50 cm² to 200 cm². Since the root surface area of the typical human dentition (excluding 3rd molars) is 75 cm², these estimates appear too large. The goal of this study was to relate linear periodontal probing measurements to the dentogingival surface area (DGES). The DGES comprises both the sulcular and junctional epithelium, present in health, as well as any intervening pocket epithelium present in periodontitis. Formulas to estimate the DGES from clinical measures were derived from a meta-analysis of root surface areas, published values of root length, and a study that related the percent remaining root surface area to the percent remaining root length. These formulas were applied to a survey of the adult US population, the Veterans Affairs (VA) Dental Longitudinal Study, and a population of individuals visiting a periodontist. Individuals without periodontitis had a typical DGES of 5 cm². Among individuals with periodontitis, the mean DGES in the three samples ranged from 8 cm² (ranging from 1 cm² to 29 cm²) to 20 cm² (ranging from 2 cm² to 44 cm²). It was concluded that the mean DGES among individuals with periodontitis ranges from 8 cm² to 20 cm², considerably smaller than the range of 50 cm² to 200 cm² currently assumed.

Studies of the association between periodontitis and certain medical conditions have reported that the surface area of periodontal pockets may be a link between periodontitis and these conditions (1-3). There are two commonly proposed biological mechanisms by which periodontal pockets may influence systemic conditions. First, bacteria and their products may pass through the periodontal pockets into the bloodstream causing systemic complications. Second, cells within the connective tissue underlying periodontal pockets may secrete inflammatory mediators, which in turn may lead to systemic problems ranging from low birth weight to cardiovascular disease (4). Some studies reporting an association between periodontitis and systemic diseases indicate that the surface area of pockets is equivalent to that of the palm of the hand (50 cm² to 75 cm²) or that of the ventral surface forearm (±200 cm²) (1, 2, 5, 6). These estimates appear inflated since the root surface area of the typical dentition (excluding 3rd molars) is approximately 75 cm². Most studies reporting these large pocket surface areas have not taken into account existing work on the root surface area of the human dentition.

A more accurate estimate of the surface area of periodontal pockets may be helpful in several ways. First, in non-experimental research, the assessment of dose-response relationships between a suspected cause and outcome may provide clues towards establishing causality (7, 8). If periodontal pockets are causally related to systemic disease, a dose-response relationship between the pocket surface area and the risk for systemic consequences may provide insight into biological mechanisms. Second, an estimate of the pocket surface area may permit a comparison with other diseases that involve ulcerated epithelium such as inflammatory bowel disease, and thereby allow for a re-evaluation of biological plausibility.

Several studies have evaluated the relationship between linear measurements of periodontitis and loss attachment surface area (9-13). The goal of this study was to provide simple formulas to relate linear measurements of clinical periodontitis to the dentogingival epithelial surface area (DGES). These formulas were applied to a representative sample of the adult US population (1985-1986), a sample of 1021 individuals at their initial visit to
periodontal specialists, and the participants of the Veterans Affairs (VA) Dental Longitudinal Study.

Material and methods

When probing periodontal tissues, the periodontal probe tip may be located coronal to or apical to the cemento-enamel junction (CEJ). In health, the periodontal probe tip is mostly coronal to or at the CEJ, whereas in gingivitis and particularly periodontitis the probe extends apical to the CEJ. This study’s primary goal was to estimate the surface area of the dentogingival epithelium (DGES). This descriptive term, DGES, is meant to encompass any sulcular, pocket or junctional epithelium found at the dentogingival junction. Pocket and junctional epithelium are the permeable portions of the dentogingival epithelium, and account for most of the dentogingival epithelium at diseased sites. Therefore, an estimate of the dentogingival epithelial area will reflect the surface area most relevant to any proposed pathogenic mechanism. Since the periodontal probe tip penetrates inflamed connective tissue adjacent to the junctional epithelium, probing depth measurements will lead generally to an overestimation of the DGES, particularly at diseased sites.

Relationship between linear loss and the lost root surface area

Despeignes reported the relationship between the proportion of linear loss along a root, and the proportion of surface area loss (9). These reported proportions were transformed to amount of linear loss and amount of surface area loss using root lengths published in anatomic atlas (14) and the results from a meta-analysis of the root surface area of the human dentition (15). The relationship between surface area (mm²) to linear distance measure (mm) was modeled using polynomial equations of the following form:

\[ Y(\text{Root Surface Area between CEJ and } x) = a_1x^5 + a_2x^4 + a_3x^3 + a_4x^2 + a_5x + a_6 \]  

(1)

where x is the linear distance from the CEJ.

Such equations were fitted for each of the 14 tooth types. Applications of this function provided estimates of the root surface area between the CEJ and a plane perpendicular to the root’s axis at distance x from the CEJ.

Obtaining estimates of variability of surface areas when no information regarding variability in root length is available

The estimates obtained using equation (1) are for a tooth of typical anatomy and length. For any individual tooth within a particular patient, the resulting estimate may be an over- or underestimate depending on whether the tooth is larger or smaller than the typical tooth, and of different anatomical shape. As long as root length has no causal relationship to periodontitis and recession, the assumption that all individuals have a typical root length and surface area does not influence the mean surface area estimate for populations. It does, however, influence the variability of the mean. To obtain an estimate of the DGES’s variability (i.e. standard deviation), the following three steps were taken.

First, for each tooth, the proportion of pocket surface area, \( p \), was calculated by dividing the calculated DGES on a typical tooth by the root surface area of that typical tooth.

Second, for each tooth \( j \) within individual \( i \), a root surface area (RSA) was randomly generated using the following function:

\[ \text{RSA}_j = \text{mean (RSA)} + (N(0,0.89) + N(0,0.45)) + \text{SD (RSA)} \]

where mean (RSA) and SD (RSA) are the mean and the standard deviation of the root surface areas as derived from a meta-analysis (15), \( N(0,0.89) \) is the between-individual variability and \( N(0,0.45) \) is the within-individual variability.

In a third step, the proportion \( p \) obtained in the first step is multiplied with the randomly generated root surface area leading to a DGES estimate for a tooth with a randomly generated size. These adjusted estimates of DGES were used to estimate the variability in the DGES.

Relating linear clinical measures of periodontitis to surface areas of tooth facing epithelium

The application of these formulas is illustrated in three different populations that used different clinical measures of periodontitis. It should be noted that periodontal probing tends to overestimate the actual depths of periodontal pockets and sulci, as the probe tip tends to penetrate inflamed tissues beyond the bottom of the pocket (19, 20). Consequently, the use of probing depth as a basis for the determination of DGES also contributes to overestimation of the DGES.

1. DGES based on clinical attachment level measurements and recession measurements in the National Survey of Oral Health in U.S. Adults and Seniors (1985–1986) — Mesial and buccal recession and probing depth measurements were obtained using a color-coded periodontal probe with a pressure that did not exceed 25 grams. These measurements were obtained from all teeth, except 3rd molars, in randomly selected mandibular and maxillary quadrants.
Detailed descriptions of the study goals and methods have been published elsewhere (16).

Of the 15,132 individuals examined, 14,219 individuals had 6 or more teeth. This population was divided into three subgroups: i) 3,748 individuals with periodontitis, defined as having a site with 3 mm attachment loss and 3 mm probing depth (17), ii) 16 individuals with no attachment loss at any of the sites measured, and iii) 10,361 individuals with some attachment loss but no site with 3 mm attachment loss and 3 mm probing depth (94 individuals had missing probing measurements).

The DGES apical to the CEJ was estimated as the difference between the root surface area coronal to the attachment level minus the root surface area coronal to the recession level but apical to the CEJ. Both surface areas were estimated using the formulas presented in Table 1.

The DGES coronal to the CEJ was estimated by inserting the amount of probing depth coronal of the CEJ into the formulas presented in Table 1. Since crown diameter typically increases coronal to the CEJ, while root diameter typically decreases, use of the formulas in Table 1 will lead to an underestimation of the DGES. This underestimation can be expected to be small since the amount of probing depth coronal to the CEJ is small (≤ 1 mm for 99% of the pocket depths), and the change in diameter coronal or apical to the CEJ is small within the first millimeters. The total DGES was obtained as the sum of the DGES coronal to and apical to the CEJ.

Application of the above methods to both the mesial and the buccal sites results in a DGES estimate coronal and apical to the CEJ. If the two sites are representative for the attachment loss and the recession around the tooth, the average of the two surface areas provides an unbiased estimate for the DGES of the tooth. Similarly, if the randomly selected quadrants represent an unbiased estimate for the mouth, a doubling of the half-mouth estimates results in an unbiased estimated of the total pocket surface area.

2. DGES based on probing depth measurements among 1021 individuals seen by periodontists for a detailed initial examination (1988–1995) — Chart reviews were conducted on a sample of 1021 periodontal patients between the ages of 40 to 65 years and probing depth measurements were obtained at 6 sites around each tooth, except wisdom teeth (18). Probing depths were obtained with manual probes. When only probing depth measurements are available, the location of the pocket with respect to the CEJ is unknown. Therefore, it is not possible to estimate the DGES above and below the CEJ, but it is possible to estimate the total DGES by assuming that all pockets start at the CEJ. Due to root tapering, this assumption may result in an

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<th>Table 1. Polynomials used to relate a linear length in mm to a root surface area in mm²</th>
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1 These numbers represent the coefficients of a₀ through a₅ of the polynomial a₀ + a₁x + a₂x² + a₃x³ + a₄x⁴ + a₅x⁵. For instance, if the attachment loss is 5 mm on a central incisor, the exposed root surface area is 12.3905² + 0.1374² + 0.6717² + 0.5258² - 0.07090² + 0.00589² - 0.0001855² - 0.000383² - 0.01129² - 0.000383² - 0.0001855². If representative probing depth and attachment level measurements for a tooth are available, the root surface area covered by pocket epithelium can be estimated. Because of the overestimation of pocket depth with probing, there is a built-in overestimation of the surface area.

2 RSA is the root surface area for a typical individual obtained from a meta-analysis (15).

3 Length refers to root lengths from the cemento-enamel junction to the apex as published in an anatomical textbook (14).

4 A real length estimate of 13 mm, rather than the published value of 14 mm, was used.
over- or underestimation of the actual surface area depending on whether the pocket is coronal or apical to the CEJ. Since periodontitis is typically associated with recession (i.e. most pockets are apical to the CEJ), the resulting estimate is most likely to be an overestimation.

For all 6 sites around a tooth, the DGES can be estimated as the root surface area from the CEJ to the most apical probing level. If the 6 estimates are considered to be representative of the probing depths around a tooth, the DGES can be estimated by averaging these 6 estimates. By summing all tooth estimates, an estimate per individual is obtained.

3. DGES based on radiographic measures in the Veterans Affairs Dental Longitudinal Study (1969–present) — This longitudinal cohort study of 1,231 male veterans was initiated in 1968 with the goal of determining causation factors in oral health changes in aging individuals. At the baseline examination, mesial and distal bone loss for each tooth was measured on radiographs using a Schei ruler (21). The bone loss was classified on a nominal scale ranging from 0 (no bone loss) to 5 (bone loss up to the apical point of the tooth). For any score between 1 and 5, the average percent bone loss between the worst and the best bone level for that score was assumed. For instance, a score of 3 was interpreted as 50% bone loss. For this population, periodontitis was defined as the presence of at least one site with a radiographic score of 2.

Of the 728 individuals that were seen at all examinations, 415 had periodontitis according to this definition. For the first 6 triannual examinations, the only probing measurement for which data were available was probing depth recorded as an ordinal value. For the present study, the radiographic data were used to estimate the DGES. By doing so, we could illustrate that the proposed methods may provide approximate estimates when no probing measurements are available. The radiographic measurements of periodontitis available at baseline do not provide estimates of the DGES coronal to the CEJ. The DGES apical to the CEJ was estimated by assuming that no gingival recession had occurred at any of the sites. Because recession was likely present, the resulting estimate can be interpreted as the maximum possible estimate of DGES apical to the CEJ.

For each site around a tooth, the DGES can be estimated as the root surface area between the CEJ and the estimated level of bone loss using the formulas in Table 1. For simplicity, wisdom teeth were assumed to be identical to second molars. Since wisdom teeth are typically smaller than 2nd molars, this probably led to a slight over-estimation of the DGES. Similarly to the previous examples, if both the mesial and the distal site are representative for the bone level all around the tooth, the average of both surface area estimates provides an unbiased estimate of the DGES for that tooth.

Results

A 6th power polynomial was fitted relating linear attachment loss to lost attachment surface area. The resulting linear models for all 14 teeth had an R² > 0.998. The coefficients of the model are presented in Table 1. These models allow the calculation of the root surface area between the CEJ and a plane perpendicular to tooth axis at distance x from the CEJ.

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Most studies on the association between periodontitis and systemic disease are performed in populations that are not selected based on a visit to the periodontist. The current study suggests that these types of populations may have smaller DGES than those individuals visiting a periodontist.

In the Veterans Affairs (VA) Dental Longitudinal Study, the DGES apical to the CEJ among individuals with periodontitis was 6.2 cm², a value very similar to the one obtained for the National Survey of Oral Health in U.S. Adults and Seniors. Possibly, in studies of the association between periodontitis and systemic diseases, such as the Physician’s Health Study (45) and the NHANES I study (46), the typical DGES is smaller and similar to the values reported here for the VA Dental Longitudinal Study and the National Survey of Oral Health in U.S. Adults and Seniors. Periodontists are more likely to see those individuals with periodontitis that have deep pockets and larger DGES; however, these individuals are less common in general populations that are typically evaluated in the studies that link periodontitis to systemic disease.

One potential approach to explore potential relationships between the periodontal pocket surface area and systemic disease will be to exploit the substantial variability in the pocket surface area among individuals with periodontitis. One quarter of the individuals visiting a periodontal specialist had a DGES larger than 23 cm² (ventral surface area of two fingers) and about 5 percent had a DGES between 30 cm² and 44 cm² (ventral surface area of three to four fingers). By contrast, in the US survey, 25% of the individuals with periodontitis had a DGES of less than 6 cm². If pocket surface area is linked to an increased risk for systemic diseases, risk differences should be observed for those individuals that have less than 6 cm² versus those that have over 30 cm² of DGES.

The measurement limitations within the three populations may have led to a number of biases. Within the US survey, the DGES may be underestimated due to the inability of the mesial and mesio-buccal sites to properly reflect periodontal destruction around a tooth. Within the VA Dental Longitudinal Study, the DGES apical to the CEJ may be underestimated because of the assumption used in the calculations that no site had gingival recession apical to the CEJ. For those individuals visiting a periodontist, the DGES may have been underestimated because all pockets were assumed to start at the CEJ. The ideal clinical circumstance to obtain an unbiased estimate of the DGES requires random selection of the sites (47) and the availability of both recession and attachment loss measurements.

Additional limitations with respect to methodology include the limitations of the Despeignes study (9), the absence of reliable information regarding the variability in root length from the CEJ to the apex (12), and the absence of information regarding possible relationships between periodontitis and root size. No unbiased stereological studies exist regarding the magnitude of the root surface area and root length. Another limitation is the possible overestimation of the DGES due to partial penetration of the inflamed tissue apical to the pocket during probing. Pending new scientific work in the area of measuring root surface areas, the methodology presented here provides a more reasonable estimate of the DGES than is currently available.

The histology of the DGES has been studied extensively. Although the pocket surface is often considered to be ulcerated and, therefore, a wide-open portal of entry for bacteria and their products into the host tissues, the histopathology of periodontal pockets suggests otherwise (48). In its undisturbed state, the pocket epithelium forms a more or less continuous lining over the diseased soft tissues. This is particularly evident in pockets that do not bleed on probing (49). In pockets that exhibit bleeding on probing or suppuration, micro-ulcerations may be detected in localized regions, where heavy migration of polymorphonuclear leukocytes from the tissues into the pocket leaves discontinuities in the epithelial lining. However, the epithelial lining is generally quite thin and readily disrupted by mechanical stimuli such as a periodontal probing. Since junctional epithelium is quite permeable to assorted soluble products (50, 51), it is likely that this is also the case for pocket epithelium. Since the DGES includes pocket and junctional epithelium as well as sulcular epithelium, the surface area of the pocket epithelium is likely to be overestimated by using the DGES to estimate the surface area of pocket epithelium.

In summary, depending on the population studied, periodontitis leads to a mean increase in the DGES of between 3 cm² and 15 cm². Among individuals with periodontitis in the general population, the increase in DGES associated with periodontitis was typically small, about the ventral surface area of a fingertip. Among individuals visiting a periodontist, the increase in surface area was about five times larger, in the range of the ventral surface area of one to two fingers. The hypothesis that the size and the inflammatory status of the pocket surface area is causally related to systemic diseases remains to be tested. It needs to be shown whether the absolute size of the DGES or its relative increase in periodontitis is of sufficient
magnitude to represent a clinically important risk factor for systemic health.

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