Comments on Garlic Chemistry: Stability of *S*-(2-Propenyl) 2-Propene-1-sulfinothioate (Allicin) in Blood, Solvents, and Simulated Physiological Fluids

Sir: We have found a number of problems in the publication by F. Freeman and Y. Kodera (*J. Agric. Food Chem.* **1995**, *43*, 2332−2338). In brief, this publication uses extremely high concentrations, incorrect premises, and an invalid test to make allicin, a sulfur compound for which there is abundant and widely respected evidence as the main known pharmacologically active compound released from garlic, appear to be potentially harmful, useless, and unreleased from commercial garlic powder tablets. A few of the specific problems are listed.

1. Allicin is presented as possibly being harmful ["These results suggest that the iron in hemoglobin may be rapidly oxidized by interaction with allicin." (p 2336)] because it is shown in Figure 8 to oxidize the iron in hemoglobin. However, the dose used in Figure 8 (53 mg/mL of blood) is 25 000 times the maximum possible dose achievable by consumption of a typical 3 g clove. To achieve this dose, a person would have to eat his/her own body weight in fresh garlic in 1 min. Furthermore, the concentration used in this experiment is far greater than the allicin levels used in any of the other experiments (0.1−1.2 mg/mL; Figures 1, 3−5, 7), implying that this effect on hemoglobin could not be observed at substantially lower concentrations. There is no evidence that normal consumption (one clove per day) of garlic, which releases allicin when crushed or chewed, leads to any harmful effects when eaten with a meal. In fact, there is good agreement that allicin from fresh garlic exerts an antibiotic effect in the intestinal tract and considerable evidence that it indirectly exerts positive cardiovascular effects.

2. A main premise for the research presented in the subject paper, as stated on p 2333, is "Before one can evaluate the effectiveness of allicin in the body, it must be determined whether or not it can actually reach the target organs." The results of the paper then correctly show that allicin is very rapidly metabolized by blood (shown also in three prior publications, none of which are cited), implying, according to the above statement, that allicin cannot be an effective agent because it cannot reach target organs. However, this statement ignores the likelihood that the metabolites of allicin are the ultimate active agents (Lawson and Wang, 1993). Many nutrients have no pharmacological activity until they are metabolized in the body, for example, linoleic acid, retinol, niacin, and pyridoxine. Metronidazole is an example of an antibiotic that must first be metabolized to have activity. The effectiveness of a compound is based on end results, not on its stability in blood.

3. Also found on p 2333 is the statement "Owing to its instability in fatty oil and in organic solvents, it is doubtful whether allicin is responsible for some of the pharmacological effects attributed to garlic." This statement is misleading because the large majority of the clinical cardiovascular studies on garlic have used alliin/allicin standardized garlic powder tablets, which are produced without the use of oil or other organic solvents. Furthermore, the environment of the intestinal tract and the rest of the body is not similar to that of a fatty oil or an organic solvent.

4. The abstract, based on the data in Table 3, states that "The ... allicin-producing potential of commercial garlic preparations ... was severely suppressed under simulated digestive conditions (sequential combination of SGF and SIF)." This is an erroneous statement because none of the "commercial garlic preparations" (listed in Table 1) were tested in this experiment, which instead used only a simple untableted garlic powder. It has been known for several years that alliinase, the enzyme that rapidly converts the alliin present in garlic and garlic powder to allicin, is completely inhibited by the acidic conditions found in the stomach (Blania and Spangenberg, 1991; Lawson and Hughes, 1992). This is why most of the brands of products listed in Table 1 use an enteric coating or other means of offering resistance to gastric acid. A recent study in which 28 brands of garlic powder tablets were exposed to simulated digestive conditions showed that high amounts of allicin were formed if the tablets were acid resistant and contained high amounts of alliin (Koch and Lawson, 1996).

5. Table 1 gives the allicin potential (a measure of alliin content plus active alliinase) of several brands of garlic powder products; however, on the basis of the given lot numbers, most of these products were produced in 1989−1990. Much more recent lots should have been used for this 1995 publication. Due to various improvements in quality over the past several years, many of the values reported here are considerably lower than have been recently reported by others (Schardt and Liebman, 1995). For example, the lot of Garlicin brand reported in Table 1 (560 μg/g) was a softgel capsule containing a suspension of garlic powder in vegetable oil. When it was learned that the allicin potential of this product was not stable (50% loss per year), it was discontinued and a much more stable tablet was produced (March 1991). An independent report showed that a 1994 lot of this product had an allicin potential of 2500 μg/g (Schardt and Liebman, 1995).

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