Letters to the Editor

A comment on »Salivary IgA in periodontal disease»

In a recent issue of this journal *Lindström and Folke* (1973) published data on the quantitation of IgA in whole saliva and parotid fluid from patients with periodontitis. These authors are, apparently, surprisingly unaware of available information relevant to their study. Their method of quantification entails so many possible sources of errors that the presented data are likely to be misleading rather than to contribute to a better understanding of local immunity.

Several studies have demonstrated that it is of little value to quantitate salivary IgA without taking flow rate into consideration. »Unstimulated» parotid secretions thus contain about three times more IgA than stimulated (*Brandtzaeg*, 1971). It has therefore been advised to report the output of salivary proteins in secretion rates (μg/min). This can, of course, be done reliably only for secretions collected from duct openings. The contribution to whole saliva from the minor, submandibular, and parotid glands vary greatly according to the rate of flow (*Kerr*, 1961). Centrifugation, concentration, and storage of saliva will moreover introduce a variable and uncontrolled loss of immunoglobulins (*Brandtzaeg et al.*, 1970). Secretory stimulation by paraffin chewing should definitely be avoided since the wax absorbs organic material, and the chewing enhances leakage of plasma proteins into the oral cavity. While the 7S fraction of parotid IgA has been estimated to be about 10 per cent, it is 13—17 per cent in unstimulated whole saliva, depending on the state of the gingiva (*Brandtzaeg et al.*, 1970). The relative concentration of IgG is also significantly increased in whole saliva, and this has been shown to be a function of the degree and extent of gingival inflammation (*Brandtzaeg et al.*, 1970). In addition to such sampling problems, immunological quantitation of IgA involves many technical difficulties with regard to type and protein determination of the standard antigens, and specificities of the antisera. This has been discussed in detail elsewhere (*Brandtzaeg et al.*, 1970).

Lindström and Folke (1973) found a raised level of IgA in whole saliva from individuals with periodontitis. Without any attempt at physicochemical analyses they ascribed this increase to admixture of 7S IgA from the gingival fluid. However, *Brandtzaeg et al.* already in 1970 found a significantly increased level of 11S IgA in the whole saliva from such patients. Since the immunoglobulin levels were measured as mg/100 ml rather than as μg/min, the latter authors discussed two possible explanations for this result: (1) The salivary flow rate might be different in patients and controls; or (2) There could be a more intense stimulation of IgA immunocytes in the salivary glands of patients with periodontitis because of larger aggregates of live bacteria in contact with their oral mucosa. Such stimulation

might well be restricted to the submandibular glands and thus not be reflected in higher levels of IgA in parotid secretions. However, in a recently published abstract *Chandler et al.* (1972) reported a positive correlation between the prevalence and severity of periodontal disease and the secretion rate of parotid IgA.

Extensive and well-defined studies have to be carried out in order to obtain useful information with regard to an association between oral disease and local immune responses. In addition, much basic research is required to throw light on the function of salivary antibodies. As a working hypothesis it has been postulated that while glandular 11S IgA responses represent a »first line of defense» in the oral cavity, a »second line of defense» is constituted by IgG and 7S IgA responses taking place in the buccal mucosa and gingiva (*Brandtzaeg*, 1972; 1973).

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