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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Second Edition

New York

LANCASTER PRESS, INC., LANCASTER, PA.

CONTENTS

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.

*COX, GERALD J., M.S., PH.D.: *Mellon Institute of Industrial Research, University of Pittsburgh, Pittsburgh, Pa. (Jan. 30, 1941).* [For himself and co-workers: MARY L. DODDS, M.S., PH.D.; W. E. WALKER; SARA F. DIXON, M. L.; *and* MARGARET C. MATUSCHAK, B.S. in Chem.]

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

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 prevention 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).

*CSERNYEI, A. JULIUS, M.D.: *Odontoiatric Clinic, University of Bologna, Milan, Italy (Dec. 29, 1940).*

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 predilectionous points where bread and other food particles are retained. Aqueous extracts of bread respond to tests for phosphoric acid, which dissolves the submicroscopic 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 qualitative 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.

Using vitamin D in oil only (3000 internat. units per grm.), nine out of ten cases showed barrier stimulation. Controls, using boiled olive oil, showed no response.

References: Some observations on the conservative treatment of the dental pulp, M.D.S. thesis, University of Durham, 1935; *Den. Rec.*, 1939 (492).

HODGE, HAROLD C., B.S., M.S., Ph.D.: *School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* (*Aug. 14, 1939*).

Chemically, carious whole-teeth differ from sound ones apparently because the carious process removed relatively more enamel than dentin, and more inorganic than organic dentinal tissue. Carious whole-teeth show higher contents of moisture but little or no difference in inorganic percentages, nor in calcium, phosphorus or magnesium percentages of inorganic residue. There seems to be no relation between tooth hardness and caries incidence. Physically, three fundamental properties have been reported on a few teeth each. Quantitative x-ray absorption measurements show a hypercalcified dyke between the active caries-zone and the transparent and opaque reaction-zones. Hardness measurements also show a dyke of increased hardness (up to 100 percent harder) between the putty-like carious area and the reaction zones. In some cases this dyke has consisted of transparent dentin. Normal, transparent and opaque zones have identical x-ray diffraction patterns, and therefore identical molecular constitutions of the principal inorganic component (hydroxylapatite). Fluoride, added to a caries-producing diet given to rats from the time of weaning, reduced incidence of both fractures and caries in the molars. Since at least the first and second molars were fully calcified and had erupted before any fluoride was given, the molars chiefly involved did not have mottled enamel.

References: Numerous since 1933; chiefly in *J. Am. Den. Assoc.*, 1933; *J. Biol. Chem.*, 1933; *Am. J. Roentg. Rad. Therap.*, 1934; *J. Den. Res.*, 1934, 1937; *Ind. Eng. Chem.* (*Anal. Ed.*), 1938.

*HOFFMAN, O. E., D.D.S.: *Iowa State Department of Health, Des Moines, Iowa* (*Jan. 21, 1939*).

Fifty-six case-records, selected by chance or random sampling from the practice of Dr. James M. Prime—showing effects of treatment with Howe's solution to arrest proximal incipient caries—were subjected to statistical analysis by the Division of Dental Hygiene. The applications of Howe's solution were repeated for periods ranging from a few months to more than six years, the treatment-interval varying from about three months to more than six. The total number of etched (minimal carious) areas thus treated was 887; the total number of treatments was 5304—approximately 6 per carious surface.

Caries was arrested in every case. A questionnaire, issued by the same Division, on causes and control of caries, included the recent responses, in *Table 3*, from 699 dentists in Iowa—12 not in active practice.

TABLE 3

Responses to a questionnaire on causes and control of caries

Question A. What do you believe to be the cause or causes of tooth decay?

(1)	Inadequate nutrition	502	(14)	Insufficient coarse foods	5
(2)	Improper oral hygiene	414	(15)	Poor assimilation	3
(3)	Overingestion of carbohydrate food, starches and sugars	390	(16)	Worry and fear	3
			(17)	Insufficient rest and relaxation	2
(4)	Vitamin deficiency	386	(18)	Improper prenatal care	1
(5)	Hereditary factors	308	(19)	Bolting food	1
(6)	Aciduric group of microörganisms	257	(20)	Deep sulci	1
			(21)	Insufficient rest-period for mother before child birth	1
(7)	Endocrine dysfunction	193			
(8)	Streptococcus odontolyticus	70	(22)	Demineralized soil	1
(9)	Improper mastication	11	(23)	Acid saliva	1
(10)	Malocclusion	11	(24)	Chocolate foods	1
(11)	Improper prenatal nutrition	9	(25)	Poor operative dentistry	1
(12)	Metabolic imbalance	8	(26)	Malformation	1
(13)	Childhood diseases	7	(27)	Chewing gum	1

Question B. What preventive treatment do you find successful for deciduous teeth?

(1)	Silver nitrate solution (Howe's)	343	(21)	Carbo-eugenol	4
(2)	Adequate nutrition for child	256	(22)	Pulprotex	3
(3)	Tooth brushing	136	(23)	Inlays	2
(4)	Copper amalgam	76	(24)	Extraction	2
(5)	Early cavity filling	65	(25)	X-ray	2
(6)	Prophylaxis	65	(26)	Milk of magnesia	2
(7)	Early and frequent examinations	52	(27)	Cod-liver oil	2
(8)	Prenatal nutrition	41	(28)	Zinc chloride-eugenol	1
(9)	Copper cement	39	(29)	Zinc oxide and silver nitrate	1
(10)	Amalgam	34	(30)	Pustolene	1
(11)	Calcium-phosphate	21	(31)	Alkalizing	1
(12)	Zinc oxide-eugenol	20	(32)	Merthiolate, 1–1000 sol.	1
(13)	Red copper cement	15	(33)	Public school instruction	1
(14)	Child-dent alloy	13	(34)	Formalin	1
(15)	Clean out decay	9	(35)	Neo-siltrate	1
(16)	Eugenol	6	(36)	Acriflavine	1
(17)	Sterilizing agent (not specified)	6	(37)	Thymozine	1
(18)	Grinding and polishing roughened surfaces	5	(38)	Reduction of carbohydrates in diet	1
			(39)	No candy	1
(19)	Black copper cement	5	(40)	Phenol	1
(20)	Silvo-dent	5	(41)	Sodium bicarbonate	1

Reference: Dental Report No. P.D. 5, Div. Maternal and Child Health, Ia. State Dep. Health, 1939.

Addendum (*Mar. 17, 1941*): Conservative estimates, based on data received from dentists in private practice in this State, indicate that the life of 750,000 deciduous and 840,000 permanent teeth is being prolonged annually in Iowa by the current widespread use of Howe's solution. Responses to a related questionnaire recently distributed to Iowa dentists, now being received, provide the information in the following summary as of the date of this addendum:

Question—"Check the following items in which you find the use of Howe's

saliva (8 mgms. or less per 100 cc.) is characteristic of susceptibility to caries; but *Bunting, Karshan, Scrivener,* and *Youngburg* disagree with this conclusion. [In the second edition *Scrivener* omitted his allusion to ammonia.]

2. *Oral bacteria*

There is general acceptance of the conclusion that caries is initiated by oral acidogenic bacteria; that *L. acidophilus* is the attacking organism;[6] and that proteolytic bacteria destroy the organic matter in the path of the carious invasion. Several authors either do not recognize bacterial initiation of caries (*Bregstein, Broderick, Mellanby*), or believe that bacteria are present in the carious lesion only incidentally (*Briggs, Proell*). A number of authors have found that microörganisms other than, or in addition to, *L. acidophilus* are active in the initial process (*Belding, Berke, Bibby, Fosdick, Hearman, Hine, Lyons, Okumura, Westin*). Acidogenic bacteria, including *L. acidophilus,* have been found frequently in mouths in which caries did not occur or had been arrested. The following comment, by *Köszeg,* illustrates findings of differences in the bacteriology of the mouths of persons and animals: "When human saliva is mixed with sugar-broth . . . the pH generally attains values between 4.0–5.5—acid enough to attack enamel. When dog saliva is mixed with sugar-broth, acidification does not occur, but the ensuing pH is in the alkaline range. Dogs are immune to caries; lactobacilli do not live in their mouths." *Fish* states that "the dog's immunity to caries appears to be due to an anti-bacterial principle. A human carious tooth fixed in a dog's mouth becomes *sterile* in from two to four days. In a susceptible monkey's mouth the organisms do not die out and *S. mutans* can be recovered from an implanted human tooth after eleven days." *Gottlieb* presents this "problem for further research: Can resistance [to caries] be improved or immunity established, by increasing, by dietary or medicinal means, . . . [the] anti-bacterial power of saliva?"

Methods of determining resistance or susceptibility to caries, by estimations of the number or activity of oral bacteria, have been indicated by *Blayney, Bunting, Fosdick, Hansen, Hatton, Wach.*

3. *Enamel*

a. Not a "vital" tissue. There is general agreement that enamel, after its formation, is not a vital tissue; that the processes in fully formed enamel are physical (diffusion), and chemical (reaction), not metabolic (enzymic construction, repair, elimination); and that enamel is unable to resist the convergent actions of acidogenic oral-bacteria. *Bodecker, Bregstein,* and *Karlström* present views in disagreement with the prevailing conclusions, but *Karlström* states the reservation that metabolic processes in enamel "are very insignificant in the outer layer"—where caries is initiated. *Bodecker,* in support of his conclusion that enamel has vitality, and is a defensively reactive tissue, alludes to the fact that often, in proximal caries, only one of two contacting surfaces is carious. He ascribes this result to differences in vitality of the enamel at the two contacting surfaces, active defense against caries being inadequate in the enamel of one of the teeth, adequate in that of the other. This outcome is attributed by *Hearman* to local bacterial alkalosis; by *Krasnow* to micro-anatomical or micro-chemical abnormality. The import of the prevailing conclusion is stated briefly and directly by *Weaver,* as follows: "The way in which minute structure may influence liability to caries has not been explained. How can a tooth, however well formed, resist an attack which is apparently primarily chemical; what is there in the teeth which can prevent acid from dissolving the enamel of those teeth with which it remains in contact? Is it conceivable that, as fast as the enamel is decalcified, the vitality or 'resistance' of the tooth effects a recalcification?"

"Enamel cuticle protects enamel from food acids in transit through the mouth, but does not prevent acid (formed by bacteria) in prolonged contact with it from initiating caries" (*Wallace*).

b. Remineralization. Bisnoff, Boyd, Gore, Gysi, Köszeg, Kraus, refer to possibilities of remineralization of decalcified enamel. *Applebaum, Belding, Weaver* dissent.

c. Mottled enamel, despite its imperfect calcification (structure), is not more, and may be less, liable to caries than normal enamel (*Applebaum, Atkins, Dean, McKay, Piperno*).

d. Fluorine (excess of which in drinking water causes mottled enamel) is a normal constituent of teeth (*McClendon*); is reduced in caries (*Brekhus*); may be in caries-resistant equilibrium with magnesium (*Csernyei*); may be necessary for development of caries-resistant teeth (*Cox*). In drinking water, in proportions that do not mottle enamel, fluoride has reduced incidence of caries (*Dean, McKay, Piperno*) and occurrence of oral *L. acidophilus* (*Dean*); stronger aqueous solution, used as a dentifrice, has had similar effects (*Atkins*). In diets given to rats, during periods of enamel formation, fluoride increased resistance to caries in mottled molar enamel (*Cox*); also in molar enamel that had not become mottled (*Hodge*).

4. *Tooth resistance to caries*

After prolonged research *Mellanby* states the following conclusion: "We still have much to learn about the immediate as well as the predisposing causes

[6] [See the summary by *Rettger,* on page 198, and the further related comment in the supplementary analysis on page 264.]

slightly cervical to the contact point, differs consistently from that of enamel slightly lingual or buccal to that point? Do histologists agree that the quality of enamel at the deepest occlusal depression differs consistently from that just slightly distant?" (*O'Brien*).

b. Remineralisation (see also page 249). One author ascribes to "normal saliva" power to "'remineralize' carious teeth and thereby arrest caries" (*Branson*); another refers to salivary "power to remineralize enamel and dentin" (*Turkheim*). "When there is adequate concentration of Ca and PO_4 ions in saliva, remineralization of enamel takes place. . . . Constant, slight, superficial decalcification and remineralization probably occur alternately" (*Gore*).

c. Mottled enamel (see also page 249). "Mottled teeth, which may be more resistant than normal teeth to the *onset* of caries, are structurally weak. Caries once started in mottled teeth extends rapidly" (*Smith*). "Teeth of 50 pupils of [the] older Bauxite group—showing characteristic hypoplasia of moderate and severe types of . . . mottled enamel—disclosed only about half the caries experience of [the] mottled-enamel-free Benton group" (*Dean*). "An endemic-fluorosis area in Northern India showed 100 percent mottling of both deciduous and permanent teeth, and an extremely low incidence of caries. Since mottled enamel is structurally the most defective, these results present strong evidence against the widely accepted belief that the degree of calcification is the all important factor in caries susceptibility or immunity" (*Day*). "Incidence of caries among groups who have mottled enamel, or live in areas where this condition is endemic, was equal to the incidence among those not suffering from mottled enamel, or living in areas where this condition is endemic. A diet high in fluorine-containing food has not been associated with an incidence of caries significantly different from that in areas where the diet does not contain fluorine in appreciable amounts" (*Agnew*).

d. Fluorine (see also page 249). Some observers found that incidence of caries may be diminished by treatment with fluoride (*Bibby, Cheyne, Cox, Dean, McClendon, McClure, Sognnæs*). There is more fluorine in enamel of sound teeth than in that of carious teeth (*Armstrong*). "No significant differences were found for contents of F in the molar teeth of carious and non-carious rats" (*McClure*). "If fluorine is present in optimal amount during the formation of enamel, the subsequent caries-resistance of teeth is greatly increased" (*Cox*). "For the molar teeth of mature rats that received 20 parts per million of fluorine (as sodium fluoride), in water for sixty days, there was no change in the proportion of fluorine in the *dentin*; but in the *enamel* of the same teeth, there was an increase of 36 percent. The extra fluorine in the enamel was evidently absorbed through the outer surface of the tooth, not *via* the dentin" (*Armstrong*). "Addition of F to the diet, in rats, brought about proportional increases in the F contents of erupted molar teeth. This result indicates a systemic-absorption capacity for F in the erupted tooth—dentin and enamel—and demonstrates the possibility of chemical modification of erupted-tooth enamel and dentin, which, there is reason to believe, is more apt to occur through body fluids than through oral surface-adsorption. The deposition of this secondary F in erupted molar-teeth, in rats, was not associated with proportional immunity in induced occlusal caries" (*McClure*). "Fluorine in mottled tooth-structure apparently offers little resistance to decalcification, when salivary fluorine is withdrawn. . . . Radioactive fluorine . . . makes its appearance in the oral cavity, along the salivary route, almost immediately after injection into the blood stream" (*Cheyne*). "Sodium fluoride, given by injection, apparently had no influence in inhibiting induced caries in rats" (*McClure*).

"Whether the action of fluoride [in reducing incidence of caries] is inimical to the abnormal biology of the colon, or induces a condition of the colloidal system that is unfavorable to growth of atypical intestinal flora and to multiplication of *L. acidophilus* associated with caries, remains to be determined" (*Anderson, T*). "Salivas of school children, whose drinking water averaged 1.8 p.p.m. F, showed no change in amylolytic action compared with salivas of children using water practically free from fluorine" (*Dean*). *Cheyne* "does not believe that fluorine itself will prove to be the key to natural caries-inhibition, but suggests that work with this element may offer clues to other mechanisms that are physiologically operative."

4. *Tooth resistance to caries*

(*See also page 249*)

"Susceptibility [to caries] is primarily dependent on tooth form and tooth arrangement in the arch, which determine the degree of food retention—which, in turn, supports the growth of acidogenic organisms" (*O'Brien*). This quotation states the general opinion of those who conclude that teeth are inherently defenceless at locations on tooth surfaces where food débris recurrently lodges, ferments, and yields destructive acid. *Strusser* emphasizes the significance of the bilateral (symmetrical) incidence of caries. "When caries occurs in atypical spots, it is usually symmetrical" (*Turkheim*). *Day* questions the assumed relation between content of calcium in teeth and incidence of caries. "Deficiencies in calcification of enamel rods increase the vulnerability of enamel to caries" (*Asper*). "Enamel lamellae are defective calcification areas and cracks, which may lead into the dentin. Folds of hornified enamel-cuticle