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The Chelation and Proteolysis-Chelation Theories of Dental Caries: Their Origin, Evolution and Philosophy

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A concise explanation of this too-neglected theory by its brilliant originator.

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MILLER'S ACID THEORY. ACCEPTANCE ON FAITH

The Chelation Theory[1] and the Proteolysis-Chelation Theory[2-4] were first reported in 1954 and 1955. To understand how and why these theories originated, and how they differ from one another and from other theories, it is necessary to know what dental investigators were doing, how they were thinking, and how little had been accomplished by 1950. When we began work on caries in 1953, we were surprised to discover how often acid was unequivocally declared to be the cause of tooth decay, as if it were a fact. Numerous statements to that effect are still being made by well-known authorities; they appear in many textbooks and journals; and they emanate even from such agencies as the American Dental Association and the United States Public Health Service.[5-8] But no one has ever proved that acid causes caries.

We were therefore able to quickly understand the unscientific nature of so much research being done at that time. We realized that most investigators had accepted the Acid Theory on faith, and then naively equated tooth decay with simple acid decalcification. Consequently, the real objective of their research was not to determine what caused caries. They were convinced that had already been done by W. D. Miller. Instead, they aimed in one way or another to provide "more proof for Miller's Acid Theory," as they put it. Some of these researchers were literally shocked when we told them they would never achieve their objective if caries resulted from something beside acid. They had never considered any other possibility. Some of them even seemed incapable of

understanding what we were saying.

THE PHILOSOPHY OF "PROOF"

We were also dismayed by how many people did not understand what proof is. They became nonplussed when we informed them that proof is qualitative, not quantitative. If something is proved, it is completely proved; no further proof is necessary. Until it is proved, there is no proof at all. In other words, unless something has been conclusively established, it has not been established at all. For until then, there is always the possibility of error. Proof is therefore absolute. It is all or nothing. There is no such thing as partial proof, or degrees or stages of proof. "More proof" would not be needed to substantiate Miller's Acid Theory if it had ever been proved in the first place.

UNJUSTIFIED ASSUMPTIONS, INADEQUACIES, MISCONCEPTIONS AND FALLACIES

We were also taken aback by the lack of understanding of acid. Almost everybody was playing the game called "Follow the Leader" by equating acid with hydrogen ions, and disregarding the anions and undissociated molecules of acids. True, the more sophisticated investigators did distinguish between hydrogen ion concentration, which they measured as pH, and titratable acidity. However, it seemed that nobody was aware of the fact that the anions and undissociated molecules of certain acids could also decalcify enamel and dentin, just as hydrogen ions do, but by an entirely different mechanism, i.e., the formation of chelates and other complexes.[9] Despite numerous reports on the lack of correlation between decalcifying ability and pH or titratable acidity, researchers then ignored (as many still do) the demineralization resulting from the complexing ability of acid anions and Undissociated acid molecules.

What we therefore saw in 1953 was not an overemphasis or even just a plain ordinary emphasis on acid per se, but a psychological fixation or fetish, so to speak, with the hydrogen ion. Miller's disciples had in effect converted his original Chemico-Parasitic Theory into a virtual Acid Theory, without being aware of how they had restricted and thereby distorted their master's views. We prefer to use the expression Acid Theory rather than Hydrogen Ion Theory because acid as such has become indelibly associated with Miller's concept. The term Acid Theory also more accurately reflects how Miller's followers have overemphasized the "chemical" part of his Chemico-Parasitic Theory and have almost completely disregarded the "parasitic" aspect. They have therefore created and have been working within the framework of what we call an Acid Theory, in contrast to his original Chemico-Parasitic Theory. In other words, those who declare their allegiance to Miller have really not been following his precept.

We were also perplexed, in 1953, by the seemingly endless number of publications on the pH of saliva, dental plaques, and fermenting debris in cavities. Tooth decay is not a disease of saliva, dental plaques, or fermenting debris in cavities. It is a disease of teeth. Nobody has ever determined the pH at the site where and at the time when caries first begins. Until that is done, those who claim that acid causes caries are expressing only wishful thinking and deluding themselves along with others. Since they do not know whether the earliest stage of caries occurs in a microcosmos that is acidic at that time, they do not really know whether hydrogen ions cause caries.

Reports which claim that acids produce apparently typical caries in vitro or in vivo have not provided proof that the action is due to hydrogen ions. Indeed, some of these studies show that

anions and undissociated molecules may be more important than hydrogen ions. Even when an enamel surface is exposed to a solution that is acidic, it is not only hydrogen ions which diffuse into the tooth. Anions and undissociated molecules likewise do so, and can demineralize by complexing calcium beneath the surface of the tooth where the first observable loss of minerals is supposed to take place. No one knows what the pH is when and where this earliest decalcification begins. Therefore, no one really knows whether this loss of minerals is due to hydrogen ions.

The disciples of Miller have also overlooked important implications of the fact that they are dealing with a two-phase system where reactions occur at a solidliquid interface.[10] They naively assume that the pH at the plaque-enamel interface is the same as the pH within the liquid phase of the plaque. However, numerous studies of clay, soils, and biogeochemical weathering have shown that this is usually not true for such systems. The pH within the bulk of a plaque or within fermenting debris of a cavity may therefore be quite different from the pH at the plaque-enamel interface of an intact tooth, or the debrisenamel or debris-dentin interfaces in a cavity. The pH within intact enamel and within intact dentin may be still another matter.

A serious criticism may also be directed at the belief, widely accepted but unproved, that the earliest observable loss of minerals is the earliest stage in the caries process. Decalcification, regardless of how early it is detected, can be the resuit of previous effects (some of which may involve organic components) that made possible the subsequent loss of mineral matter. Also open to serious question it the belief that the initial attack in caries must be on mineral components because the organic matrix appears to be intact after minerals have been removed. One cannot justifiably conclude that the organic matrix has not been altered prior to, during, or as a result of decalcification merely because it appears normal when viewed microscopically. The appearance of the organic matrix would not be altered if only its coordinate covalent linkages to mineral components were ruptured. Nevertheless, this kind of change on a molecular level could have most drastic implications with respect to tooth structure, susceptibility versus resistance to caries, loss of minerals, etc. In any event, Miller's disciples have disregarded the considerable and convincing evidence that proteolysis may well precede the loss of minerals in the caries process.[11, 12]

HOW ACIDS AND CHELATING AGENTS DISSOLVE TOOTH MINERALS

It is necessary to know how Miller's disciples think, because their thinking reveals why they are inevitably doomed to failure in their efforts to provide proof (or "more proof") for their Acid Theory. Tooth minerals normally dissolve to a small but finite esxtent in aqueous media. To understand how hydrogen ions and chelating agents dissolve tooth minerals, let us, for simplicity, consider $Ca_3(P0_4)_2$ in distilled water. This salt normally dissolves to a limited extent to form a dilute but saturated solution containing calcium ions and phosphate ions:

A fixed amount of the compound will dissolve under given conditions. This amount is defined by the solubility product K_{sp}:

$$Ca_3(PO_4)_2 \rightleftharpoons 3\ Ca^{++} + 2\ (PO_4^{\equiv})$$

When an acid such as HCl is added, the hydrogen ions from the acid reduce the concentration of phosphate ions by converting them to $HPO_4^{=}$, H_2P0^{-}, and H_3P0_4. This in turn lowers the K_{sp}. More $Ca_3(P0_4)_2$ will then dissolve until the K_{sp} again equals that value which is constant for the given conditions.

$$K_{sp} = (Ca^{++})^3 (PO_4^{\equiv})^2$$

A chelating agent, on the other hand, reduces the K_{sp} by removing calcium ions from solution. It does this by tying them up in the form of undissociated complexes. More $Ca_3(P0_4)_2$ will then dissolve until the K_{sp} again reaches its fixed value. Of course, some substances dissolve $Ca_3(P0_4)_2$ by acting both ways; they may chelate calcium and they may also provide hydrogen ions. Miller's disciples have been concerned only with hydrogen ions. They can therefore never account for caries under weakly acidic or non-acid conditions. Nor can they explain the lack of correlation between cariogenicity and pH or titratable acidity.

WHAT WE CONCLUDED IN 1953

It thus became clear to us in 1953 that the concept of pH, whose general importance in science cannot be overestimated, had oversimplified the pathology of caries in the minds of most researchers. It became so easy and so fashionable to measure pH that dental researchers became infatuated with the technique. They made endless pH measurements and discovered and rediscovered, in terms of pH, that fermenting carbohydrates give rise to acids. With such experiments repeated ad infinitum, they convinced themselves that acid causes caries. The pH concept may therefore be more responsible than anything else for the lack of progress in our understanding the etiology of this disease and our efforts to control it. What we therefore concluded in 1953, when we began working on tooth decay, was that a century of caries research had been doomed to failure from the very start because it had in effect built a house on sand.

THE "EITHER . . . OR" APPROACH

Although several different theories had been developed to explain caries,[13, 14] philosophically the over-all or prevailing approach rested essentially on an "either . . .or" basis. On the one hand, the Proteolysis Theory explained caries in terms of an enzymatic attack on enamel protein. On the other hand, the Acid Theory claimed that tooth decay was due to decalcification. And this, as we have pointed out, was attributed almost exclusively to the action of hydrogen ions. There are so many unjustified assumptions, inadequacies, misconceptions, and fallacies in this approach that it was difficult for us to understand how and why so many people took it so seriously for so long.[15, 16]

For one thing, the "either . . .or" approach assumed that, in enamel, the protein (or organic matter) existed separately from the minerals; and that the initial attack must therefore be on one or the other. But if the two are joined; that is, chemically united to one another, then the initial reaction might conceivably be a rupture or breaking of the linkage, as shown in Figure 1. This kind of attack would not be directed against protein (i.e., organic matter) *per se* or minerals as such. Nor would it result in the loss of any tooth constituents. Everything would

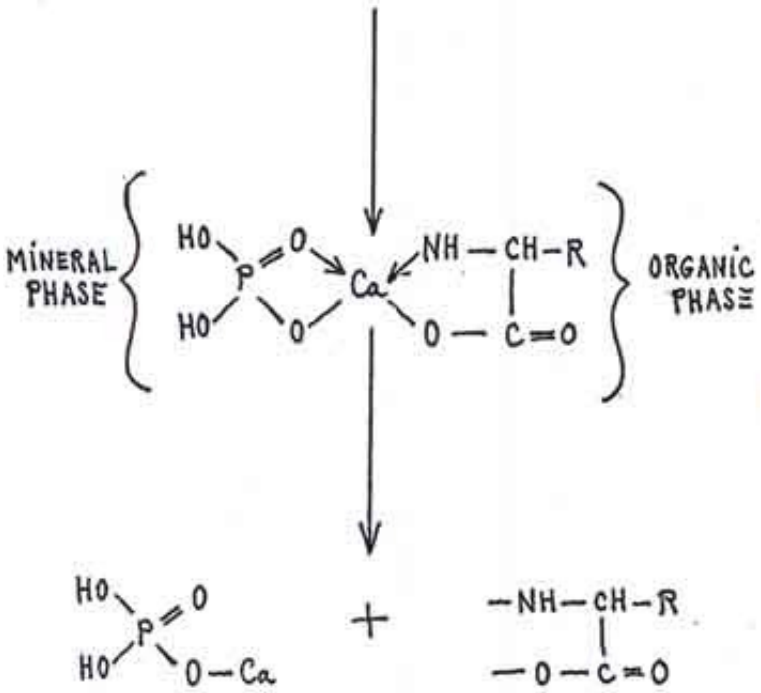

Figure 1. Schematic representation of an initial caries attack which ruptures bonds linking organic and inorganic components of enamel but does not remove any material.

still be there, but the enamel would be structurally altered or disorganized. Another kind of initial attack might result in the simultaneous loss of both organic and inorganic matter.

The "either . . . or" approach did not take such possibilities into account. It considered only a loss of protein by enzymatic degradation or the loss of minerals by acid dissolution. And it chose the latter. The "either . . . or" approach also failed to take into account the ability of acid anions and undissociated acid molecules to dissolve tooth minerals by complexing calcium. Demineralization, as we have indicated, was attributed almost exclusively to hydrogen ions. Moreover, although all acids have hydrogen ions, only those acids originating from the fermentation of carbohydrate food in dental plaques were considered responsible for caries. Thus, the "either . . . or" approach disregarded the possibility that amino acids derived from the breakdown of tooth protein might also decalcify, by chelation.

Despite these and other weaknesses, the "either . . . or" approach dominated caries research for almost a century. Most investigators assumed that there were these two and only these two possible causes of caries; namely, proteolysis and acid. The problem, as they saw it, had been to decide between them. Having chosen acid, they deluded themselves into thinking they had the answer.

THE CHELATION THEORY

What particularly impressed us in 1953 was that hardly anyone was engaged in research on other possible mechanisms beside acid. We quickly realized that caries was unique among major diseases in that so much time, money, and effort continued to be invested in an approach which had been so unprofitable for so long.

We formulated the Chelation Theory of dental caries in 1954 because our early studies revealed that chelation was widely operative throughout nature, and was the most important mechanism for

the dissolution, transportation, and utilization of minerals, both *in vitro* and *in vivo*. Our alkaline blood maintains calcium in solution by means of chelation. The formation, normal turn-over or replacement, and pathologic demolition of bone involves chelation. This is also the most plausible mechanism whereby primary tooth roots are resorbed. The two most important pigments in the plant and animal kingdoms are chelate complexes. Hemoglobin is an iron chelate, and chlorophyll is a magnesium chelate. Enzymes which require trace metals such as zinc, copper, iron, cobalt, and manganese operate as chelate systems. Chelation appears to be the major mechanism in biogeoa chemical weathering, pedogenesis (i.e., soil formation), and soil fertility. The assimilation and transport of mineral nutrients by plants is accomplished by chelation. In the alkaline ocean, from which our alkaline bodies have evolved, the cycle of mineral elements is mediated and made possible by means of chelation.

From a comparative biochemical point of view, it was inconceivable to us that a mechanism so important and so widely operative throughout nature would not

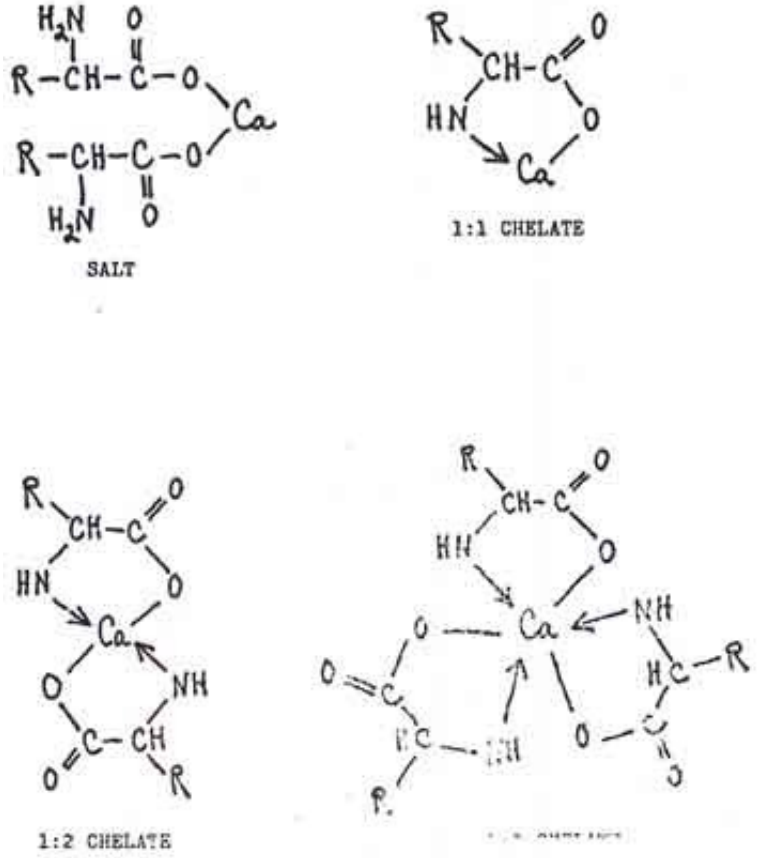

Figure 2. Salt and chelate structures for calcium-amino acid compounds. Three complexes with calcium: amino acid ratios of 1:1, 1:2, and 1:3 are shown.

also be involved as a means of decalcification in the caries process. So, on the basis of these considerations and our experimental results we formulated the Chelation Theory[1] in 1954. According to this concept, tooth decay results from demineralization by chelating agents which dissolve enamel minerals by forming complexes. Figure 2 illustrates the structures of some of these complexes and also shows a salt structure for comparison. An amino acid is used here as an example of a chelator. The Chelation Theory took into account a wide variety of sequestering agents which include acidic, neutral, and alkaline compounds that form calcium chelates under acid and non-acid conditions. Because dental researchers did not understand the chemistry of these coordinate covalent compounds, we discussed this subject in detail[9] in a report in 1958. It is

interesting to note, in this respect, that Eggers-Lura's so-called non-acid complexing theory[17] is no more than a special case of our Chelation Theory.[1] He attributed decalcification to the ability of sucrose to chelate calcium. We had previously considered this in our first report[1] published in 1954.

Our original report on the Chelation Theory appeared in Spain[1] because editors in the United States rejected this manuscript and others which we submitted for publication in their journals. The reasons they gave for refusing to print our papers were:

(a) It had been proved that acid causes tooth decay.

(b) Tooth minerals can be dissolved only by acids, not by chelating agents, and certainly not under neutral or alkaline conditions.

(c) Chelation does not occur *in vivo* and cannot be of interest or importance in tooth decay.

To those editors who refused to publish our papers for these reasons, we replied: "Never has so much ignorance been concentrated in so few words!"

Having satisfied ourselves that chelation could be responsible for the loss of minerals in tooth decay, we next sought answers to such questions as the following:

(a) Which compounds are the most important chelating agents in caries?

(b) Where do they come from?

(c) How are they formed?

As we discussed these aspects of the problem among ourselves and with others, we became aware of a flaw in the Chelation Theory. We had explained demineralization in terms of complexation by acid anions and a wide variety of other organic and inorganic compounds capable of sequestering calcium. But the agents we considered were essentially of exogenous origin; that is, they arose from outside the body.

We therefore found ourselves in a dilemma. If caries were due simply to demineralization by exogenous agents, then tooth decay would be nothing more than highly localized erosion. This would be true regardless of whether the decalcify-

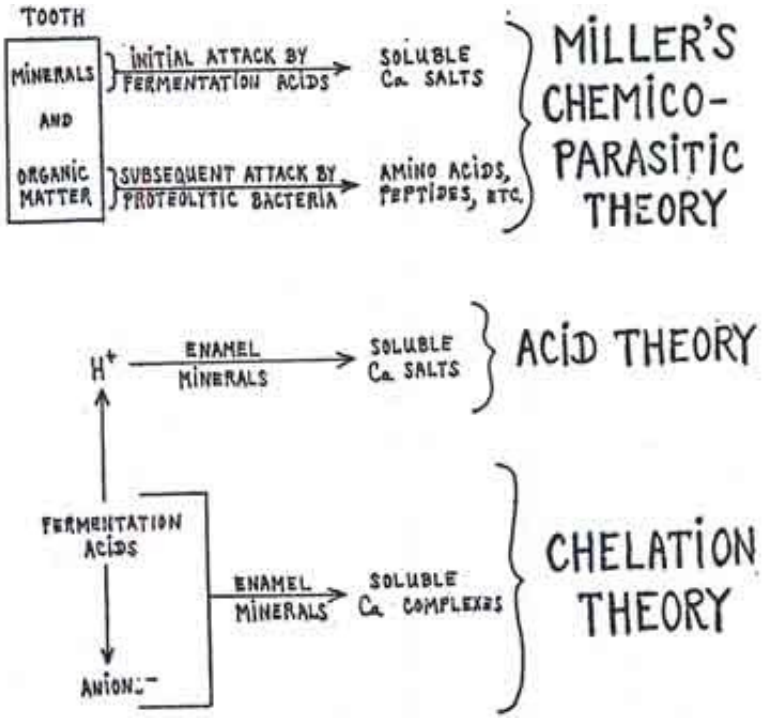

Figure 3. Schematic representation of Miller's Chemico-Parasitic Theory, the Acid Theory, and the Chelation Theory. For reasons which are discussed in the text, we have distinguished between Miller's original Chemico-Parasitic Theory and the Acid Theory which his followers have in effect developed.

ing agents were hydrogen ions or chelating agents. We also encountered another dilemma. If caries resulted from decalcification by compounds produced by bacteria in dental plaques, then it could not possibly be an infectious disease. As we have already pointed out, Miller's disciples overemphasized the strictly "chemical" action of exogenously produced acids and disregarded the "parasitic" aspect of his Chemico-Parasitic Theory. We became concerned when we realized that our Chelation Theory was open to the same criticism. We had considered demineralization only and agents of exogenous origin only. So we too had not viewed caries as an infectious disease.

Bacteria which live in dental plaques or cavities and feed on food that we have eaten are not actually invading our bodies. They are therefore neither parasites nor pathogens. Infectious diseases are caused by organisms which penetrate our bodies. These are parasitic because they utilize parts of our living bodies as their food supply. They are pathogenic because their metabolic activities within our body tissues produce an abnormal or pathologic condition. This is what we call "disease."

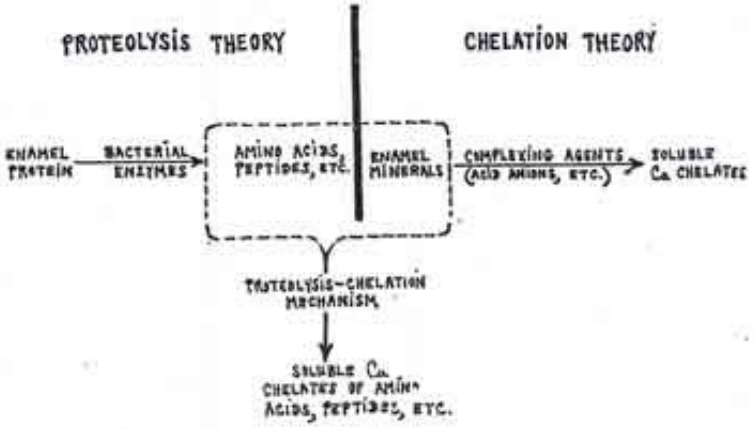

Figure 4. Schematic representation showing the Proteolysis Theory and the Chelation Theory, and how the idea of a proteolysis-chelation mechanism was developed into the Proteolysis-Chelation Theory.

THE PROTEOLYSIS-CHELATION THEORY

It was in an effort to resolve these and other dilemmas that we formulated the Proteolysis-Chelation Theory of dental caries in 1955. What originally suggested proteolysis-chelation as a mechanism for tooth decay was our realization that the Proteolysis Theory provided for the formation by parasitic organisms of those compounds *in vivo* or *in situ* which, according to the Chelation Theory, demineralized by sequestering calcium. We therefore named our new theory Proteolysis-Chelation to indicate its origin. In developing this theory, we demonstrated the susceptibility of organic matter in normally calcified enamel to direct microbial attack. We even showed that keratinous proteins were subject to attack by oral microfloras.[18-21] But we did not restrict our theory to protein or limit it only to keratinous proteins as some of our critics mistakenly assume. Nor did we confine the Proteolysis-Chelation Theory to chelation *per se*. The concept is applicable to and takes into account all organic constituents in the tooth and all complexation reactions, of which chelation is a special case. The term Proteolysis-Chelation was therefore used in a very broad sense.

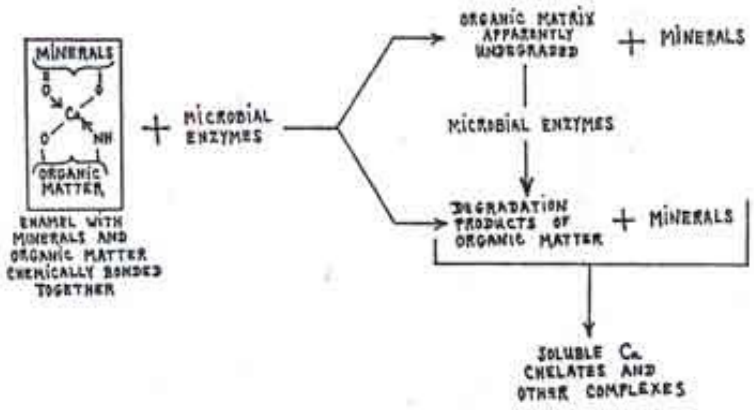

Figure 5. Schematic representation of the Proteolysis-Chelation Theory showing different possible pathways of attack on enamel. The reactions would be essentially the same for dentin.

We also showed that the proteolysis-chelation mechanism was operative over a broad range of acid, neutral, and alkaline conditions. We pointed out that our theory could explain those cases of caries that occurred under non-acid conditions, which the Acid Theory could not. But we never restricted the Proteolysis-Chelation Theory to non-acid conditions, as some of our critics mistakenly assume. We also did not attempt quantitative calculations in terms of the stability constants, as other critics pointed out, because stability constants are not applicable to in vivo systems which are chemically heterogeneous and continuously changing in chemical composition. These constants are derived from and can be applied only to welldefined, homogeneous systems under equilibrium conditions.

The theories of dental caries with which we are concerned in this report are schematically represented in Figures 3, 4, and 5. The Acid Theory and our original Chelation Theory both view caries as a disease resulting from decalcification. In the former, this is brought about by hydrogen ions; in the latter, by complexing agents. The Proteolysis Theory, on the other hand, is concerned

exclusively with enamel protein, the enzymatic breakdown of which is believed to result in caries. The Proteolysis-Chelation Theory differs fundamentally from Miller's original Chemico-Parasitic Theory. According to the latter, caries results from two separate and sequential actions. The first attack on the tooth is a decalcification by fermentation acids of exogenous origin. Then there is a subsequent attack on tooth protein by proteolytic bacteria. The Proteolysis-Chelation Theory, on the other hand, postulates two interrelated actions: an enzymatic attack on organic constituents and a more or less simultaneous demineralization initiated by substances, able to complex calcium, which arise endogenously from the degradation of the organic constituents.

The Proteolysis-Chelation Theory considers the possibility, already mentioned, that the earliest change in tooth decay could be a rupture or breaking of bonds linking organic and mineral components of enamel. This may take place without the loss of anything from either phase. In one sense, such an attack cannot be said to affect either the minerals alone or the organic matter alone, because they do not exist independently of one another at the tme the attack occurs. From another point of view, however, such an attack does affect both components simultaneously since it makes them separate entities, whereas they were previously joined together. Incidentally, this kind of change may be initially responsible for the formation of chalky enamel. Most caries researchers mistakenly assume that the least detectable loss of enamel mineral is the earliest lesion in caries. They do not realize that many changes may have taken place before then. The Proteolysis-Chelation Theory is concerned, among other things, with those biochemical changes or biochemical lesions, so to speak, that have occurred on a molecular level before there is any loss of enamel mineral whatsoever.

From the point of view of what is first removed in the decay process (organic matter or minerals?) the Proteolysis-Chelation Theory considers an initial attack on organic constituents, but with a more or less simultaneous loss of both organic and mineral matter. This can happen because amino acids, peptides, and many other breakdown products of enamel organic matter are capable, as soon as they are formed, of dissolving calcium phosphates by chelation.[22-23] The ProteolysisChelation Theory is not therefore particularly concerned with the question: "What is removed first, organic matter or minerals?" Instead, it challenges the assumption on which that question rests. For such a question may just not be applicable to the earliest stage of caries if neither component is removed, or if both mineral matter and organic material are lost simultaneously.

Although the Proteolysis-Chelation Theory is primarily concerned with how teeth decay, and especially with the initial stages of this disease, it has also tried to explain such aspects as why caries occurs, the biochemical nature of enamel susceptibility versus resistance, the post-eruptive maturation of enamel, the role of nutrition, the influence of systemic factors, unilateral interproximal caries, fluoride action, the effects of trace elements, and preventive measures.[23-28]

WHAT IS ENAMEL? WHAT IS CARIES?

What we did in developing the Proteolysis-Chelation Theory was to reorient our thinking about enamel and about the etiology and prevention of caries. Most researchers look upon enamel as a mineral structure. They know, of course, that a small amount of organic matter is present, but this is residual material left over from amelogenesis. For them, it is only of incidental interest and has little or no importance in the disease process. Since they view enamel as essentially mineral in nature. they believe that caries results from a loss of minerals, and that the initial attack is a

demineralization.

But we look upon enamel quite differently. To us, it is essentially organic, just as all other parts of our bodies, including bone, dentin, and cementum, are fundamentally organic. Ectodermal structures such as nails, hooves, claws, horns, feathers, hair, wool, porcupine quills, scales, etc., are all mechanically modified and adapted for the functions they are required to perform. Teeth have to be very hard, especially their outermost layer. The high degree of mineralization of enamel, which is another ectodermal structure, is the way it has been mechanically modified and adapted for its particular functions. Nature has used the same means to strengthen bone, dentin, and cementurn. Nevertheless, enamel is still *essentially* organic in nature. We therefore consider caries to be an infection or invasion of this organic ectodermal structure by microorganisms capable of feeding on some of its protein, carbohydrate, fat, and other organic components. Because so much inorganic material is present, it is not at all surprising that the disease affects a large amount of minerals in one way or another. Indeed it would be surprising if this were not so.

Since we differ philosophically from Miller's disciples in how we define enamel and caries, our whole approach is fundamentally different from theirs. They concentrated on physical-chemical studies of calcium hydroxyapatites, especially structure and solubility. We, on the other hand, applied information about the biochemistry, physiology, and pathology of skin and other ectodermal structures to enamel in an effort to understand its structure, maturation, and susceptibility to caries; and the role of such factors as age, diet, trace elements, hormonal balance, saliva, etc., in this disease. We paid particular attention to keratinous proteins because ectodermal structures consist largely of these proteins.

WHAT CAN WE LEARN FROM HISTORY

There was a time, not long ago, when the great majority of dental researchers unquestioningly accepted lactobacilli as the organisms which cause tooth decay. Those few individuals who expressed doubts were considered to be uninformed or naive, and were not infrequently persecuted for what was considered heresy. Now, no one seriously believes that lactobacilli cause caries. But no one points out that:

(a) All those authorities who built reputations and achieved positions of power based on that mistaken notion were completely wrong.

(b) The great majority of dental researchers who allowed those authorities to think for them were also wrong in accepting without question, what they were told to believe. But there is a more serious charge against them. They were intolerant of others who had different ideas.

(c) The few so-called heretics who questioned the role of lactobacilli and challenged the authorities were the true scientists. They insisted on thinking for themselves. History has justified and vindicated them.

At that time, not long ago, the great majority of dental researchers looked upon the authorities who had established the "lactobacillus cult" as their leaders. Those men were honored and acclaimed for their supposedly important contribution to understanding and controlling caries. But we now know that those authorities and leaders were completely wrong about lactobacilli. And because they were wrong, and because they were intolerant of others who were right, those acclaimed authorities and honored leaders are directly and personally responsible for having retarded

progress in understanding and controlling this disease.[29-34]

Now streptococci instead of lactobacilli cause caries. Now some of the acid that decalcifies comes from the fermentation of streptococcal polysaccharide, originally formed from dietary sugar. But it is still acid, acid, acid, acid with the great majority of dental researchers who continue to use their pH meters more than their heads. They still concentrate on hydrogen ions and ignore the fact that the anions and undissociated molecules of fermentation acids can dissolve tooth minerals by complexing calcium. This is true regardless of whether these acids derive from sucrose or from polysaccharides. For them to admit this would open a Pandora's Box that would wreak havoc with the *status quo* and lead them inexorably first to the Chelation Theory and then to the Proteolysis-Chelation Theory.

WILL HISTORY REPEAT ITSELF?

We have now reached a stage where history may soon judge those authorities who have built their reputations on the Acid Theory, and have achieved positions of power in an "acid cult." Like their predecessors who established the "lactobacillus cult," these leaders have been intolerant of others who insist on thinking for themselves and who question the role of acid. Like their predecessors, the acid authorities have also been honored and acclaimed for their supposedly great contribution to the etiology and prevention of caries. But they have never proved that acid causes caries. Nor have they prevented this disease by counteracting acid and nothing else. Indeed, there is increasing evidence that acid does not cause caries. More and more people now see the proverbial handwriting on the wall of the temple of acid which the authorities have erected, and they know that it says: "You have been weighed and found wanting."[29-34]

History may decide that those who conjured up acid as a cause of caries may be just as wrong as those who invoked lactobacilli in the etiology of this disease. History may justify and vindicate the few free and indomitable spirits who have insisted on thinking for themselves and have questioned acid as a cariogenic agent. It will then be recognized that those great authorities and renowned leaders who perpetrated and perpetuated the "acid cult" are directly and personally responsible for having retarded progress in understanding and controlling this disease.

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