DENTAL CARIES

AMERICAN DENTAL ASSOCIATION

DENTAL CARIES

Findings and Conclusions on Its Causes and Control

Stated in 237 summaries by observers and investigators in twenty-six countries

Compiled for the Research Commission
of
The American Dental Association

By

The Advisory Committee on Research in Dental Caries:

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prevented, to a large extent, by polishing all rough surfaces and filling every flaw, groove, pit, or fissure where stagnation occurs. This method of care is not "100-percent effective" against caries, but with it *no permanent teeth were lost by 366 children up to ages 10 to 14 years.* Oral mucous-glands discharge their secretion directly on the teeth. Normally this secretion is germicidal and *inhibits bacterial action on carbohydrates in retained food debris,* thus preventing formation of the acid that otherwise would start caries. In susceptible persons, caries varies in proportion to the inhibiting quality of the oral mucus. Worry, fear, and other disturbing mental conditions, frequently influence the germicidal power of the mucous secretions, permitting caries. There is no relationship between incidence of caries and (*a*) degree of hardness of teeth (in soft teeth the process is more rapid), or (*b*) adequacy of diet (children receiving deficient diets may be free from caries; children on adequate diets may show excessive caries).

Some children showing little evidence of home care, and plenty of lodgment of food debris, may be free from caries. *Why*—with all necessary destructive factors present? Since caries is an individual disease, and not communicable, the *conditions that allow it to be produced* are also individual. These individual mental or digestive disturbances frequently affect the bacteria-inhibiting ability of oral mucous-secretions and thus favor initiation of caries. In one person it may be nervous indigestion; in another, overeating rich foods; in another, consumption of too much sugar (which is also highly irritant to sensitive oral membrane); in another, too much cereal food—the inability of some persons to utilize cereals suggests a form of allergy. *Continuation* of these conditions usually affects the inhibitive capability of the oral mucous-secretions.

Reference: J. Am. Col. Den., 6, 65, 1939.

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In an experimental study in *rats*—in which "cusp caries" produced by coarse corn-meal was used as an index—increased caries-immunity in the young resulted after feeding haliver oil to mothers, during pregnancy and lactation, in excess of the amount necessary to prevent rickets. A wide range in ratio of calcium to phosphorus was tolerated by mothers without effect on caries-resistance in the young, but an extremely low ratio reduced resistance. A 100-percent-meat diet, supplemented with calcium carbonate, reduced the number of cavities from 10 per control to 2.5 in the young of mothers on the meat diet. In experiments with diets that initiate caries, fermentable carbohydrates and

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thermal shock had no effect. In experiments with diets that alter the rate of progress of caries, fermentable carbohydrates promoted enlargement of existing cavities; factors operating through metabolic channels—vitamins A and D, and increased calcium and phosphorus—had no effect. Sodium fluoride, given orally to rats *during pregnancy and lactation,* caused development of significant resistance to caries in the young; their molars were not mottled. Mottled enamel was produced in molars by direct daily doses of sufficient amounts of sodium fluoride to suckling rats. The technique provides a means to study the relation of caries to mottled enamel, as distinguished from the optimum fluorosed enamel. From these data and critical examination of findings of others the author concluded that, (*a*) if fluorine is present in optimal amount during the formation of enamel, the subsequent caries-resistance of teeth is greatly increased, and (*b*) structure of enamel is the dominant factor in pre- vention of the initiation of caries.

References: Den. Rays, 1937 (8); *Science,* 1939 (83); *J. Am. Water Wks. Assoc.,* 1939 (1926); *J. Den. Res.,* 1939 (481); *J. Am. Den. Assoc.,* 1940 (1107).

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In caries after the initial lesion of enamel, caused probably by an external agent—indicated by the "brown spot"—there follows reaction of the pulp, causing dissolution of mineral components in the corresponding sector of dentin where Tomes fibres pass from pulp to brown spot. Brown spots on interstitial surfaces of teeth, opposite cavities, seem to be produced by solvent actions of phosphoric acid carried from the cavities by saliva. Caries begins at predilec- tionous points where bread and other food particles are retained. Aqueous extracts of bread respond to tests for phosphoric acid, which dissolves the sub- microscopic crystals of hydroxylapatite in enamel. Bacteria in carious cavities are there only as saprophytes—living on the demineralized organic substance of dentin. In test-tubes containing bouillon-saliva-powdered dentin, the growing microörganisms taken originally from carious cavities do not split any phosphoric acid from the dentin.

Aqueous extracts of deeper layers of carious dentin are often acid (bromcresol- purple), but tests for lactic acid or calcium lactate in these extracts have been negative—acidity was due to phosphoric acid. These facts demonstrate that, in carious dentin, hydroxylapatite is resolved into its components. Since the quali- tative test for lactic acid is not specific, is also given by other substances that may be oxidized into acetaldehyde, and the intensity of the test does not increase in a fermenting saliva-sugar mixture, lactic acid does not appear to be a factor in the progress of caries.

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