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The Diet Dilemma in Dentistry

The very fact that the American Society for Preventive Dentistry has deemed it appropriate to sponsor a conference on the subject of diet and dentistry is proof enough that the proper place of diet in stomatology is still unresolved. In practical terms, therefore, there must be two or more camps. On the one hand, there are experts who contend that diet plays a minimal or no significant role in the genesis of oral pathosis; in contrast, there are other authorities who argue that diet is a cardinal factor in oral health and disease.

The position of this report in this controversy will be developed through an analysis of the following **eight** points.

First, the evidence seems abundant that disease is the result of the interplay of environmental challenges and man's ability to withstand these environmental bombardments (Figure 1).[1] Thus, what eventuates on the right as health or disease can be altered by (1) modifying on the left the environment while ignoring host state, (2) changing host conditions on the left with no attention to the environment, or (3) altering both variables on the left side of the equation. It follows that the ecosystem for **oral** health and disease on the right (Figure 1) requires the interplay of host states and the oral environment on the left.[2-7] It is fair to say that, in the case of oral pathosis (which largely takes the form of dental caries and periodontal disease), it is now conceded that the oral challenges eventuate in plaque (Figure 2). Thus, it is generally held in dentistry that plaque (on the left in the upper equation) is the **cause** and oral disease (on the right) is the **effect**. The fact that the formula is not that simple can be readily demonstrated.[8] For example, the correlation between plaque on the left and oral pathosis on the right is not perfect. Some people with less plaque have more oral pathosis than others with more plaque. This alone suggests that other factors may be operative. Also, one must still ask, "What causes plaque?" In other words, what are the host and environmental factors (on the left) which make for plaque on the right in the bottom portion of this illustration (Figure 2)?

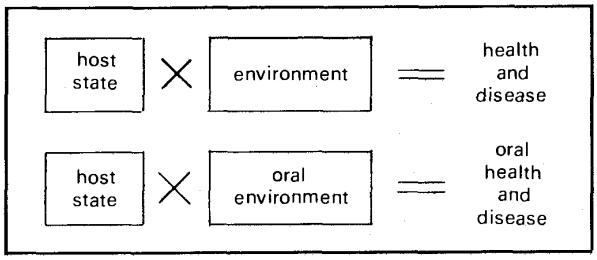

Figure 1 The ecology of health and disease. Whether one remains unscathed or succumbs to illness (on the right) is a function of the many and diverse environmental challenges (on the left) **and** the organism's ability (on the left) to withstand the environmental bombardment.

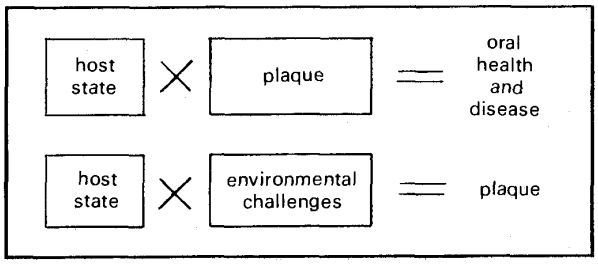

Figure 2 The role of plaque in the genesis of oral health and disease. On the one hand, plaque may be viewed as the cause (on the left) with oral health and disease as the effect (on the right). On the other hand, plaque may be regarded as an effect (on the right in the lower formula) which raises the question as to the environmental and host factors as causative agents (on the left).

The resolution of these questions is not too difficult provided one employs the following experimental model (Figure 3). One can take a group of presumably healthy subjects and scale the teeth on one side of the mouth while supplying a placebo pill to be swallowed. This is represented by the top line. Reexamination of the tissues at a later date will

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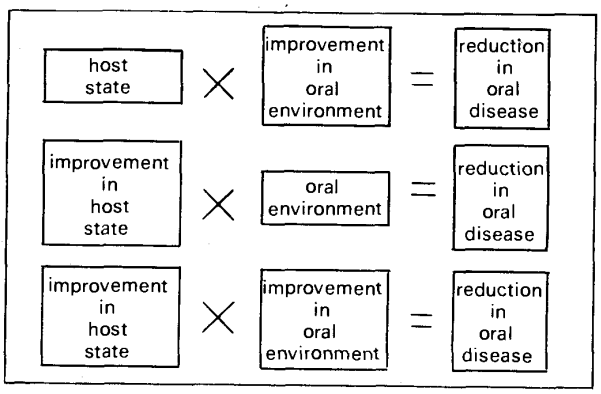

Figure 3 The changes in oral health and disease (on the right) which follow changes singly and in combination with host and environment. The top formula pictures the effect of a change in the environment. The center equation describes the effect of host change. The bottom formula depicts the effect of combined change (host and environment).

provide a measure of the merits of modifying the oral environment versus not changing the oral cavity. Clearly, host state is unchanged by virtue of placebo supplementation. A second group of subjects can likewise be scaled and, in addition, provided with a vitamin or mineral or hormone in a pill indistinguishable from the placebo supplement earlier described. This is the middle line in the illustration. Finally, one can evaluate the relative efficacy of host therapy with and without changes in the oral environment. This is shown by the bottom formula. In short, this type of experiment will provide an analysis of the relative merits of (1) no therapy, (2) only local treatment, (3) only host modification, and (4) local and host therapy. Parenthetic mention should be made that this report will conclude with just such an analysis. **For the moment, however, and with regard to the first point, the position taken is that oral health and disease are multifactorial systems.**

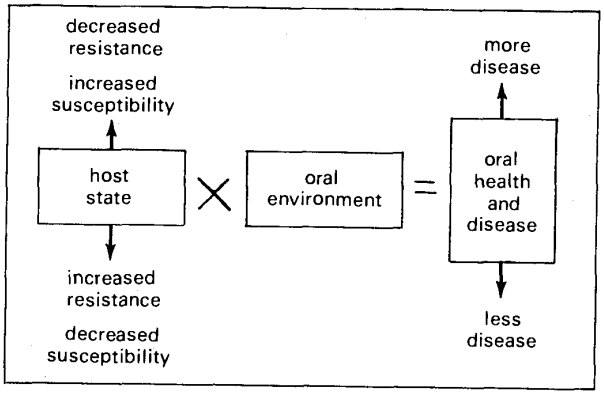

Figure 4 The effects of resistance and susceptibility agents (on the left) upon oral health and disease (on the right). Host improvement follows the addition of a resistance agent, the elimination of a susceptibility agent, or a combination of both. Conversely, host worsening follows the removal of a resistance agent, the addition of a susceptibility factor, or a combination of both.

Second, this report recognizes that what is loosely designated as **host state** actually represents a constellation of factors which invites or discourages one's ability to cope with the environment (Figure 4). These factors may be viewed as **resistance** and **susceptibility** agents.[9] By definition, a resistance agent is one which, when administered, tends to **discourage** disease. Thus, physical activity may be rightly classed as a resistance agent because its addition tends to discourage cardiovascular pathosis. On the other hand, a susceptibility agent is one which, when introduced, tends to **invite** disease. The obvious susceptibility factor is refined carbohydrate foodstuff because it encourages the development of dental caries. Thus, host state consists of a series of resistance factors (which may be viewed as pluses) versus susceptibility factors (which may be expressed as minuses). In simple terms, healthy people possess many pluses and few minuses; sick people display more minuses than pluses. Common sense would dictate that the elimination of susceptibility factors and the addition of more resistance variables would shift the subject to greater and better health. Clearly, certain dietary and nondietary elements have been identified as pluses. Vitamin C discourages scurvy, physical activity encourages cardiovascular fitness, and so on. On the other hand, certain dietary and nondietary variables tend to invite disease and thus must be viewed as minuses. Refined carbohydrate foodstuffs invite dental caries, tobacco encourages lung cancer, and so forth. Perhaps what is most exciting about this concept is that a susceptibility agent in one part of the body does not convert to a resistance factor in another area. Thus, refined carbohydrate foods are deleterious to the teeth. They are also harmful in diabetes mellitus, acne, obesity, and a host of other well-established syndromes. Conversely, physical activity is helpful in discouraging cardiovascular disease. There is some evidence that physical activity invites a reduction in subclinical tooth mobility. **It is, therefore, the position of this report, as judged by this second point, that there must be dietary factors operating in the genesis of oral health and disease just as there are dietary factors operating in the causation of extraoral disease. Some of these factors encourage, while others discourage, pathosis.**

Third, the common sense principle will be employed that recognizes that health and disease are simply not a question of black versus white.[10] Traditional medicine assumes that scurvy, for example, is the **first** and **earliest** evidence of a Vitamin C deficiency state (Figure 5). The fact of the matter is that scurvy is the **terminal** stage of avitaminosis C. Hence, there must be clinical, admittedly subtle, earlier evidence of hypovitaminosis C. Phrased another way, common sense would dictate that man is not either consuming just enough nutrients or none at all. It would seem reasonable, for example, that

some persons are consuming half of the recommended daily Vitamin C intake and so have, if you will, semiscurvy (Figure 5). **Thus, the position taken here, as a third point, is that man displays an infinite number of gradations of nutrient intake, which influences host state as well as the environment, on the left side of the equation and there is then the possibility that this spectrum of nutrition may parallel the spectrum from optimal to ill health as shown on the right side of the formula.**

Fourth, it seems clear that the nutrients, particularly the vitamins, gain respectability only as they become associated with a specific syndrome with a specific designation. Thus, it is recognized that without thiamin, beri-beri will ensue; without niacin there will be pellagra; without Vitamin C, scurvy will follow. In this type of name-calling environment, Vitamin E becomes a vitamin in search of a disease! **The position taken here, with regard to this fourth point, is that nutrients take on more meaning when they are viewed in terms of their role in metabolism rather than their identification with a specific textbook-structured syndrome.** This point will receive more consideration later in this position report.

Fifth, time is a cardinal factor in the development of chronic disease.[11] Not only must there be a problem but it must extend over a critical period of time (Figure 6). This may be likened to racing cars which may travel at different speeds from the moment the race begins. However, the differences in their positions only become evident after several laps. The time factor is critical to an understanding of chronic disease; and, after all, the two major stomatologic problems, dental caries and periodontal pathosis, are of a chronic nature. **Hence, the position here, with regard to this fifth point, will be that small dietary aberrations over a relatively long period of time exert a more deleterious effect than a larger deficit, for instance, over a relatively shorter interval.**

Sixth, actually there is not much argument that diet exerts an effect upon oral health and disease. The fact that refined carbohydrates are cariogenic establishes unequivocally the importance of diet in stomatology. The real question is how well fed are Americans. There are experts who contend that Americans are generally well-fed; there are other equally distinguished authorities who regard the American diet as a disaster. There is no point outlining the evidence here. It is, however, appropriate to underline at this time that there is a simple model for resolving this question (Figure 7). All one need do is supply, under double-blind conditions, a group of presumably healthy Americans who eat well with supplement versus placebo preparations. If the subjects show clinical improvement with the supplement and not with the placebo, then there is some

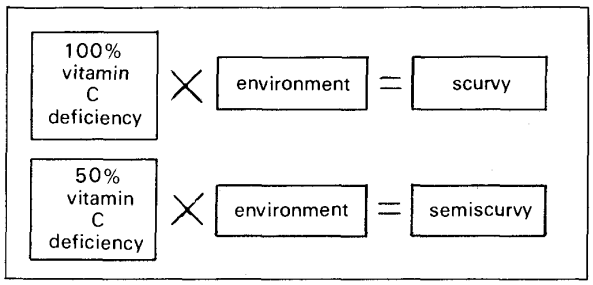

Figure 5 The ecology of the spectrum of vitamin C deficiency emphasizing the infinite number of gradations between optimal health and total disease.

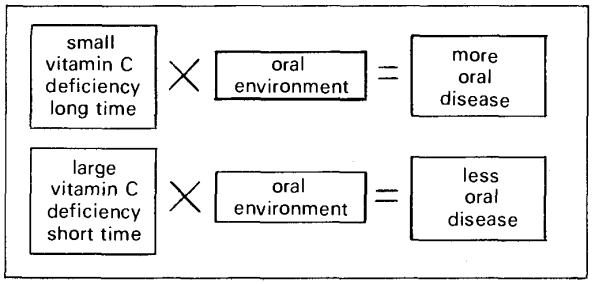

Figure 6 The role of time in the development of chronic disease.

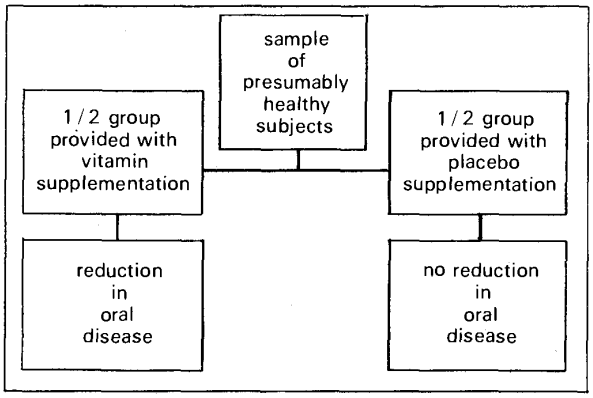

Figure 7 The experimental model needed to demonstrate the adequacy or inadequacy of the American diet.

evidence that the American diet leaves much to be desired. **On this sixth point, the position will be that Americans are generally not undernourished, meaning starved. However, utilizing the experimental technique just described, there is reasonable evidence to suggest that a significant segment of America is malnourished, meaning consuming food of poor quality.**[12]

Seventh, it appears that man has created an artificial world which now demands an artificial lifestyle (Figure 8). For example, there is evidence to suggest that, when a rat is placed in an experimental smog not unlike that of Tokyo or Los Angeles, the rat will develop the problems which man acquires under these conditions, namely bronchitis, bronchiectasis, and bronchopneumonia. However, if the rat under these exotic conditions is fed relatively large amounts of Vitamin E, then the respiratory findings

do not follow. The environment which leads to dental disease is indeed artificial and man-made. The solution, dental therapy, is also an artificial answer. **The position is taken here, with regard to this seventh point, is that our lifestyle demands heroic countermeasures; and diet, in megavitamin form, may sometimes serve as a solution.**

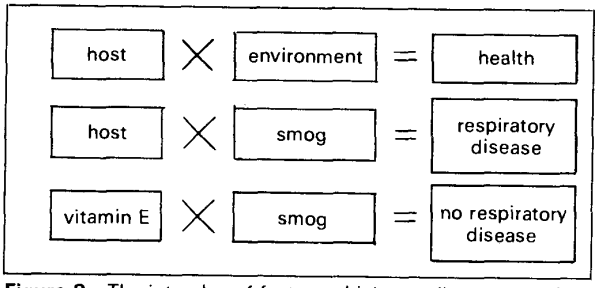

Figure 8 The interplay of factors which contribute to respiratory disease. Smog is a minus and, as such, contributes to respiratory disease. Vitamin E, as a plus, cancels out the minus of the smog.

The eight and last point centers about the semantics of causation. In traditional circles, if **B** follows **A**, then **A** is held to be the cause and **B** the effect. The possible illogic of this type of thinking can be readily demonstrated. For example, if a brain-tumor patient with a headache is given a shot of morphine and his headache vanishes for a period of time, may one assume that the subject had a morphine deficiency? The point being made is that it is frequently difficult, if not impossible, to identify a single cause. For example, it is generally agreed that hemophiliacs bleed more easily than nonhemophiliacs following simple injuries like a fall or dental extraction (Figure 9). Since the injury antedates the bleeding, can one say that hemophiliac bleeding is **caused** by an hemophiliac propensity. The answer is no. The bleeding is a result of the interplay of the environmental trauma **and** the hemophiliac propensity. Thus, it is unrealistic to assume, as many have, that plaque causes oral disease and that other factors are simply conditioning variables. Put in very simple terms, which is more important in an automobile, the wheels or the motor?

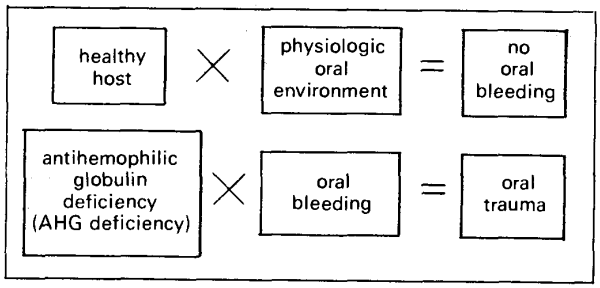

Figure 9 The semantics of causation.

Hence, the eighth and final position taken is that many factors enter into the causation of disease; and, more times than not, it is difficult if not impossible to assign relative importance. The mere fact that results will follow a change in one variable does not preclude the possibility that the same change may not be brought about by modifying another factor. Also, even a greater change may follow altering two or more conditions.

There are, of course, many differences between the practice of medicine and dentistry. The cardinal one, it would seem, is the fact that practically every dental manipulation injures the patient. Surely, this is the case with a dental extraction. Clearly, tooth movement for orthodontic purposes deliberately destroys tissue. In a more subtle form, the placement of a matrix for an interproximal restoration damages tissue. Finally, even simple cleansing and polishing of the teeth is not without trauma as judged by the frequent bleeding. In short, the dentist regularly damages the patient and then discharges him or her with absolutely no knowledge of the individual's capacity to heal. Quite apart, one should underline the point that there is a considerable body of fact which suggests that Vitamin C plays a crucial role in the wound healing process. Finally, there is some evidence that Vitamin C deficiency is one of the common shortcomings in the American diet. With these items in mind, let us recount a simple experiment which can be readily reproduced in a private practice environment.

A three-millimeter wound was deliberately inflicted in a presumably healthy junior dental student. At least, by the usual definitions, the student would not be regarded scorbutic. Each day the wound was painted with toluidine blue and photographed. Parenthetic mention should be made that this particular dye adheres to nonepithelial tissues. The daily painting procedure was continued until the wound was completely healed as judged clinically by the absence of staining with toluidine blue. After a two-week rest period, a similar wound was placed in the same area on the contralateral side of the mouth.

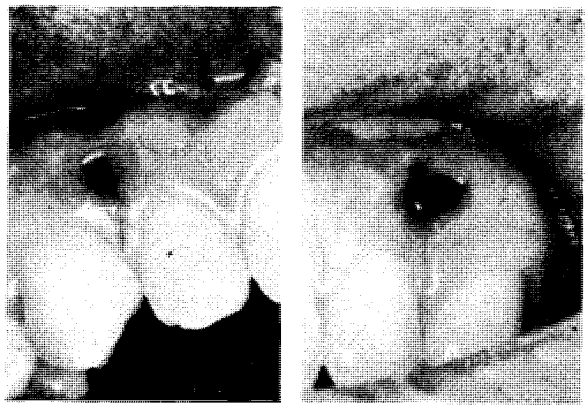

Figure 10 Healing on the third day without supplementation (on the right) and with vitamin C supplementation (on the left).

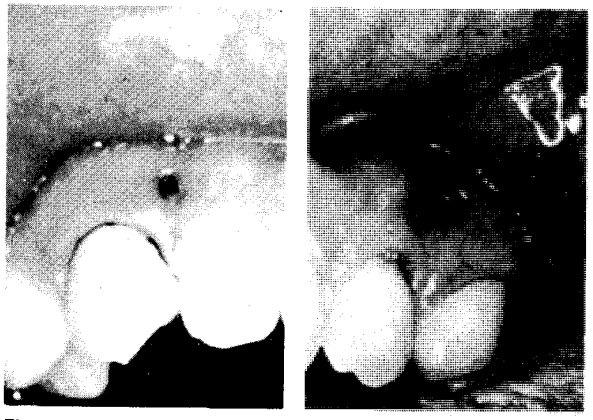

Figure 11 Healing on the sixth day without supplementation (on the right) and with vitamin C supplementation (on the left).

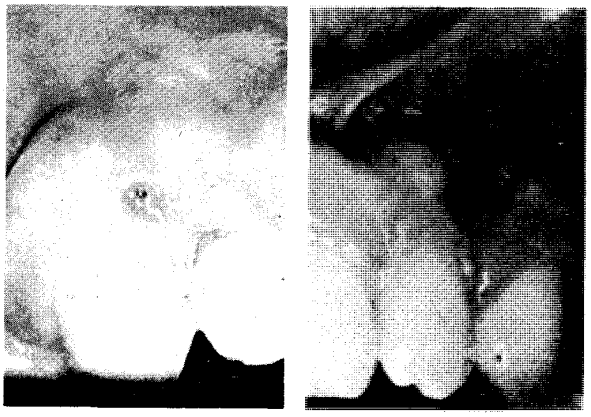

Figure 12 Healing on the eighth day without supplementation (on the right) and with vitamin C supplementation (on the left).

Daily painting with the dye and daily photography was once again carried out until the wound was completely closed. The photographs were cut and repositioned so that both sides for the same subject and the same day could be compared. The only difference was the fact that, during the second experiment, the subject received one thousand milligrams of Vitamin C daily by mouth in four equally divided dosages.

Figure 10 shows, on the right, the wound without supplementation on the third day. The illustration on the left is the same subject on the same day with ascorbic acid supplementation. It is abundantly clear that the wound on the left is significantly smaller. Figure 11 pictorially portrays the clinical state on the sixth day, and the pattern continues on course. Finally, Figure 12 shows the clinical condition on the eighth day. The wound following supplementation (on the left) is completely closed. **In brief, within the limits of this study, wound healing can be significantly accelerated approximately 40 percent with Vitamin C supplementation.**

What is particularly relevant to this report is that this clinical demonstration underlines five of the position points discussed earlier (Figure 13). First, it emphasizes the point that dietary factors may be operative in oral disease. Second, it underlines the gradation concept since junior dental students are clearly not classically scorbutic. If there is a Vitamin C deficit, it must be in the gray area. Third, this exposition brings into focus the relationships of Vitamin C to wound healing rather than to a specific textbook syndrome (e.g., scurvy). Fourth, this experiment suggests the possibility that the American diet may be sub-optimal as judged by the fact that improvement followed Vitamin C therapy in subjects presumably not needing Vitamin C. Finally, this exercise raises the interesting question of megavitamin therapy since the dosages here are approximately twelvefold the recommended dietary allowances.

wound healing experiment		ITEMS IN POSITION REPORT	vitamin C prophylaxis experiment
	1	multifactorial nature of oral health and disease	•
•	2	diet contributes to oral health and disease in the form of resistance factors which discourage disease and susceptibility factors which invite disease	•
•	3	disease exists in shades of gray	•
•	4	nutrients affect metabolism rather than cause specific clinical syndromes	
	5	time is an essential ingredient in the development of chronic disease	
•	6	the American diet is generally inadequate	
•	7	rationale for megatherapy	
	8	semantics of causation	•

Figure 13 The positions summarized by the two experiments.

While the wound healing experiment tends to support some of the positions taken in this report, it does not others. Thus, a second experiment is described (Figure 14) in which one group of subjects received placebo supplementation while another group was given one hundred milligrams Vitamin C thrice daily by mouth.[13] Additionally, one-half of the teeth were scaled and polished. Gingival inflammation was graded initially and three weeks later. This method permits a study of the relative efficacy of (1) no therapy, (2) local treatment, (3) host therapy, and (4) combined host and local treatment. It will be observed that the side with no treatment showed essentially no statistically significant change in inflammation. This is pictorially portrayed as the stippled column marked "d". In contrast, the contralateral side in these very same subjects who received prophylaxis, pictured as the stippled column "c", demonstrates a 30 percent reduction in gingival inflammation. This clearly underscores the general clinical observation that prophylaxis is desirable. However, as shown by the

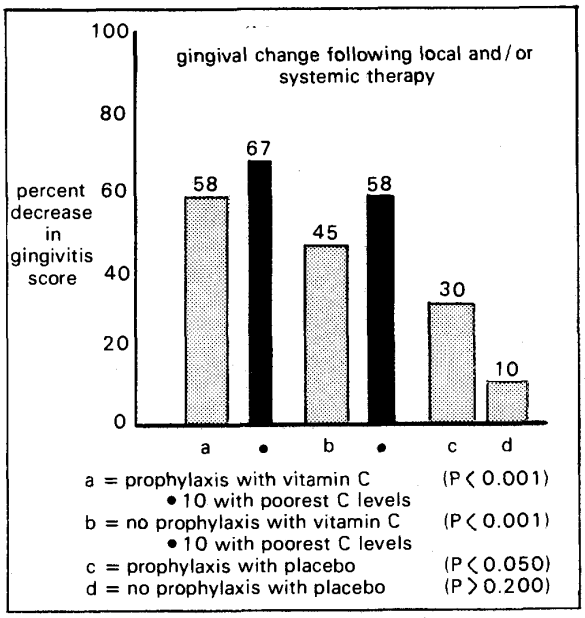

Figure 14 The relative effects of host and local therapy singly and in combination with regard to change in gingival inflammation.

stippled column "b", Vitamin C therapy without prophylaxis yields a 45 percent gingival improvement. Most significant is the 58 percent improvement in stippled column "a" in the group subjected to both local and host therapy. Every such study which has been performed to study other parameters[14,15] (e.g., sulcus depth, clinical tooth mobility, subclinical tooth mobility, oral hygiene, calculus) and other nutrients and other dosages clearly indicates that combined therapy (column "a") produces the greatest change; no therapy (column "d") yields no change. There are, however, differences among the different studies with regard to the relative merits of local ("c") versus host ("b") therapy as single variables. Sometimes local treatment is more efficacious than host therapy; sometimes the converse obtains. This is a function of the supplement, the host state of the subjects, the technical ability of the operator, and other known and likely unknown variables. Hence, it cannot be said that Vitamin C is superior to prophylaxis as one might try to conclude from this particular illustration. Parenthetic mention should be made that, if the therapy is customized by supplying Vitamin C to those who most need it (those with the poorest Vitamin C levels as shown by the black columns), efficacy is further increased about 10 percent.

Returning to the earlier position chart (Figure 13), one finds that this experiment covers four of the items described. First, here is definite evidence that oral health and disease fit best a multifactorial system. One learns, secondly, that, by the definitions set forth earlier, Vitamin C becomes a resistance agent because it tends to discourage, in this

particular study, gingival inflammation. Clearly and third, these subjects were presumably healthy and likely not suffering with classical scurvy. Hence, one is dealing either with a form of miniscurvy or subclinical scurvy or an unnamed syndrome. Finally, this experiment clearly emphasizes why it is difficult to assign specific and single causes to disease states.

Shown here is clearly not the entire story. For example, item five in Figure 13, the time factor, has not been considered at the experimental level nor have other points been introduced. This is all in the interest of expedition though it should be pointed out that published material is available.[16-20]

In the final analysis, whether one remains well or succumbs to illness, oral or otherwise, depends upon the environment and the organism's ability to withstand the environmental stressors including plaque. Of the many investigators who have addressed themselves to this issue, none has expressed it more eloquently than Doctor Jacques May[21] when he said:

> *"It is as though I had on a table three dolls, one of glass, another of celluloid, and a third of steel, and I chose to hit the three dolls with a hammer, using equal strength. The first doll would break, the second would scar, and the third would emit a pleasant sound."*

There seems to be general agreement that the clinical picture is not infrequently out of proportion with the environmental challenges. Additionally, it is often noted that the therapeutic success is quite variable in seemingly similar cases. There appears to be little doubt that man is a food-dependent creature. Adding up all of these seemingly independent points, this position report must take the stand that diet plays a significant role in oral health and disease. □

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