Annotation

Identification of C 3a, IgG, IgM in Inflamed Human Gingiva

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The host reaction to bacterial antigens in the gingival sulcus in gingivitis suggests a combined hypersensitivity response as IgG, IgA, IgM, and IgE have been reported both in the gingiva and the sulcus fluid (SCHWARTZ & DIBLEE: *J Periodontol* 46:171, 1975; BYERS, TOTO, & GARGIULO: *J Periodontal* 46:387, 1975; BRILL: *Acta Odontol Scand* 18:421, 1960). Evidence of hypersensitivity of the host to bacterial antigens in gingivitis indicates that the inflammation process is antigen-antibody induced (NISENGARD & BEUTNER: *J Periodontol* 41:223, 1970; SNYDERMAN: *JADA* 87:1020, 1973; IVANYI & LEHNER: *Arch Biol* 15:1089, 1970). Autoimmunity has been indicated as a pathogenetic mechanism in gingivitis (BRANDTZAEG & KRAUSE: *Odont T* 73:281, 1965.)

Antigen-antibody complexes by IgG and IgM bind complement which liberate C 3a fragments by the direct pathway which cause the anaphylotoxic chemotactic and cytotoxic events seen in hypersensitive reactions in the host (MULLER-EBERHARD: *Ann Rev Biochem* 38:389, 1969). Fluorescent antibody methods have been used to identify IgG, IgM, and IgA in human gingivitis (COONS: In *General Cytochemical Methods.* Vol. I. J.F. DANIELLI, (ed), New York, Academic Press, 1958).

This study employed goat antihuman IgG, IgM, and C 3 bound to fluoresceine isothiocyanate to identify such proteins in human inflamed gingiva. The antiserum was reacted with human erythrocyte to eliminate binding to blood group substances. Twenty-five patients with chronic periodontitis requiring gingivoplasty were used for the collection. Tissue fragments approximately 0.5 × 0.5 cm were cut, frozen on a cryostat and sections 8 to 10 μ were cut. The sections were washed with phosphate buffered saline to remove unbound globulins. Sections were incubated with the specific antisera for 45 minutes at 37 C, washed 3 times in phosphate-buffered saline and mounted in glycerine. The sections were examined with a fluorescent microscope.* The presence of green fluorescence was scored positive; blue fluorescence was scored as autofluorescence. Hematoxylin and eosin control slides were made of all specimens.

All specimens showed evidence of chronic inflammation containing plasma cells, lymphocytes, and polymorphonuclear leukocytes.

All specimens reacted positively to the presence of IgM, IgA, and C 3. IgG was found in the cytoplasm of plasma cells and basement membrane of the epithelium and capillaries (Fig 1). Similarly, IgM was seen on the basement membrane of the epithelium and intercullularly (Fig 2). C 3a was seen in the basement membrane of the epithelium and capillaries.

No assessment could be made as to whether the basement membrane was "shaggy" or smooth as the fluorescence extended intercellularly in the epithelium; however, the fluorescence appeared irregular. The evidence of IgG, IgM, and C 3 in the basement membrane and intercellularly suggests that antigen-antibody complexes fixing complement are present in all cases of gingivitis.

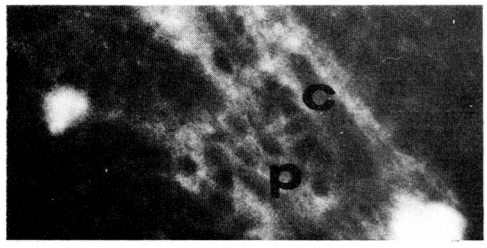

FIG 1. — IgG in the cytoplasm of a cluster of plasma cells (P) and in the basement membrane of a capillary (C).

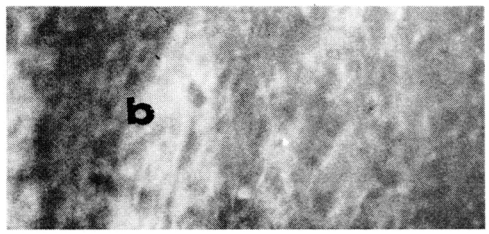

FIG 2. — IgM shows as a pale deposit on the epithelial basement membrane (B), tangentially cut section.

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*Zetopan with HBO 200 Hg light source, A.O. Co., Buffalo, NY.