Communications

Glycogen Deposits in Gingival Tissue

Glycogen deposits have been associated with keratin production in wound healing (1), hair growth (2), and cyclic changes in the endometrium (3), where the glycogen is regarded as a possible source of energy for the production of keratin. Heavy deposits of glycogen are present in the keratinized marginal epithelium of inflamed, human gingival tissue but not in the adjacent nonkeratinized crevicular epithelium that lines the gingival pocket. Gingival tissue from the gingival margin and interdental papilla was, therefore, examined (4) to see if any relationship existed; biopsy material was collected from 15 patients with clinically normal gingivae or only very slight marginal gingivitis and 41 with gingivitis or periodontitis.

In normal gingivae the marginal epithelium was keratinized and free from glycogen deposits. In the inflamed gingivae, where leucocytic infiltration of only the connective tissue occurred, glycogen was deposited in the superficial cells of the marginal epithelium where the ribonucleic acid (R.N.A.) content was low, irrespective of the state of keratinization of the epithelium. Glycogen was not deposited in those portions of the marginal or crevicular epithelium where leucocytic infiltration extended from the connective tissue into the epithelium; R.N.A. content was low in these areas.

Menkin (5) has shown that in inflammation in diabetics, protein of the damaged tissue is broken down to glucose (gluconeogenesis) with a resultant increase in glucose in inflammatory exudates. In nondiabetics, the glucose is usually removed by glycolysis, but it is suggested that, in the inflamed gingiva, the glucose is not all broken down. This glucose could diffuse out of sites of inflammation (6), and on a cellular level glucose could diffuse into and accumulate in adjacent epithelium and be stored as glycogen.

Concentration of R.N.A. is high where growth is taking place, and in my material glycogen was not deposited in the basal cells of the epithelium where the R.N.A. concentration was high; these cells apparently use any extra glucose in growth and cell division. In the Malpighian and more superficial layers, however, R.N.A. was low and mitotic figures entirely absent; these cells apparently do not use any extra glucose as a source of energy but deposit it as glycogen. These results are supported by observations made on skin (7).

The nonoccurrence of deposits of glycogen in the inflamed nonkeratinized crevicular epithelium may be explained by infiltration of this tissue by leucocytes and other elements; leucocytes possess both amylase and nuclease. It is known that some intercellular substance is lost in this region, so that enzymes could more readily diffuse into the epithelium, either from the gingival pocket or from the inflamed subepithelial zone; also glucose could more readily diffuse out of the epithelium.

It is suggested, therefore, that the reason for glycogen deposition in inflamed gingiva is the presence of excess glucose (from gluconeogenesis) that diffuses into the epithelium. The glycogen that is deposited is apparently from glucose in excess of that which might be needed for keratinization and other syntheses and appears to be a mechanism for disposal of unwanted glucose.

A detailed report of this work will be published in the Journal of Periodontology.

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16 April 1954.

Dust-Snow Storm in the Minneapolis-St. Paul Area on 12 March 1954

Dust storms are very common in the United States and have been discussed in the literature many times (1). The present notice is, therefore, only a local record on one of such storms in the Twin Cities. This dust-snow storm took place on Friday, 12 March 1954, after an extremely dry winter. In the early morning, the sky became yellowish-reddish in color, and the snow was a notably dirty, pinkish color. At places the dust accumulated on the surface of the snow, owing to sorting by the wind, which reached velocities of 40 to 50 mi/hr. Similar "red snow" was reported by the local press and radios over all of the upper Midwest as well as in other states. The snowfall on the next day was represented by usual pure white snow in Minneapolis. The dust was too fine for usual microscopic study, and only a few small angular quartz grains and some indeterminable organic (?) tissue have been observed. An x-ray study of the dust collected at the University of Minnesota, Minneapolis campus, showed the presence of quartz and feldspar patterns. The abundance of these two minerals was typical also for the dust storm in 1918 and other storms. The presence of quartz was recorded also on a thermodifferential curve made from the dust. The bitumina extraction of 2 g of dust with carbon disulfide in a Sohxlet-Allihn extractor showed a presence of lipoids in the amount of about 0.5 percent by weight. The origin of the red color of the snow is somewhat uncertain. It may be explained, perhaps, by the presence of small amounts of iron hydroxide pigment in the dust.

The water precipitated during the day of the storm, as reported by the weather bureau, was 0.66 in. A spot sample of snow collected on the campus showed 2.98 g of dust per liter of water. This gives an average amount of dust of 128.8 tons/mi². Even reducing this number to 50 tons because of the possibility of uneven distribution of dust, it would amount to a minimum of 75,000 tons of dust falling on the basic Twin City area—about 1500 mi².

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14 June 1954.

On the Fluorometric Determination of N¹-Methylnicotinamide

Huff and Perlzweig (1) developed a fluorometric method for the estimation of N¹-methylnicotinamide based on the condensation of it with acetone under alkaline conditions. Huff (2) has shown the condensation product to be a highly fluorescent napthyridine derivative. More recently, Kato et al. (3) have reported that the intensity of fluorescence may be increased when samples of N¹-methylnicotinamide are treated with alkaline hydrogen peroxide before the analytic procedure is applied. We have found that pretreatment of samples with alkaline or neutral hydrogen peroxide may completely destroy N¹-methylnicotinamide, depending on the concentration and on length of time in which the samples are in contact with



Fig. 1. Effect of hydrogen peroxide on the fluorescence of N¹-methylnicotinamide condensed with alkaline acetone: A, N¹-methylnicotinamide samples pretreated with neutral hydrogen peroxide; B, N¹-methylnicotinamide samples treated with hydrogen peroxide after the addition of acetone and alkali. Data obtained by the analytic procedure of Huff and Perlzweig (1) using 0.8 µg. N¹-methylnicotinamide in a final volume of 10 ml. The fluorescence of the uncatalyzed reaction was arbitrarily taken as zero.

peroxide (4). Huff and Perlzweig have previously mentioned the rapid destruction of N¹-methylnicotinamide in alkaline solution.

Hydrogen peroxide, however, is effective in catalyzing the reaction when the peroxide is added to the reaction mixture after the acetone and alkali additions (Fig. 1). A plot of peroxide concentration versus the change in fluorescence from that of the uncatalyzed reaction demonstrates the concentration of peroxide necessary to obtain maximum fluorescence. At concentrations greater than the optimum, N¹-methylnicotinamide may be destroyed. Pretreatment of the samples with neutral hydrogen peroxide, immediately before application of the analytic procedure, resulted in a lower fluorescence response at the optimum concentration of peroxide and in greater destruction of N¹methylnicotinamide at high concentrations of peroxide.

The data also indicate the necessity for rigid control of peroxide concentrations in order to obtain reproducible results.

In a study of the factors affecting the analytic procedure, we have found that a large number of inorganic elements catalyze the formation of a fluorescent derivative. Iridium and cerium salts have the greatest activity at concentrations of 5×10^{-6} and 8×10^{-6} M, respectively. These salts are almost 1000 times more active than hydrogen peroxide at optimum concentrations. A complete report of the factors affecting the formation of the napthyridine derivative is in preparation and will be published elsewhere.

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5 February 1954.

A New Enemy of the Oyster Drill

It has been reported that the oyster drill, Urosalpinx cinerea (Say), and Eupleura caudata (Say) is its own worst predator (1). However, the moon snail, Polinices duplicata (Say), may in some areas destroy more oyster drills than the drills themselves destroy.

Dead drills from the Lower Miah Maull area of Delaware Bay, 937 in number, were examined for cause of death. Of these, 100 contained the large, heavily countersunk hole typical of the moon snail, and 76 contained the small, slightly countersunk hole typical of the oyster drill. These dead drills were obtained from material removed from the leased oyster beds by drill screens and a drill dredge. Drill screens and drill dredges are screening devices used by the ovstermen to remove drills from their oyster beds.