3) The approximate breakpoint in the pleasantness function occurs at a constant sweetness level for the foods (approximately 4.5 to 5.0 for the category rating of sweetness and between 1.0 and 1.2 for the logarithm of the magnitude estimate of sweetness).

4) Foods have a higher sweetness level for their breakpoint than sucrose solutions do, but the form of the sweetness-pleasantness relation is similar for model systems and for these foods. The fact that foods have the higher sweetness tolerance before the breakpoint suggests partial cognitive control over the pleasantness function that interacts with the perception of sweetness.

5) The downward sloping portion of the pleasantness function is no steeper (in absolute value) than the upward sloping portion.

In light of the present results it appears possible to disentangle the sweetness (discriminative) and pleasantness (affective, hedonic) aspects of a taste impression, both in foods and in model systems, and to represent each by its own unique function. It is important that real foods, evaluated by the same procedures as model systems, conform to topographically similar functions. This similarity suggests that there may be a general system of functions to govern the relation between chemical concentration of tastants in foods, taste intensity, and taste pleasantness. Caution is warranted about extensions of the present functions to foods that are not usually sweetened, however. Unsweetened foods, when sweetened, may yield functions for "unpleasantness of taste" that differ in shape from those found here (especially with respect to the absence of a breakpoint in the unpleasantness function).

The present results and approaches to sensory and hedonic measurement bear upon two major areas. First, the existence of a breakpoint at a *fixed sweetness* (not a fixed concentration) is evidence that the pleasantness response is predicated upon sensory intensity. The critical sensory sweetness needed for the breakpoint is approximately invariant. [That is, the sweetness of 1.0*M* glucose, or 18 percent by weight and volume, and 0.21*M* sucrose (here), or 7.1 percent by weight and volume, are approximately equal by direct magnitude estimation of sensory intensity reported by Moskowitz (*4*).] Furthermore, the breakpoint, a type of yes-no phenomenon occurring at the same response level across different experiments, implies that sensory sweetness is encoded in absolute terms, not relative terms, in the sensory system. Otherwise, the sensory intensity of sweetness corresponding to the breakpoint of the pleasantness function would be expected to shift dramatically from one experiment to another, both as a function of the context and the nature of the other stimuli being judged.

Second, the existence of the breakpoint and the difference between sweetness and pleasantness functions can be used to investigate hedonic responses to taste input under varying body states (for example, hunger, satiety, obesity, and so forth). Recent studies by Cabanac (*7*) suggest that body state modifies the hedonic response to foodlike materials (such as sugar solutions). Cabanac's studies can be extended by the simultaneous use of sweetness and pleasantness scales for sucrose solutions and foods to determine whether the breakpoint continues to appear after a satiating meal, and, if it does continue to appear, whether the breakpoint occurs at the same sweetness level as before. Insofar as the present procedure yields conclusions based upon interrelations among several subjective judgments, it allows a more bias-free measurement of hedonic response than procedures that rely upon changes in the response to a single stimulus over time and treatment.

HOWARD R. MOSKOWITZ
ROBERT A. KLUTER
JUDITH WESTERLING
HARRY L. JACOBS

Pioneering Research Laboratory,
U.S. Army Natick Laboratories,
Natick, Massachusetts 01760

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Surface Charge, Protein Adsorption, and Thrombosis

The recent report by Mattson and Smith (*1*) appears to be derived from and leading to some misconceptions.

The reviews listed under reference 1 in (*1*), if read carefully, show that no agreement exists about primary causes of protein adsorption, platelet adhesion, and thrombosis. Friedman *et al.* (*2*) [reference 3 in (*1*)] did not state "that the free energy of the surface is responsible for its thrombogenicity" (*1*) but rather that it "depends primarily on blood flow rate, time of exposure of the surface to blood, and possible platelet activity, rather than properties of the surface (which may, however, affect sequelae to adhesion)" (*2*).

Perhaps the net charge of a surface plays a role in thrombosis under certain conditions. Unfortunately, it is a negative rather than a positive charge (on glass, collagen, and phospholipid micelles) that enhances blood clotting. Stoner and Srinivasan (*3*) [reference 5 in (*1*)] erroneously reported that, according to me (*4*), the Hageman factor —possibly the first clotting factor in the sequence leading from contact with a surface to the clotting of blood— would be activated by positively rather than negatively charged surfaces.

The material used by Mattson and Smith (*1*) was 60 percent clottable fibrinogen, in other words, 40 percent something else. Their methods are unsuited for the identification of adsorbed protein, and not very well suited for quantification. Vroman and his coworkers have found that ellipsometry allows the identification of the adsorbed protein, if used in conjunction with specific antisera to identify adsorbed matter (*5*).

Mattson and Smith seem to suggest that the behavior of their crude fibrinogen preparation bears a relationship to the behavior of fibrinogen in whole plasma. I believe that, for such a comparison, their preparation is not quite crude enough. In several publications (*5*) Vroman and his co-workers reported that fibrinogen is deposited more or less preferentially by normal plasma onto several surfaces but is, within 30 seconds, modified by the plasma itself. Elsewhere Zucker and

Vroman (*6*) state that platelets adhere preferentially to glass coated with unmodified fibrinogen. Others (*7*) found a similar correlation; however, it is not a perfect one. For example, on some positively charged surfaces, such as surfaces coated with protamine sulfate and 2-hydroxy-3-methadcryloyloxypropyltrimethylammonium chloride polymer, fibrinogen appeared to be adsorbed out of plasma, but on a surface coated with polybrene fibrinogen was not adsorbed. On the other hand, platelets did not adhere to the surfaces that were coated with protamine sulfate (*8*).

Suggesting that simple net charge determines thrombogenicity is as disrespectful to the complexity and beauty of nature as suggesting that it is simply our body weight which keeps us alive.

LEO VROMAN

Interface Laboratory,
Veterans Administration Hospital,
Brooklyn, New York 11209

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We are in complete accord with Vroman's remarks that little agreement exists among the investigators in this field regarding the relative importance of protein adsorption, platelet adhesion, and mechanical effects in thrombus formation. Vroman implies that we suggest ". . . simple net charge determines thrombogenicity. . . ." We did not make such a generalization, and we hope that our report (*1*) was not taken in that context by others. However, we are suggesting that net surface charge is a factor in the adsorption of blood proteins, and therefore may well be a contributing factor in thrombogenicity.

Baier *et al.* (*2*) have made it clear that formation of an adsorbed protein film precedes thrombus formation at foreign surfaces. It is not unrealistic to believe that this initial protein film may serve to propagate some thrombogenic property from the surface to the blood. Sawyer (*3*) has observed that various metals tend to thrombose in an apparent relationship to their position in the electromotive series, the noble metals being the most thrombogenic. Epstein and Dalle-Molle (*4*) have examined the surface charge characteristics of low-temperature, isotropic, pyrolytic carbon, a material which, when highly polished and clean, displays excellent thromboresistance. Their results indicate that this material has a high point of zero charge [approximately +100 mv relative to a saturated calomel electrode (SCE) in plasma and +300 mv relative to an SCE in 0.89 percent (by weight) NaCl] and that it exhibits a very slightly negative surface charge at its rest potential in both plasma and saline (*4*).

It was our intention in our report (*1*) to show adsorption at the solid-liquid interface *directly* using infrared internal reflection spectroscopy, while carefully controlling and reporting the actual surface *charge*, as well as the applied potential, of the metal. The fact that we used a crude fibrinogen preparation unfortunately draws attention away from our goal. The use of plasma would have been more in accord with past practice; however, as chemists, we felt more comfortable using a solution with a bit simpler composition. We believe that the relative merits of ellipsometry versus computer-assisted infrared internal reflection spectrometry are beside the point. In subsequent experiments, using the same cell (and 60 percent clottable fibrinogen) but with the addition of a minicomputer to average spectra and obtain difference spectra, we have determined that enhanced protein adsorption occurs on germanium at potentials much lower than the −200 mv observed in (*1*). At the reported point of zero charge of germanium, −900 mv, no enhanced adsorption is observed for as long as 5½ hours, although strongly enhanced adsorption is observed rapidly at −350 mv (relative to an SCE).

Recognizing the limits of our technique, we readily acknowledge that ellipsometry is capable of detecting very thin (about 5 Å) films. Infrared internal reflection spectrometry offers an advantage over ellipsometry in that it provides qualitative information on the composition of the adsorbed film. When used in conjunction with a dedicated minicomputer, it can provide quantitative measurements on films in the 25- to 1000-Å range. In either case identification of the adsorbed proteins must depend on the use of specific antisera, as Vroman mentions.

JAMES S. MATTSON

Division of Chemical Oceanography, Rosenstiel School of Marine and Atmospheric Science, University of Miami, Miami, Florida 33149

CARROLL A. SMITH

Division of Ocean Engineering, Rosenstiel School of Marine and Atmospheric Science

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"γ-Glutamyl Cycle"

In setting forth a proposal of a γ-glutamyl cycle in amino acid transport (*1*), Meister and co-workers did not mention a much earlier proposal of a similar mechanism (*2*). That earlier proposal was limited to tissues concerned with transmural transport because many differentiated tissues quite active in amino acid transport were known to be devoid of the activity (*3*); the system is also found in certain fetal and dedifferentiated tissues (*4*). The proposal was abandoned in the course of later work because the specificity of the system was not found to correspond to the specificity of amino acid transport by renal tissue. Although isozymes (at least five) are present in renal tissue, they do not vary significantly in specificity and many readily transported amino acids and analogs are poor substrates (*5*). We have found the effects of Na^+ to be highly variable and dependent upon the state of the preparation; the purified enzymes are insensitive to Na^+ when tested with

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Leo Vroman, James S. Mattson and Carroll A. Smith

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