

activation-analysis equipment, especially neutron generators and multichannel pulse-height analyzers, provided by American and European manufacturers.

Publication of the proceedings is not planned, but abstracts of the 23 papers presented are available from the program supervisor, J. M. A. Lenihan, Regional Physics Department, 9 West Graham Street, Glasgow, C.4.

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## **Dental Caries: A New Look**

Dental caries is a multifactorial disease whose gross manifestations are preceded by events on the molecular, atomic, and subatomic levels. It was specifically to explore and delineate these events that the New York Academy of Sciences sponsored a conference on the mechanisms of dental caries 30 November-1 December 1964 in New York City. The conference represented a multidisciplinary attack on the problem, rather than the usual clinical approaches which have been exhaustively explored in previous dental symposiums.

The mineral structures of the tooth and the physico-chemical laws governing the dissolution of the mineral components were dealt with at the first session. W. E. Brown (American Dental Association) and B. M. Wallace (National Bureau of Standards) pointed out that calcium and phosphorus ions may diffuse through enamel at different rates and that, as a result, an increased concentration of calcium, phosphorus, and hydrogen ions might occur within the enamel; this could account for the subsurface dissolution of the structure. The mechanisms of diffusion of these ions through the enamel can be explained by treating the enamel layer as a semipermeable membrane according to L. S. Fosdick (Northwestern University). Similar results were obtained by M. D. Francis (Miami Valley Laboratories, Proctor & Gamble), who demonstrated a surface complex which controls the rate of dissolution of the underlying enamel structures.

The effects of fluoride were examined in a series of papers from A. S. Posner's (Cornell Medical) group



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| COMPOUND   | ACTIVITY<br>(mc/mM)                         |
|--|---|
| L-Alanine-C14 (U)  | 5-10  |
| L-Alanine-C14 (U)  | 75-110                                      |
| [Aqueous solution]   | E 10  |
| -Arginine-C14 monohydrochloride  | 150.220                                     |
| (U) [Aqueous solution]   | 130-220                                     |
| L-Asparagine-C14 (U)   | 4-30  |
| L-Aspartic-C14 acid (U)  | 5-10  |
| L-Aspartic-C14 acid (U)  | 100-150                                     |
| [Aqueous solution]   | E 10  |
| [Mono-ammonium salt]   | 5-10  |
| L-Glutamic-C14 acid (U)  | 125-180                                     |
| [Aqueous solution]   |   |
| L-Glutamine-C14 (U)  | 5-40  |
| Glycine-C14 (U)  | 5-10  |
| Glycine-C14 (U) [Aqueous solution]   | 50-70                                       |
| L-Leucine-C14 (U)  | 150.220                                     |
| [Aqueous solution]   | 130-220                                     |
| L-isoLeucine-C14 (U)   | 5-10  |
| L-isoLeucine-C14 (U)   | 150-220                                     |
| [Aqueous solution]   |   |
| (U)  | 5-10  |
| L-Lysine-C14 monohydrochloride<br>(U) [Aqueous solution]   | 150-220                                     |
| L-Phenylalanine-C14 (U)  | 5-10  |
| L-Phenylalanine-C14 (U)  | 200-320                                     |
| [Aqueous solution]   | F 10  |
| L-Proline-C14 (U)  | 125 190                                     |
| [Aqueous solution]   | 125-160                                     |
| Protein hydrolysate-C14 (U) 200-<br>[From Chlorella Vulgaris]  | 300 µc/mg                                   |
| L-Serine-C14 (U)   | 5-10  |
| L-Serine-C14 (U)   | 75-110                                      |
| [Aqueous solution]   | E 10  |
| L-Inreonine-C14 (U)  | 100 150                                     |
|  | 100-150                                     |
| L-Tyrosine-C14 hydrochloride (U)   | 5-10  |
| L-Tyrosine-C14 hydrochloride (U)   | 200-320                                     |
| [Aqueous solution]   | <b>F</b> 10                                 |
| L-Valine-C14 (U)   | 5-10  |
| [Aqueous solution]   | 125-180                                     |
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at the Hospital for Special Surgery. They found that the incorporation of fluoride increases the sharpness of the x-ray diffraction patterns of hydroxyapatite, indicating an improvement in the crystallinity of the mineral toward a more "perfect" apatite. In this respect, Koulourides, Pigman, and Feagin (University of Alabama) reported that while fluoride accelerates the remineralization of enamel, it is ineffective unless calcium and phosphate ions are incorporated into the remineralizing solutions.

The other sessions dealt with the biological structures of the tooth and with their biochemistry and pathology during caries. The concept that enamel is not a dead tissue like hair and nails was stressed by T. B. Coolidge (University of Chicago) in his demonstration of the existence of submicroscopic channels along the apatite crystals. These channels are normally closed at the completion of calcification but are found to be reopened at the onset of caries. H. H. Neumann and N. A. Disalvo (Columbia University) presented the thesis that the process of chewing, in which measurable lengthening and shortening of the teeth occur, influences by compression the molecular structures in the teeth.

The biological dynamism of this once thought "dead" structure was further demonstrated by G. Neil Jenkins (King's College, England), who reported that the plaque was laid down from the proteins of the saliva and only subsequently colonized by acidproducing bacterial flora. S. Wah Leung (University of British Columbia) and I. D. Mandel (Columbia University) emphasized anew that human saliva is made up of a number of components. Mandel's report of differences in the proteins of parotid and submaxillary salivas was of particular interest

The role of bacteria in the production of acid was studied by H. V. Jordan (National Institutes of Health), using gnotobiotics. He found that plaque and caries were absent in germfree hamsters but could be produced at will by infecting the animals with cariogenic streptococci. However, the role of bacteria in the production of acid was disputed by V. F. Lisanti and B. Eichel (Institute of Stomatological Research, Brookline, Massachusetts). They attributed the greater glucolytic activity to mammalian leukocytes present in the oral cavity. A masking effect which covers the acid production by bacteria in the human mouth was attributed by J. Tonzetich and S. Friedman (Colgate-Palmolive Co.) to exfoliated epithelial cells and their greater metabolic activity.

The coordination of calcium by certain naturally occurring complexing agents was shown by M. L. Schole (Bronx-Lebanon Hospitals) and J. F. Frederick (Dodge Chemical Co. Research Labs.) to be a probable mechanism for releasing protons. These protons, normally displaced from biological ligands by the coordinated cation, could add to the sum total of hydrogen ions influencing the dissolution of the enamel.

The possibility of an anti-caries "vaccine" was suggested by the brilliant presentation by H. Blechman and M. Mori (New York University) of proof of antibody production and the presence of antigenic substances in carious dentin.

The audience was brought up to date on the epidemiological approach to the problem by J. Dunning (Harvard University) and on the prospects for future research by B. Bibby (University of Rochester).

The papers presented at the conference will be published by the New York Academy of Sciences.

JEROME F. FREDRICK Dodge Chemical Company, Bronx, New York

## **Forthcoming Events**

## January

27-30. Geological Soc., Southwestern Federation, Austin, Tex. (S. P. Ellison, Jr., Department of Geology, Univ. of Texas, Austin)

27-31. Neurosurgical Soc. of America, San Juan, Puerto Rico. (C. H. Davis, Jr., Bowman Gray School of Medicine, Winston-Salem, N.C.)

28–29. Interactions of Man and His Environment, symp., Chicago, Ill. (W. K. Stuckey, Dept. of Public Relations, 1802 Chicago Ave., Northwestern Univ., Evanston, Ill. 60201)

28-29. Rheology Soc., winter meeting, Santa Barbara, Calif. (R. S. Porter, California Research Corp., Richmond Laboratory, 576 Standard Ave., Richmond, Calif.)

28-30. American Geophysical Union, southwest regional, Socorro, N.M. (J. B. Franzini, Civil Engineering Dept., Stanford Univ., Stanford, Calif.)

28-30. International Medical Assembly of Southwest Texas, San Antonio. (S. E. Cockrell, Jr., 202 W. French Pl., San Antonio 12)

28-30. Large-Scale Air-Sea Interaction, symp., Bombay, India. (UNESCO, Office of Oceanography, Pl. de Fontenoy, Paris 7<sup>e</sup>, France)

22 JANUARY 1965



## Bad Day at the Fujiyama Sake Mill

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