Plaque Prevention: Suspension Theory and Ascorbic Acid and Urea

In 1966, Ecanow[1] suggested that plaque formation conformed to aspects of suspension–flocculation–coagulation theory. The process involving teeth commences with the adherence of a surfactant film on the tooth enamel. Surfactants can form a film even on clean teeth. Chemically, the surfactant may be a mucopolysaccharide, a glycoprotein, or other biosurfactant; or the surfactant may be supplied from exogenous sources.

Once the initial film of surfactant forms on the enamel surface, any insoluble particles present, such as viruses, carbohydrates, bacteria, and environmental pollutants (*e.g.*, smoke), will adhere to the film because of the surfactant's wetting action and the large specific surface area of the particles. These particles, in turn, act as loci for the adsorption of other surfactants from saliva and food, and thus the process continues. A large quantity of sorbed water molecules becomes intermixed with the developing matrix, and the result is the formation of a semisolid gel with micelles and coacervate systems present.

According to Bowen[2], the gel matrix is composed of proteins (40–45% dry weight) and carbohydrates (10–20% dry weight) and contains approximately 80% water. Unless these structured water systems are removed by brushing or dissolution, they tend to concentrate calcium phosphate and other calcium salts (which are bulk water insoluble), promoting the formation of a calculus state. This whole process is similar to that of caking in pharmacy[3].

We have found that a 40% solution of urea in water is effective in removing all but trace amounts of synthetic surfactants (dioctyl sodium sulfosuccinate[4] and benzalkonium chloride) and biosurfactant (lecithin) films from horse teeth. Clean teeth that are bathed with a solution of 40% urea will resist the initial adherence of the surfactant to the enamel surface. Even though the gel state plaque may have formed and increased in size, it has been our experience that both ascorbic acid (vitamin C) and urea are useful in plaque dissolution in the earlier stages of formation. But once the plaque has developed into a highly structured phase or once calculus has formed to a marked extent, the dissolution properties of ascorbic acid and urea fail to have the same pronounced effect.

The use of ascorbic acid in plaque prevention has been noted in the literature, but the results have been mixed. The explanation for the mechanism by which ascorbic acid acts in this role has, in general, been based on a biochemical approach. We believe that the mechanism of action can be explained on a physical–chemical basis. The observed results suggest that ascorbic acid behaves as a water structure breaker, which destroys the plaque matrix so the system becomes dispersible. Urea, which is known to be an even stronger structure breaker than ascorbic acid, should be even more effective in inhibiting plaque formation and, thus, a preventative in periodontal disease.

Urea has been used in the past for this purpose. Although the matrix breaking effects of urea have been previously reported[5], the mechanism of action as a prophylaxis for caries has been based on its ability to neutralize acids present in the plaque matrix. The similarity in dissolution processes between ascorbic acid and urea suggests that more than acid neutralization is involved[6]. This possibility is further indicated by the observation that the use of neutralizing agents is not appreciably effective. Using a model system (a horse tooth), we have found that plaque formation can be readily prevented; or once formed, it can be effectively dispersed by simple soaking in an aqueous solution containing 40% urea. Therefore, we suggest that urea be reconsidered for use (after appropriate safety tests) in concentrations up to 40% in mouthwashes as a preventative measure against plaque formation. We also recommend that insoluble colloids that are readily adsorbed on surfactant films be avoided in the formulation of oral products and that due consideration be given to the surfactant components in food, toothpastes, and mouthwashes since they may act as potential plaque formers.

Bernard Ecanow
Martin I. Blake
University of Illinois at the Medical Center
Chicago, IL 60612

Received March 3, 1978.

[1] B. Ecanow, U.S. Copyright A825763 (Ecanow, Skokie, Ill.), 1966.
[2] W. H. Bowen, *J. Am. Pharm. Assoc.*, **NS17**, 709 (1977).
[3] R. G. Wilson and B. Ecanow, *J. Pharm. Sci.*, **52**, 757 (1963).
[4] Aerosol OT.
[5] R. M. Stephan, *J. Dental Res.*, **22**, 63 (1943).
[6] S. Levin, "Vitamin C: Its Molecular Biology and Medical Potential," Academic, New York, N.Y., 1976, p. 61.

Carcinogenesis Hypothesis

My response to the letter entitled "Biological Foreign Particle Film Encapsulation," by Ecanow *et al.*[1] is: "Nonsense!" The authors' suggestion attributing "carcinogenesis" to the presence of inert "Class B" particle granules reminds me of the "spontaneous generation theory" for the origin of life. Here follows a more plausible and, ergo, less miraculous, albeit heuristic, hypothesis of my own to explain the observations reported[1].

The Class B particle complexes with the cellular material present and has the immunochemical characteristics of a Landsteiner hapteen[2]. The hapteen-like complex can initiate and provoke an autoimmune reaction. If the autoimmune reaction is massive, immunosuppression and immunological paralysis may ensue. If a virus or viral DNA, having appropriate affinity for the cellular matrix present, is available contemporaneously with a less-than-adequate immune response system, the viral triggering of a neoplasm may happen. Any chemical administered in doses large enough to be toxic will be immunosuppressive and can enable the viral initiation of a cancer.

In the studies establishing the identity of "carcinogens," the vast majority of the test animals die of infection. Only a small percentage survive long enough to develop cancers. Data accompanying official FDA reports on carcinogens never identify the infectious cause of death in the noncancerous test animals or rule out the coincidental presence of viruses. I am always amazed that prestigious journals like *J. Pharm. Sci.* fail to challenge the investigational adequacy of the mediocre research studies that important governmental policies use for rationalization. Before you accept any bureaucrat's allegations that sodium cyclamate or saccharin or Egyptian Henna are carcinogens, why don't you ask about the antibody titres of the test animals, if antibiotics were given to suppress infections, whether the presence of viruses or viral material was ruled out, and how competent and honest the investigators were?

I seem to be the only one who regularly raises these doubts. The APhA is cozy to the point where it does not exercise a virile and independent objectivity apropos federal government handouts. Beware, 1984 is only 6 years hence!

Sidney Silberberg
5725 Idlewild Avenue
Livermore, CA 94550

Received February 23, 1978.

[1] B. Ecanow, B. H. Gold, B. H. Moon, and R. S. Levinson, *J. Pharm. Sci.*, **67** (1), VIII (1978).
[2] K. Landsteiner, *Proc. Roy. Acad. Sci., Amsterdam*, **30**, 329 (1921).