

FIG. 11. THE EFFECT OF VITAMIN D ON THE STRUCTURE OF DOGS' TEETH.



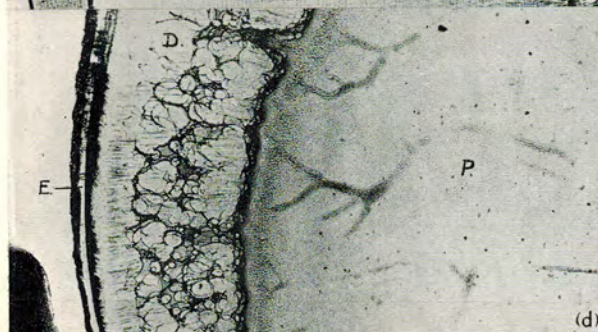
(a) Received cod-liver oil (containing fat-soluble vitamins including vitamin D). White shiny enamel.



(b) Received olive oil (no fat-soluble vitamin). Enamel very defective.



(c) Microscopical structure of a tooth of dog (a). Thick, well-calcified enamel and dentine.



(d) Microscopical structure of a tooth of dog (b). Thin and badly calcified enamel and dentine (many interglobular spaces).

E = Enamel.
D = Dentine.
P = Pulp cavity.

Photographs of the lower jaws and photomicrographs of sections of the carnassials of two puppies (a) and (b) respectively of the same age.

FIG. 12. SURFACE STRUCTURE OF DOG'S ENAMEL EXPERIMENTALLY PRODUCED BY DIET.



(a) Abundant calcifying vitamin D.



(b) Some calcifying vitamin D.



(c) Little calcifying vitamin D.



(d) Very little calcifying vitamin D.

A series of dogs' lower carnassial teeth photographed with an oblique illumination to show grades of surface roughness produced by varying the vitamin D content of their diet.

FIG. 13. THE COMPARATIVE EFFECT OF VARIOUS TYPES OF CEREAL
ON THE STRUCTURE OF THE TEETH.

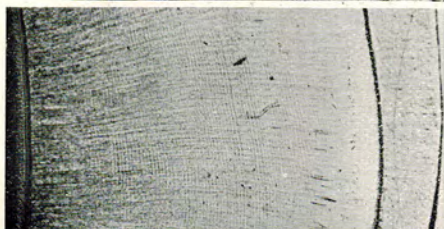


(a) White flour, "commercial wheat germ" and linseed oil.



(b) Oatmeal and linseed oil, changed later to oatmeal and cod-liver oil.

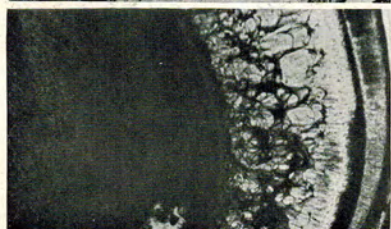
Note poorly calcified dentine followed by dentine of good structure.



(c) Oatmeal and cod-liver oil. Normal enamel and dentine.



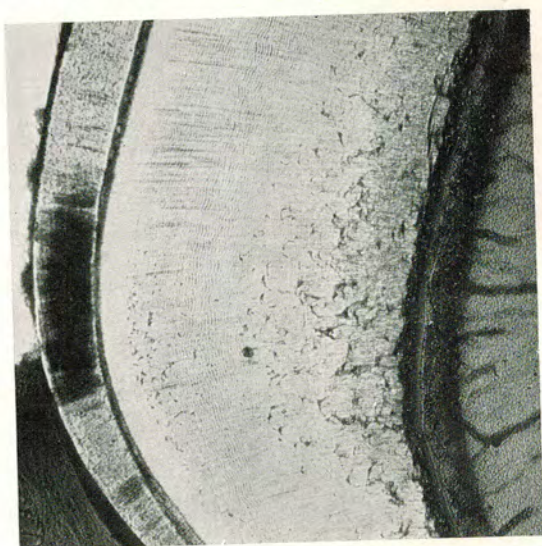
(d) White flour and linseed oil. Somewhat thin and defective enamel and dentine.



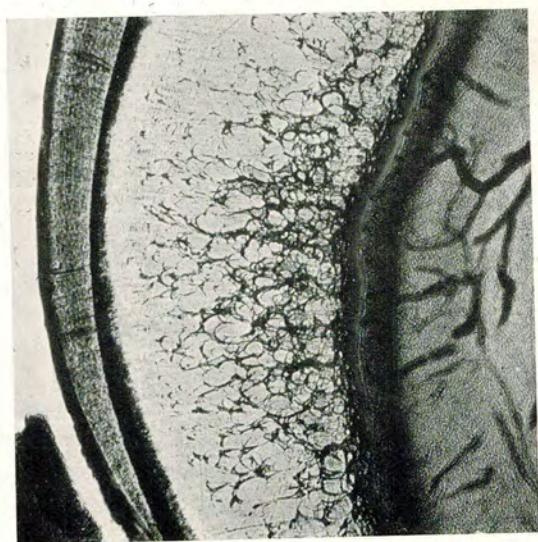
(e) Oatmeal and linseed oil. Very thin and defective enamel and dentine.

Photomicrographs ($\times 32$) of lower carnassials of dogs, to show the comparative effect of various types of cereals on the structure of the teeth when there is but little vitamin D in the basal diet. Animals of same litter and same age when diets begun and when killed.

FIG. 14. THE IMPROVEMENT IN TOOTH STRUCTURE ON THE ADDITION OF CALCIUM CARBONATE TO A VITAMIN D-DEFICIENT DIET.



(a) White flour, wheat germ and olive oil + additional CaCO_3 .



(b) White flour, wheat germ and olive oil but *no* additional CaCO_3 .

Photomicrographs ($\times 40$) of lower carnassials of two dogs to show the effect of additional calcium carbonate when the basal diet contains but little vitamin D. Animals of same litter and age at end of experiment.

FIG. 15. THE IMPROVEMENT IN TOOTH STRUCTURE PRODUCED BY ADDITIONAL CALCIUM AND PHOSPHORUS IN THE PRESENCE OF BUTTER AND OATMEAL.



(a) Butter, calcium carbonate.



(b) Butter, bone ash.

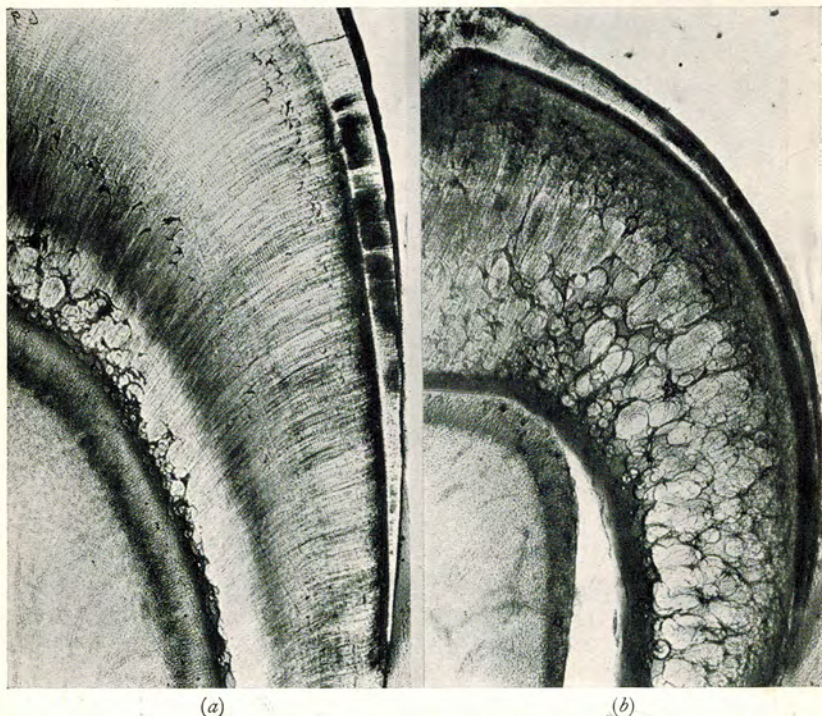


(c) Butter, no extra salt.

Photomicrographs ($\times 40$) of sections of the lower carnassials of two dogs.

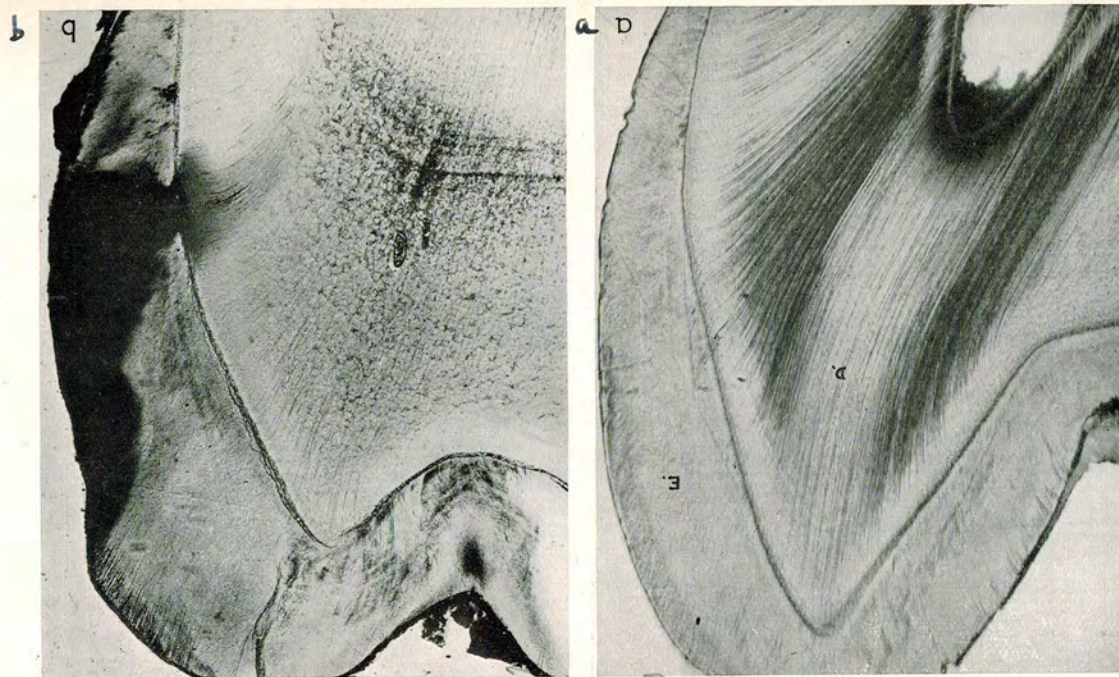
Note the great improvement in calcification of the dentine and enamel in (a) and (b) as the result of adding calcium carbonate and calcium phosphate respectively. Dogs of same litter and same age at end of experiment.

FIG. 16. THE EFFECT OF "GOOD" AND "BAD" MATERNAL FEEDING ON THE
SUBSEQUENT STRUCTURE OF THE PUPPIES' PERMANENT TEETH.



- (a) The offspring of a bitch fed on a diet containing abundant calcifying vitamin D.
 (b) The offspring of a bitch fed on a diet deficient in calcifying vitamin. The structure is much worse in (b) than in (a).
 Photomicrographs ($\times 40$).

After weaning, the two puppies ate exactly the same amount and the same kind of food which contained oatmeal and was deficient in fat-soluble vitamins.



(a) Perfectly formed molar (rarely found). (b) Defectively formed or hypoplastic molar.
 Note defects in enamel and dentine of (b). (A typical specimen as commonly found in this country.)

Photomicrographs of sections of human deciduous molars.

FIG. 18. THE EXTERNAL STRUCTURE OF MOLAR TEETH (CHILDREN).



(a) Surface enamel white and smooth.



(b) Surface enamel slightly rough.



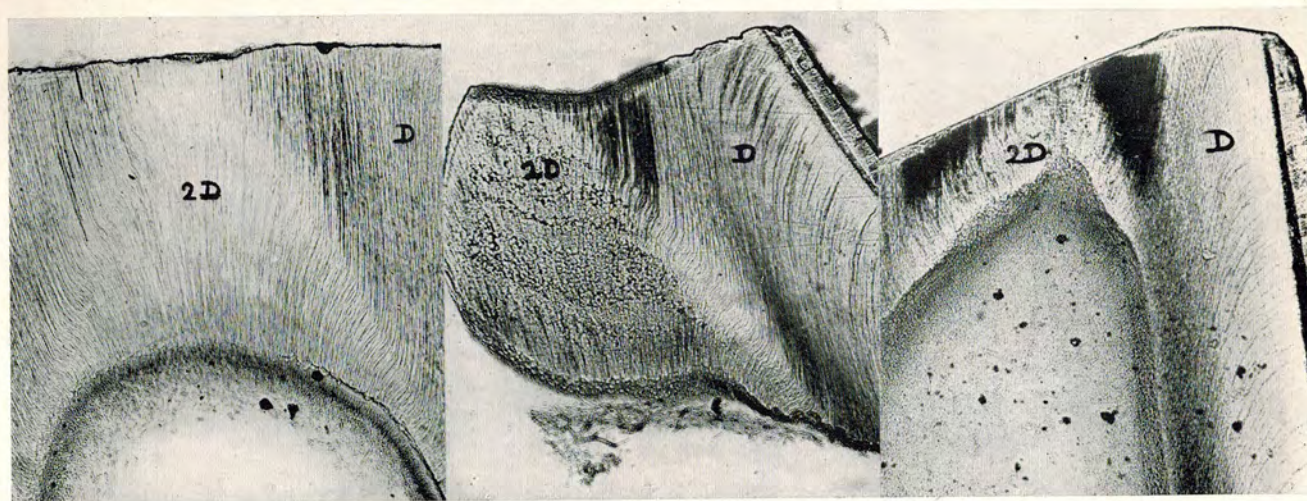
(c) Surface enamel rough.



(d) Surface enamel very rough.

A series of second lower molars photographed with an oblique illumination to show grades of surface roughness and defect. The external condition of the enamel gives a good impression of the internal structure of the teeth.

FIG. 19. SECONDARY DENTINE PRODUCED IN EXPERIMENTAL ANIMALS ON VARYING DIETS IN RESPONSE TO ATTRITION.



(a)

Puppy fed on a highly calcifying diet.

Note much well-calcified secondary dentine.

(b)

Puppy fed on a diet somewhat deficient in vitamin D.

Note much poorly calcified secondary dentine.

(c)

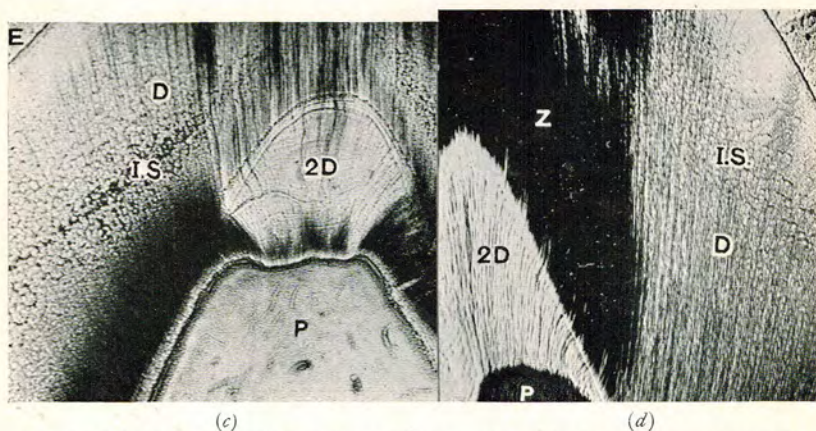
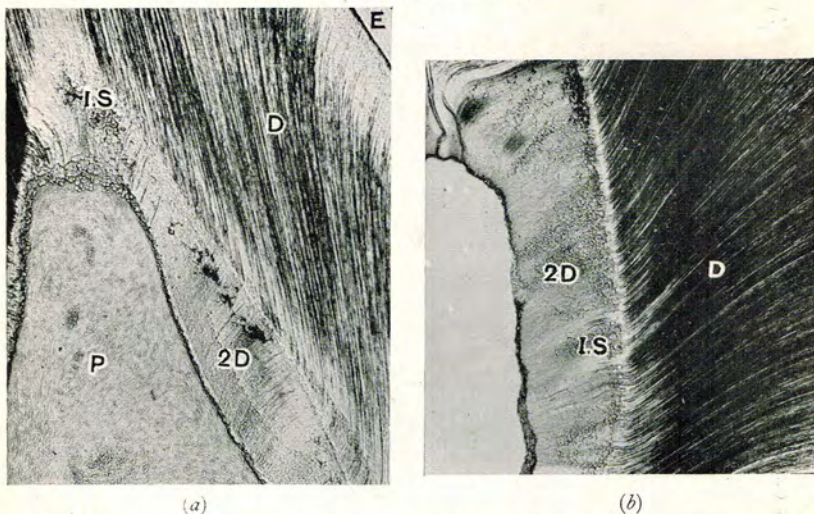
Puppy fed on a vitamin-D deficient diet.

Note very little poorly calcified secondary dentine.

D = Primary Dentine. 2D = Secondary Dentine.

Photomicrographs ($\times 36$) of the deciduous molars of three puppies to show the varying reaction of the teeth to attrition.
(The cusps were gently filed three times a week.)

FIG. 20. DENTINE AND SECONDARY DENTINE OF VARYING STRUCTURE IN CHILDREN'S TEETH.

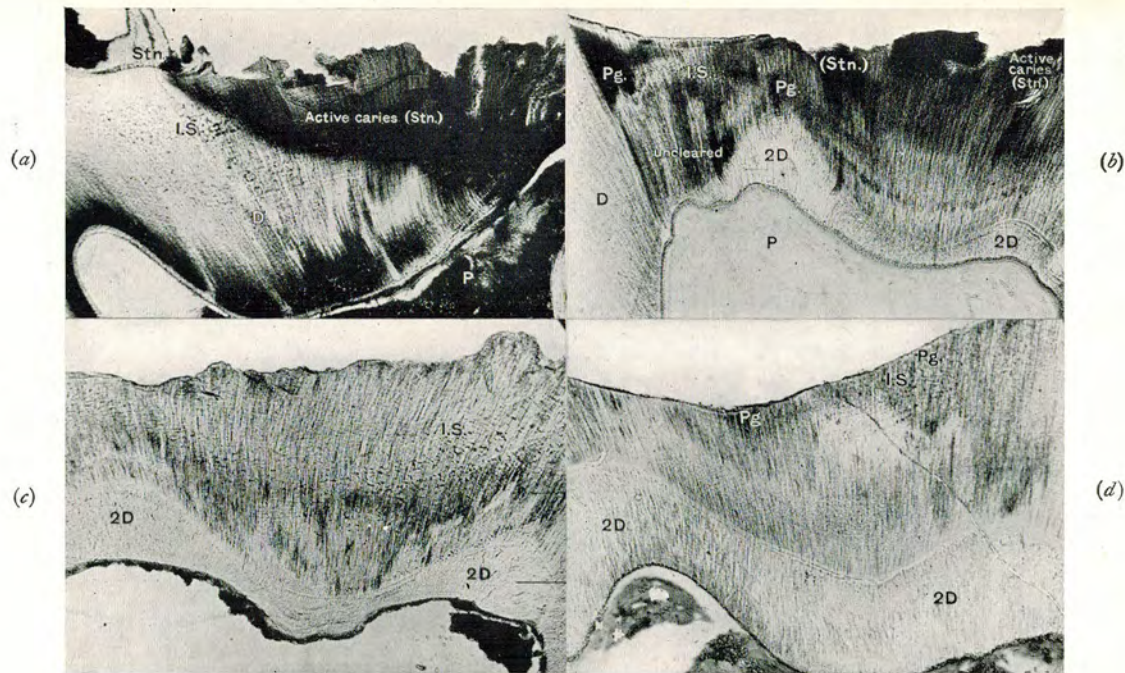


D = Dentine. 2D = Secondary Dentine. I.S. = Interglobular Spaces.

Photomicrographs ($\times 28$) of children's teeth to show types of secondary dentine formed as a protection of the tooth against injury (attrition or caries).

FIG. 21.

"HEALING" OF DENTAL CARIES IN HUMAN TEETH BY DIET.



- (a) Active caries. Much softened dentine. No secondary dentine.
 (b) Six months' treatment with vitamin D. Carious process being arrested. Surface comparatively hard to probe. Note well-calcified secondary dentine.
 (c) Three years' treatment with cod-liver oil. Caries completely arrested. Surface hard and fairly smooth. Note much well-calcified secondary dentine.
 (d) Arrested caries with smooth (and polished) surface. Note large amount of well-calcified secondary dentine.

D = Dentine.

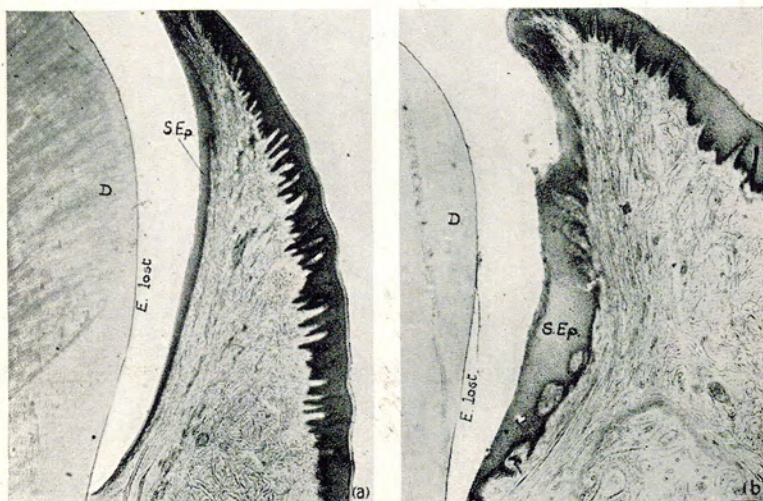
Pg = Pigment.

I.S. = Interglobular Spaces.

C = Active Caries.

2D = Secondary Dentine.

FIG. 22. EFFECT OF VITAMIN A ON THE STRUCTURE OF THE SUB-GINGIVAL EPITHELIUM IN DOGS.

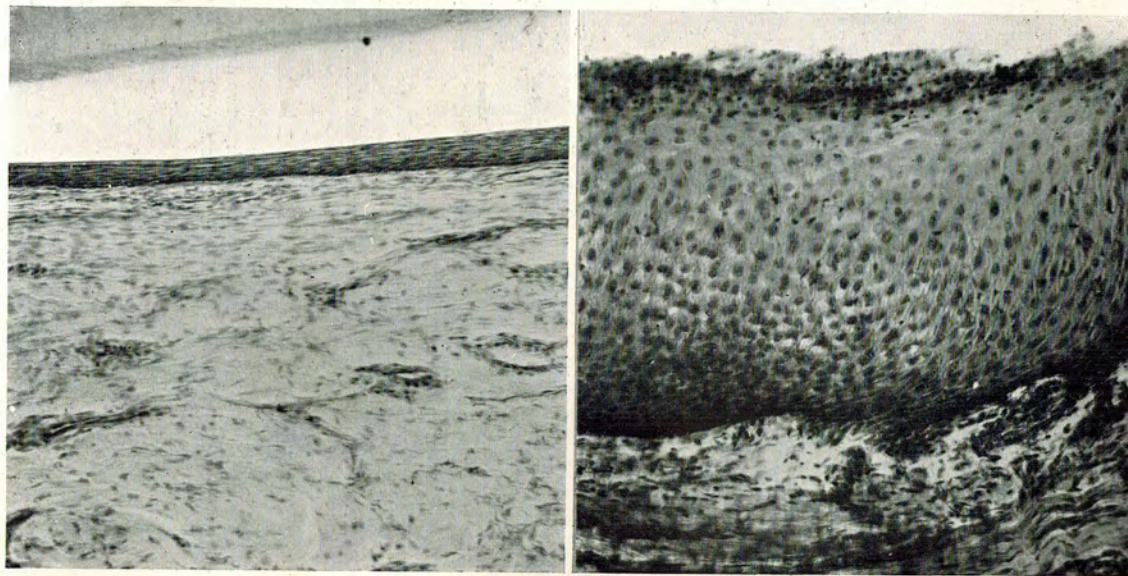


- (a) Diet contained liberal supply of vitamin A but little vitamin D. Gingival region thin. Sub-Gingival epithelium thin and regular. (Alveolar bone poorly calcified : not seen in photomicrograph.)
- (b) Diet contained little vitamin A but liberal supply of vitamin D. Whole Gingival region hypertrophied. Sub-Gingival epithelium hypertrophied and irregular, with small processes. Some cell infiltration into connective tissue. (Alveolar bone well calcified.)

S.E.p. = Sub-Gingival epithelium. D = Dentine. E = Enamel region.

Photomicrographs of the Gingival regions of the teeth of two dogs aged 15½ months. (Sections decalcified ; enamel lost.)

FIG. 23. PREVENTION AND PRODUCTION OF HYPERPLASIA OF THE SUB-GINGIVAL EPITHELIUM BY DIET IN DOGS.



(a)

Photomicrograph ($\times 140$) of the sub-gingival epithelium of a dog on a diet rich in vitamin A.

(b)

Photomicrograph ($\times 140$) of the sub-gingival epithelium of a dog on a diet deficient in vitamin A.

Note the great hyperplasia of the epithelium (E) in (b) as compared with (a). The epithelium of (b) either is, or will be, invaded by micro-organisms to produce periodontal disease.

NUTRITION AND DISEASE

THE INTERACTION OF CLINICAL
AND EXPERIMENTAL WORK

BY

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1934



CHAPTER II

DENTAL STRUCTURE AND DISEASE

IN some ways the example of recent investigations on dental structure and disease illustrates even better the advantages of combined laboratory and clinical study, for the development of the story I am about to relate has remained in the hands of one person—M. Mellanby—from the time of its initiation by her. Whereas the rickets work began as an *ad hoc* investigation, the study of the dental problem began in a chance observation made by M. Mellanby in 1917 on rickety dogs. She noticed that the structure of dogs' teeth was altered by small changes in the diet, and realising its possible significance began one of the most persistent and intensive investigations that this country has ever seen¹⁻¹⁰.

Dietetic Factors responsible for Perfect and Imperfect Teeth

The first stage of the work was to determine the factors responsible for the structure of teeth.¹⁻⁴ Since these, like bones, are largely composed of calcium phosphate the metabolic processes controlling their structure are in many ways similar to those influencing bone architecture. For purposes of study they have an advantage over bones in that, once formed, apart from absorptive processes preceding shedding of the deciduous teeth, their texture does not change

like that of bones, so that a careful microscopic examination of fully developed teeth gives a much truer history than do the bones of the metabolic changes to which they have been subjected during the developmental period. It is now possible to produce at will in animals teeth of all grades of structure—from perfect texture to the greatest degree of imperfection—by making small variations in the food ingested. Thus, if growing puppies are given a limited amount of separated milk together with cereals, lean meat, orange juice, and yeast (*i.e.*, a diet containing sufficient energy value and also sufficient proteins, carbohydrates, vitamins B and C, and salts), defectively formed teeth will result. If some rich source of vitamin D be added, such as cod-liver oil or egg-yolk, the structure of the teeth will be greatly improved, while the addition of oils such as olive or arachis oil leaves the teeth as badly formed as when the basal diet only is given (Fig. 11). Not only are the defects in the dentine and enamel obvious on microscopic examination, but external examination reveals the surface enamel also to be badly formed (Fig. 12). If, when the vitamin D intake is deficient, the cereal part of the diet is increased, or if wheat germ replaces part of the white flour (Fig. 13 (*a*)), or, again, if oatmeal is substituted for white flour (Fig. 13 (*e*)), then the teeth tend to be worse in structure, but if, under these conditions, the calcium intake is increased, then calcification is improved (Fig. 14). Calcium appears both to antagonise the anticalcifying effect of cereals and to aid the action of any vitamin D present in the diet, and this action becomes of great importance if butter is the fat of the diet. Butter in itself has a comparatively

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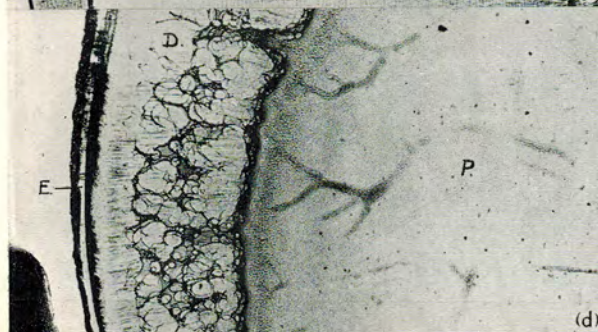
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(c) Microscopical structure of a tooth of dog (a). Thick, well-calcified enamel and dentine.



(d) Microscopical structure of a tooth of dog (b). Thin and badly calcified enamel and dentine (many interglobular spaces).

E = Enamel.
D = Dentine.
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Photographs of the lower jaws and photomicrographs of sections of the carnassials of two puppies (a) and (b) respectively of the same age.

FIG. 12. SURFACE STRUCTURE OF DOG'S ENAMEL EXPERIMENTALLY PRODUCED BY DIET.



(a) Abundant calcifying vitamin D.



(b) Some calcifying vitamin D.



(c) Little calcifying vitamin D.



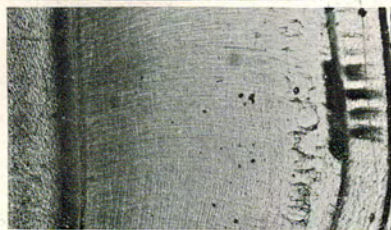
(d) Very little calcifying vitamin D.

A series of dogs' lower carnassial teeth photographed with an oblique illumination to show grades of surface roughness produced by varying the vitamin D content of their diet.

FIG. 13. THE COMPARATIVE EFFECT OF VARIOUS TYPES OF CEREAL
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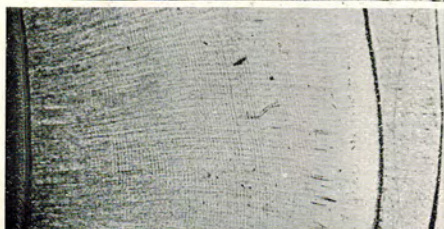


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Note poorly calcified dentine followed by dentine of good structure.



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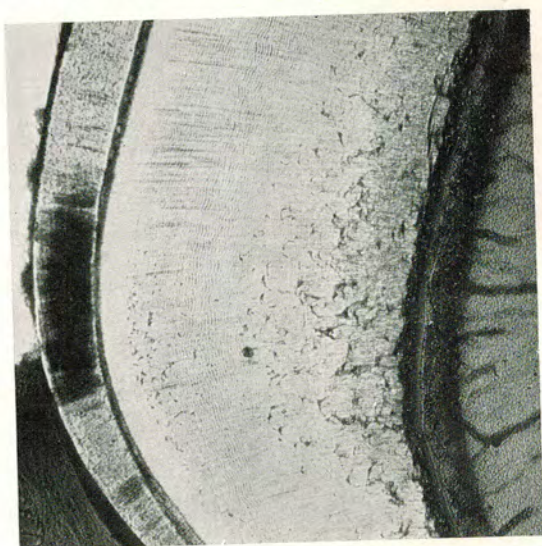
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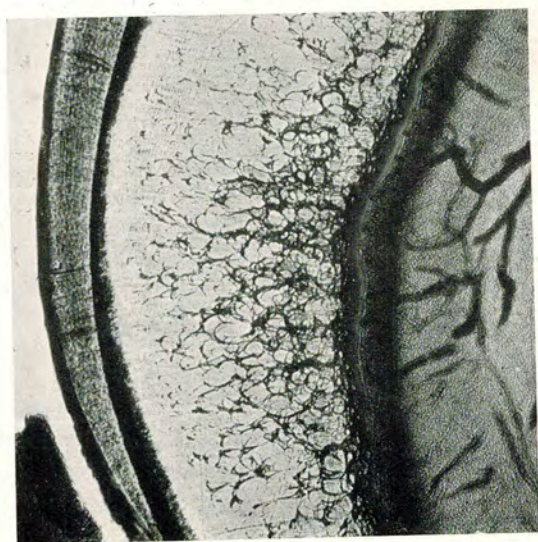
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(a) White flour, wheat germ and olive oil + additional CaCO_3 .



(b) White flour, wheat germ and olive oil but *no* additional CaCO_3 .

Photomicrographs ($\times 40$) of lower carnassials of two dogs to show the effect of additional calcium carbonate when the basal diet contains but little vitamin D. Animals of same litter and age at end of experiment.

FIG. 15. THE IMPROVEMENT IN TOOTH STRUCTURE PRODUCED BY ADDITIONAL CALCIUM AND PHOSPHORUS IN THE PRESENCE OF BUTTER AND OATMEAL.



(a) Butter, calcium carbonate.



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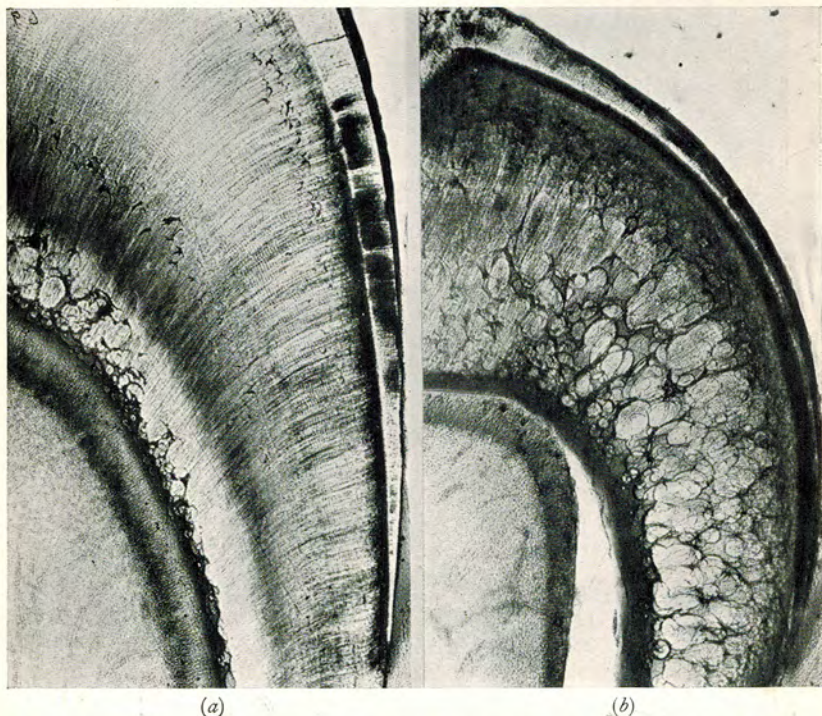


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Photomicrographs ($\times 40$) of sections of the lower carnassials of two dogs.

Note the great improvement in calcification of the dentine and enamel in (a) and (b) as the result of adding calcium carbonate and calcium phosphate respectively. Dogs of same litter and same age at end of experiment.

FIG. 16. THE EFFECT OF "GOOD" AND "BAD" MATERNAL FEEDING ON THE SUBSEQUENT STRUCTURE OF THE PUPPIES' PERMANENT TEETH.



- (a) The offspring of a bitch fed on a diet containing abundant calcifying vitamin D.
 (b) The offspring of a bitch fed on a diet deficient in calcifying vitamin. The structure is much worse in (b) than in (a).
 Photomicrographs ($\times 40$).

After weaning, the two puppies ate exactly the same amount and the same kind of food which contained oatmeal and was deficient in fat-soluble vitamins.

small calcifying influence in the presence of oatmeal, but a corresponding quantity of fat given as milk instead of butter, or an additional amount of calcium with the butter, may result in the development of perfectly calcified teeth (Fig. 15 (a), (b) and (c)).

It is obvious, therefore, that, although vitamin D holds the key-position, dental structure and incidentally bone formation cannot in actual practice be regarded as controlled by only one factor, but that these developmental processes are complicated mechanisms controlled by a series of interactions, some favouring and some antagonising perfect architecture. In addition, it may be stated that, since the earlier stages of calcification are more unstable than the later ones, it is most important to get the beneficial influences to work as early as possible; and this can be accomplished only by suitably feeding the mother during pregnancy and the offspring in its early months of life.

If two comparable bitches are fed, one (A) on a diet of high calcifying qualities, the second (B) on a poor calcifying diet, the teeth of the offspring are affected in two ways :—

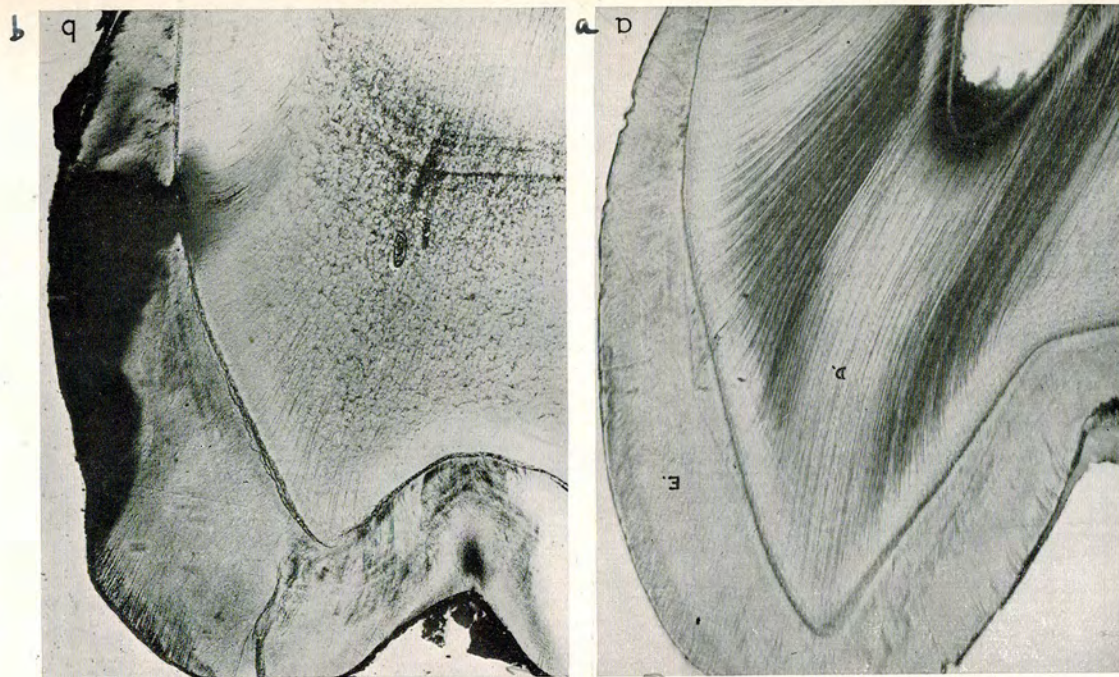
- (1) The actual calcification of the teeth taking place *in utero* of the foetuses of A is better than in the foetuses of B.
- (2) After birth, if the diets of the puppies of A and B respectively are of low calcifying qualities, the teeth of A's offspring—the well-fed mother—stand up more effectively to the bad conditions and are better formed than those of B's offspring. In other words, it would appear that if once the

mechanism of calcification gets a good start as the result of perfect conditions *in utero* it is more difficult to upset by subsequent bad conditions.

The Structure of Human Teeth

The next stage of this investigation was to see what these results meant in terms of human teeth and more particularly in terms of dental caries. It might be thought that the relation of the facts concerning structure and caries could easily have been pursued by further laboratory experiments on animals. But one great obstacle stood in the way of this, namely, that in spite of many attempts it was at the time found impossible to produce dental caries at will under controlled conditions in the animals available. Most of the earlier work on dental caries had therefore to be done on human beings^{4,5}.

It seemed on *a priori* grounds likely that if human teeth were badly formed they would be more susceptible to caries, and since this disease is widespread it would logically follow that many teeth would be badly formed. Against this idea was the generally accepted view among dental authorities that although there is widespread caries, human teeth, especially deciduous teeth, in this country are on the whole well formed. Thus an impasse was present and either the hypothesis was incorrect or the accepted views on dental structure were incorrect. To see whether the structure of teeth was really as good as was stated it was decided first to make a microscopic examination of large numbers of children's teeth. For this purpose, deciduous teeth



(a) Perfectly formed molar (rarely found). (b) Defectively formed or hypoplastic molar.
 Note defects in enamel and dentine of (b). (A typical specimen as commonly found in this country.)

Photomicrographs of sections of human deciduous molars.

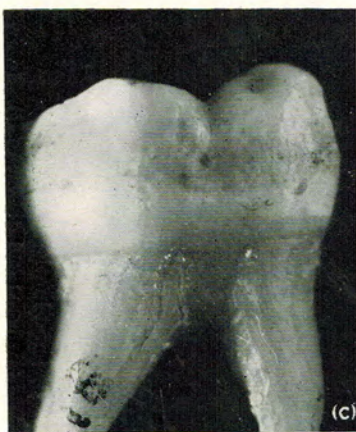
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(a) Surface enamel white and smooth.



(b) Surface enamel slightly rough.



(c) Surface enamel rough.



(d) Surface enamel very rough.

A series of second lower molars photographed with an oblique illumination to show grades of surface roughness and defect. The external condition of the enamel gives a good impression of the internal structure of the teeth.

were collected from various parts of the country and representative of the different classes of society, and after being ground down were classified according to their minute structure. In Fig. 17 photomicrographs of a perfect tooth (*a*) and a hypoplastic tooth (*b*) are seen. The following results were obtained :—

TABLE I.

Structure of the Dentine of British Children's Deciduous Teeth

	Totals.	Grades of Dentine Structure.			
		0 Normal.	1 Slightly Hypo- plastic.	2 Moderately Hypo- plastic.	3 Very Hypo- plastic.
		Per cent.	Per cent.	Per cent.	Per cent.
Incisors .	320	62·2	24·1	9·0	4·7
Canines .	180	20·0	35·6	28·9	15·5
1st molars .	300	9·3	17·7	41·7	31·3
2nd molars .	460	0·9	5·9	26·1	67·1
<i>All Types</i> .	1260	21·2	17·5	25·9	35·4

It is clear from the above table that the teeth of British children are, on the whole, very badly formed and that the previously accepted views on this point are wrong. The incisors which are most influenced by antenatal nutrition are better calcified than the later developed molars. It was also found that, on the whole, the structure of the teeth from private sources was better than that of children attending public elementary schools. There seemed, therefore, some basis for the hypothesis that dental structure and caries might be related.

Having determined the structural defects by microscopic examination, this side of the problem was further developed by relating it to the external appearance of teeth (Fig. 18). Thus, the texture of the enamel as indicated by colour and smoothness was carefully noted in a large series of teeth, and then these same teeth were ground down and subjected to microscopic examination. After much experience it became possible to gauge approximately the minute structure by careful examination of the superficial enamel appearance.

Relation of Dental Structure to Caries

A systematic examination was next made, with the idea of seeing the relation between structure (microscopical and superficial) and caries in human teeth^{2, 5, 8}. The following tables show some of the results obtained.

TABLE II.

Relation of Microscopical Structure to Caries

(All types of teeth taken together.) Deciduous teeth (1500 sections)

Degrees of Caries.	Grades of Dentine Structure.			
	0 Normal.	1 Slightly Hypoplastic.	2 Moderately Hypoplastic.	3 Very Hypoplastic.
	Per cent.	Per cent.	Per cent.	Per cent.
0	77.9	45.7	8.0	6.0
1	8.1	23.8	14.2	4.9
2	6.5	17.2	33.2	15.2
3	7.5	13.3	44.6	73.9

Note.—Some foreign teeth included in this table.

TABLE III.

Relation of Surface Enamel Structure to Caries
(Estimated while Deciduous Teeth still in the Mouth)

Degrees of Caries.	Grades of Dentine Structure.			
	0 Normal.	1 Slightly Hypoplastic.	2 Moderately Hypoplastic.	3 Very Hypoplastic.
	Per cent.	Per cent.	Per cent.	Per cent.
0	94·0	74·0	43·0	18·0
1	5·0	16·0	20·0	13·0
2	1·0	9·0	22·0	17·0
3	0·0	1·0	15·0	52·0

From these tables it is obvious that there is a close correlation between structure and caries whether the determination of tooth structure is made by the microscope or by the texture of the enamel as revealed by a superficial examination: that teeth which are of bad structure tend to be more carious, and those of good structure less carious. This applies to about 90 per cent. of the teeth examined histologically. On the other hand, there is a 10 per cent. disagreement, and this is too large a figure to be regarded as fortuitous. The immediate object of the investigation now was to find the reason for this discrepancy, and back again it came to the laboratory.

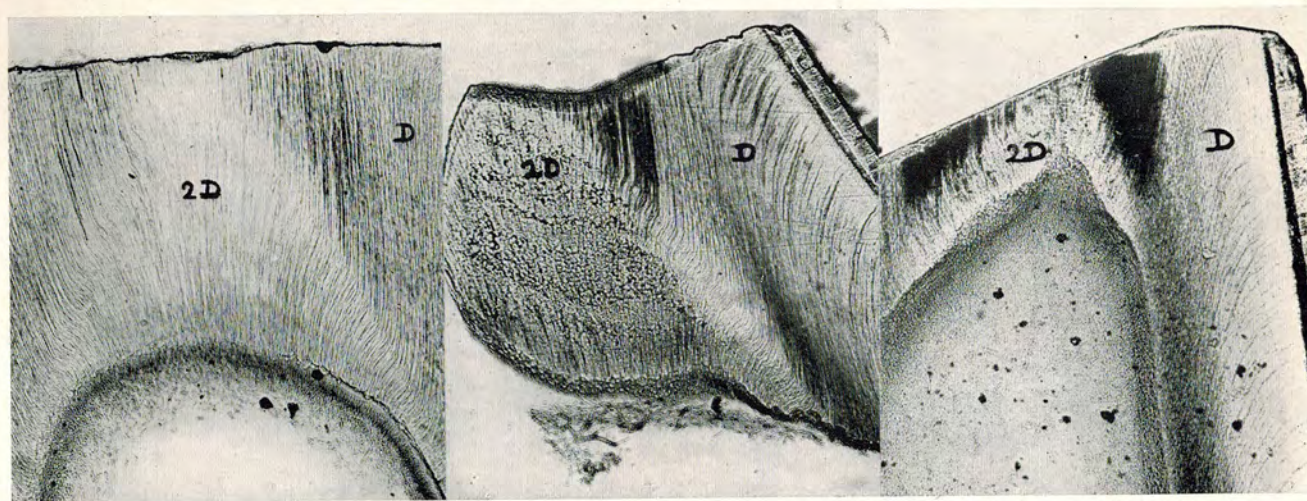
The Defence of Teeth

Since the days of John Hunter it has been known that when the enamel and dentine are injured by attrition or caries, teeth do not remain passive but respond to the injury by producing a reaction of the

odontoblasts in the dental pulp in an area generally corresponding to the damaged tissue and resulting in a laying down of what is known as secondary dentine. In 1922 M. Mellanby proceeded to investigate this phenomenon under varying nutritional conditions and found that she could control the secondary dentine laid down in the teeth of animals as a reaction to attrition both in quality and quantity, independently of the original structure of the tooth (Fig. 19)^{7,8,9}. Thus, when a diet of high calcifying qualities, *i.e.*, one rich in vitamin D, calcium and phosphorus was given to the dogs during the period of attrition, the new secondary dentine laid down was abundant and well formed whether the original structure of the teeth was good or bad (Fig. 19 (*a*)). On the other hand, a diet rich in cereals and poor in vitamin D resulted in the production of secondary dentine either small in amount or poorly calcified, and this happened even if the primary dentine was well formed (Fig. 19 (*b*) and (*c*)).

These results showed that teeth, apart from their primary structure, have a second line of defence against injury, one in fact which is dependent on its nutritional supply. Now caries, like attrition, is a harmful stimulus and human teeth attacked either by attrition or caries respond by the production of secondary dentine varying in character (Fig. 20), just as the dog's teeth make a varied response to attrition according to the nature of the diet. Thus it seemed possible that the 10 per cent. exceptions alluded to above might be explained by this second factor of defence. For instance, a badly-formed human tooth might be free from caries because its defence had been increased by good calcifying factors of a nutritional

FIG. 19. SECONDARY DENTINE PRODUCED IN EXPERIMENTAL ANIMALS ON VARYING DIETS IN RESPONSE TO ATTRITION.



(a)

Puppy fed on a highly calcifying diet.

Note much well-calcified secondary dentine.

(b)

Puppy fed on a diet somewhat deficient in vitamin D.

Note much poorly calcified secondary dentine.

(c)

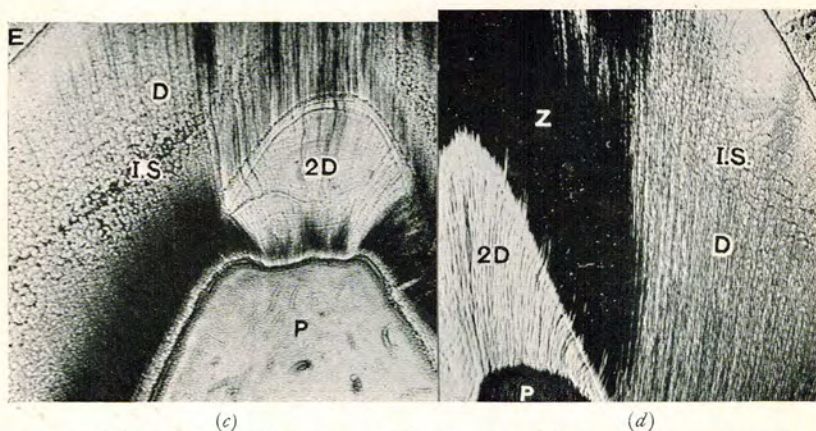
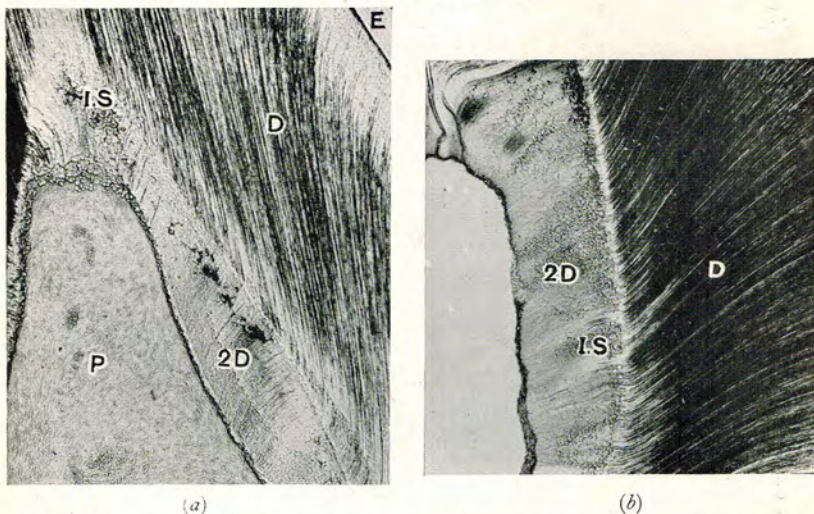
Puppy fed on a vitamin-D deficient diet.

Note very little poorly calcified secondary dentine.

D = Primary Dentine. 2D = Secondary Dentine.

Photomicrographs ($\times 36$) of the deciduous molars of three puppies to show the varying reaction of the teeth to attrition.
(The cusps were gently filed three times a week.)

FIG. 20. DENTINE AND SECONDARY DENTINE OF VARYING STRUCTURE IN CHILDREN'S TEETH.



D = Dentine. 2D = Secondary Dentine. I.S. = Interglobular Spaces.

Photomicrographs ($\times 28$) of children's teeth to show types of secondary dentine formed as a protection of the tooth against injury (attrition or caries).

nature. If so, it would produce in response to attrition abundant well-formed secondary dentine.

The result of the animal investigations again brought the subject back to the clinical side in order to see whether badly-formed teeth free from caries contained good secondary dentine and well-formed human teeth which were carious had deficient or badly-formed secondary dentine. This proved to be the case, so that ultimately the presence of caries in any tooth could be explained on the basis of (*a*) the original structure of the tooth, or (*b*) the reaction to injury as disclosed by the condition of the secondary dentine.

It will be obvious that a fact of even greater import could be deduced from these laboratory observations on the condition of secondary dentine for, if true, they foretold the possibility of directly controlling the onset of dental caries in human teeth, independently of their original structure. If, for instance, a child had badly-formed teeth, very susceptible to caries, a diet of high calcifying qualities ought to diminish the susceptibility to the disease. On the basis of this implication M. Mellanby, with the co-operation of C. Lee Pattison, M.B., and J. W. Proud, L.D.S., began in 1921 a series of clinical investigations on children to whom diets of varying calcifying qualities were given, and the rate of development of caries was tabulated. Four of these investigations were made in Sheffield, and were published in 1924, 1926, 1928 and 1932.¹⁰ In 1928, also, a large-scale test was begun under the auspices of the Medical Research Council at Birmingham,¹¹ with the assistance of Mr A. Deverall, L.D.S., and Miss M. Reynolds. A summary of the findings is given in Table IV.

TABLE IV.

Synopsis of Results of Tests made in Sheffield and Birmingham to see whether Dental Caries in Children can be influenced by Diet.

	Percentage of Teeth Carious (Deciduous and Permanent).			Percentage Increase in "Degree" of Caries (A.C.F.).
	First Inspection.	Final Inspection.	Increase.	
<i>Sheffield—</i>				
Olive oil group .	25·96	35·96	10·00	62·13
Cod-liver oil group	42·86	44·41	1·55	10·28
Vitamin D group .	45·13	46·14	1·01	6·57
<i>Birmingham—</i>				
Olive oil group .	15·59	23·22	7·63	45·85
Cod-liver oil group	19·26	22·23	2·97	9·81
Vitamin D group .	22·18	24·27	2·09	10·12

These results show that the incidence of dental caries can be greatly reduced by feeding children on diets rich in vitamin D. Since, however, the animal experiments had shown that there was another side to the problem of teeth calcification and reaction, namely, that cereals interfere with the processes, it seemed likely that, if a diet could be made not only rich in vitamin D but at the same time be deprived of cereals, the teeth of children eating such a diet might offer still greater resistance to caries. In order to test this possibility, 22 children of average age of 5·4 years were placed on a cereal-free diet for 26 weeks (M. Mellanby and C. L. Pattison). The teeth were charted and the number and extent of all carious points noted at the beginning and end of the feeding period, so that any increase in incidence or spread could be determined. Note was also taken of

the texture or "hardness" of the carious area at the beginning and end of the feeding period, so as to get some idea of the degree of arrest of the carious process.

The results are given in Table V. (Diet 8), and, with them, corresponding results in other groups of children: (1) where vitamin D only had been added to the diet, and (2) where no extra fat-soluble vitamins had been given, but some oatmeal had been included in the diet.

TABLE V.

The Effect of a Diet rich in Vitamin D and devoid of Cereals on the Incidence and Spread of Dental Caries in Children under Six Years of Age.

	<i>Diet 8.</i> Cereal-free Rich in Vitamin D.	<i>Diet 7.</i> Rich in Vitamin D. Contained Cereals.	<i>Diet 4.</i> No extra Vitamin D. Contained Oatmeal.
Number of children in group	22	21	19
Average age of teeth per child showing initiation or spread of caries . . .	0.37	1.0	5.0
Average number of teeth per child in which caries showed hardening . . .	4.7	3.9	0.2

It will be seen from these figures—

- (1) that the addition of vitamin D greatly diminished the spread of caries and caused increased arrest of this process (Diet 7).
- (2) that the removal of cereals together with the addition of vitamin D virtually suppressed all dental caries and increased the healing process (Diet 8).

Although Diet 8 contained no bread, porridge or other cereals, it included a moderate amount of carbohydrates, for plenty of milk, jam, sugar, potatoes and vegetables were eaten by this group of children. Full details of the constitution of this cereal-free diet can be seen in the publication of M. Mellanby and C. L. Pattison describing this investigation^{10 (d)}. It is of interest to note that these results are in harmony with those of Boyd and Drain,¹² who found that caries in the teeth of children on diabetic diets (devoid of cereal) did not spread or develop.

The hardening of carious areas that takes place in the teeth of children fed on diets of high calcifying value indicates the arrest of the active process and may result in "healing" of the infected area. As might be surmised, this phenomenon is accompanied by a laying down of a thick barrier of well-formed secondary denture. Illustrations of this healing process can be seen in Figs. 21 (*b*), (*c*) and (*d*). Summing up these results it will be clear that the clinical deductions made on the basis of the animal experiments have been justified, and that it is now known how to diminish the spread of caries and even to stop the active carious process in many affected teeth.

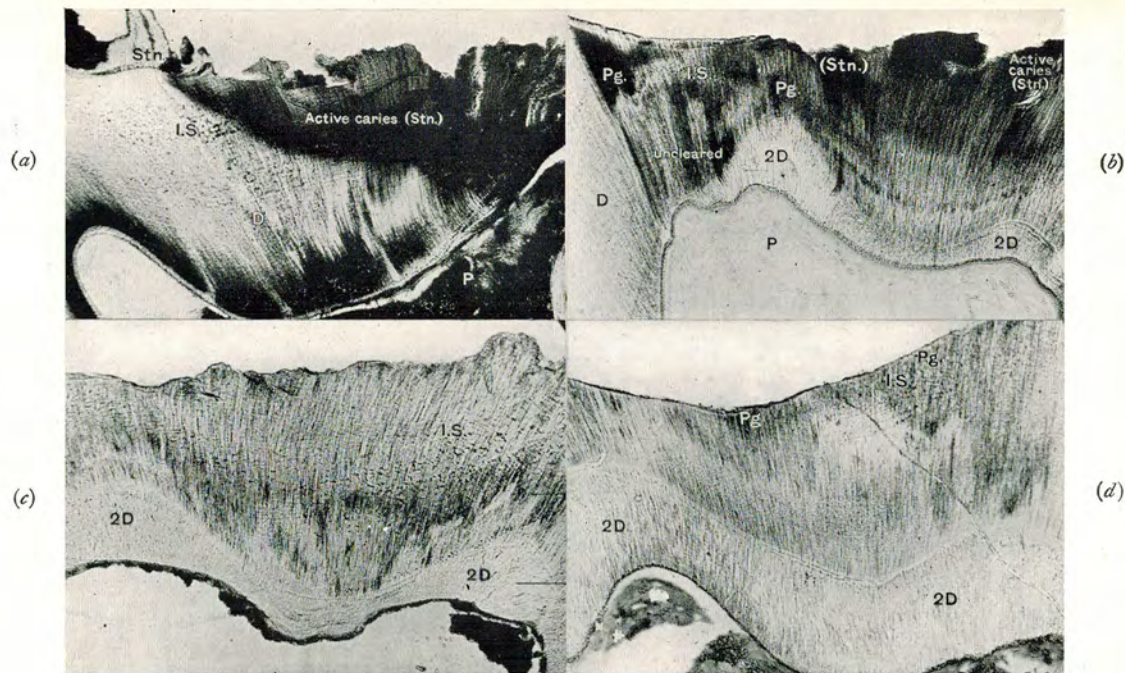
Here, then, we have an example of an investigation in which the laboratory and the clinical work have interacted like battledore and shuttlecock in the following way :—

In the laboratory the dietetic and environmental conditions for the production of perfect and imperfect teeth were found.

On the clinical side, the structure of human teeth was investigated, and was found to be

FIG. 21.

"HEALING" OF DENTAL CARIES IN HUMAN TEETH BY DIET.



- (a) Active caries. Much softened dentine. No secondary dentine.
 (b) Six months' treatment with vitamin D. Carious process being arrested. Surface comparatively hard to probe. Note well-calcified secondary dentine.
 (c) Three years' treatment with cod-liver oil. Caries completely arrested. Surface hard and fairly smooth. Note much well-calcified secondary dentine.
 (d) Arrested caries with smooth (and polished) surface. Note large amount of well-calcified secondary dentine.

D = Dentine.

Pg = Pigment.

I.S. = Interglobular Spaces.

C = Active Caries.

2D = Secondary Dentine.

commonly bad, with defects, on the whole, similar to those produced in the experimental animals. It was then found that, generally speaking, well-formed human teeth are less, and badly-formed teeth more, susceptible to caries. This rule was not without exceptions, and thus the investigation came back to the laboratory to find the reason for these exceptions.

The laboratory investigation led to the discovery that teeth, independently of structure, had a power of resistance to harmful stimuli (attrition) which could be largely controlled by diet.

Applied to the clinical side, this explanation was found to cover most of the exceptions in the relation of structure to caries mentioned above.

Extended further in the clinical aspect, it was shown that the resistance of children's teeth to caries could be directly controlled independently of original structure by feeding on special diets after full eruption of the teeth. It was, moreover, shown that active carious areas could often be "arrested," with the production of much secondary dentine, by giving the children diets of very high calcifying activity, *i.e.*, rich in vitamin D, calcium and phosphorus. If cereals were also eliminated from the diet, caries was virtually stopped.

The Ætiology of Periodontal Disease (Pyorrhœa)

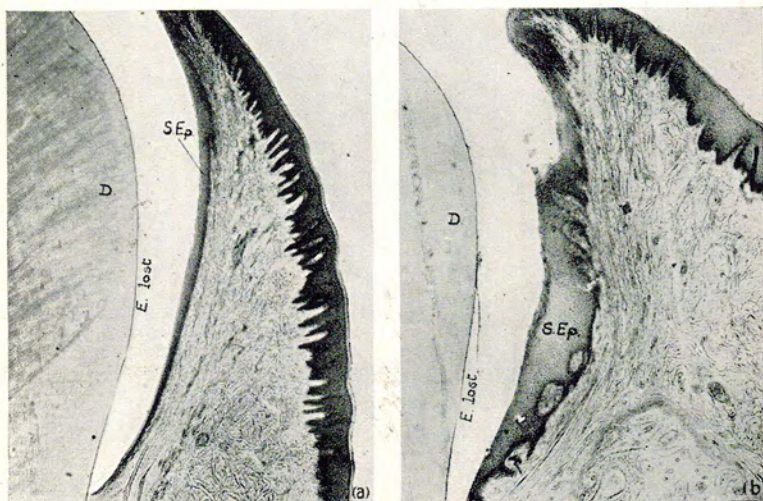
Before leaving this subject of the teeth, I should like to refer briefly to another aspect of dental disease,

namely, periodontal disease (pyorrhœa). Unfortunately, from the point of view of the present discourse, the work has not yet been extended to pyorrhœa in man, but the experimental results obtained in animals⁹ are so clear and, at the same time, so important that they may well be mentioned, especially as they have some bearing on the question of nutrition and infection discussed in Chapter IV.

M. Mellanby has shown that whereas vitamin D is mainly responsible for the calcifying process in teeth and alveolar bone, vitamin A controls the development of the gingival epithelium. If, for instance, young dogs are fed on diets deficient in vitamin A, the stratified epithelium becomes hyperplastic and overgrown. This can be seen clearly in Figs. 22 and 23, where the normal gingival epithelium of a dog fed on a diet rich in vitamin A (*a*) is compared with the hypertrophied epithelium of a dog on a vitamin-A-deficient diet (*b*). When epithelium is once laid down in this hypertrophied fashion, it tends to become infected with micro-organisms. This process seems almost inevitable for, after a period of defective feeding, a good diet very rich in vitamin A may not prevent infection. On the other hand, if the epithelium in the growing animal is well formed, a defective diet later in life is not so likely to allow the infective process to develop. It is, therefore, most important in the case of the gingival epithelium, just as in that of the teeth themselves, to see that dietary conditions during growth produce perfectly formed tissues.

It is probable that cereals also play a part in inducing the defective formation of the epithelium and the tendency to pyorrhœa, just as they do in the case of dental structure and caries.

FIG. 22. EFFECT OF VITAMIN A ON THE STRUCTURE OF THE SUB-GINGIVAL EPITHELIUM IN DOGS.

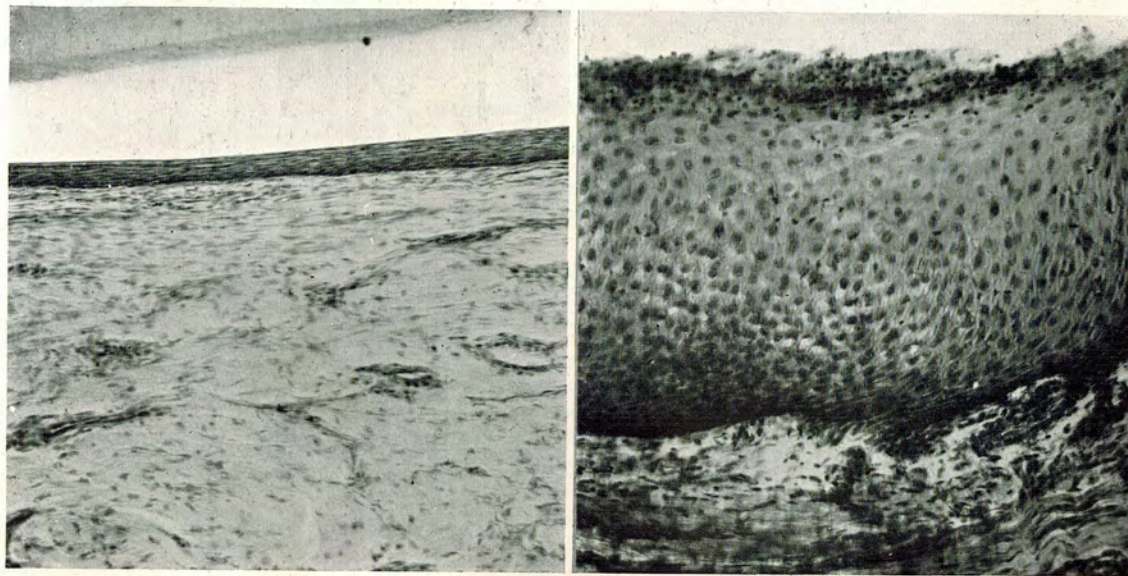


- (a) Diet contained liberal supply of vitamin A but little vitamin D. Gingival region thin. Sub-Gingival epithelium thin and regular. (Alveolar bone poorly calcified : not seen in photomicrograph.)
- (b) Diet contained little vitamin A but liberal supply of vitamin D. Whole Gingival region hypertrophied. Sub-Gingival epithelium hypertrophied and irregular, with small processes. Some cell infiltration into connective tissue. (Alveolar bone well calcified.)

S.E.p. = Sub-Gingival epithelium. D = Dentine. E = Enamel region.

Photomicrographs of the Gingival regions of the teeth of two dogs aged 15½ months. (Sections decalcified ; enamel lost.)

FIG. 23. PREVENTION AND PRODUCTION OF HYPERPLASIA OF THE SUB-GINGIVAL EPITHELIUM BY DIET IN DOGS.



(a)

Photomicrograph ($\times 140$) of the sub-gingival epithelium of a dog on a diet rich in vitamin A.

(b)

Photomicrograph ($\times 140$) of the sub-gingival epithelium of a dog on a diet deficient in vitamin A.

Note the great hyperplasia of the epithelium (E) in (b) as compared with (a). The epithelium of (b) either is, or will be, invaded by micro-organisms to produce periodontal disease.

FIG. 24. PERIODONTAL DISEASE (PYORRHOEA). EFFECT OF VITAMINS A AND D.



(a) Diet from age of $1\frac{1}{2}$ months included a liberal supply of vitamins A and D in the form of cod-liver oil. Tissues in good condition.



(b) Diet from $1\frac{1}{2}$ months same as for (a) except that it contained comparatively little of vitamins A and D. Advanced periodontal disease (pyorrhoea). Deep pockets containing pus. Much absorption of alveolar bone.

ABS = Absorption. P = Pulp.

Enlarged radiographs of the lower molar region of two dogs of the same family, taken at the age of $5\frac{1}{2}$ years. The influence of vitamins A and D on the development of the periodontal tissues and their resistance to disease is shown.

The prevention and development of pyorrhœa in older animals brought up under controlled dietary conditions can be seen in the radiographs in Fig. 24 (a) and (b).

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