TECHNICAL NOTES

Homogenized Milk: Is It Really the Culprit in Dietary-Induced Atherosclerosis? 1

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ABSTRACT

Xanthine oxidase activity was assayed in commercial samples of homogenized milk subjected to pH ranging from 6.7 to 2.0 and held at room temperature for 5 min. Activity decreased sharply between pH 5.5 and 3.2. Below pH 3.2 no activity was detected. Also, rabbit anti-bovine xanthine oxidase failed to crossreact immunologically with xanthine oxidase of mouse milk. These results cast doubt on the hypothesis that dietary xanthine oxidase participates in the formation of atherosclerotic plaques.

INTRODUCTION

Reports by Oster (2, 3) related homogenized milk with the etiology of atherosclerotic plaque formation. It was hypothesized that the smaller fat globules along with associated xanthine oxidase (XO) were absorbed directly into the lymphatic system. It further was presumed that upon entering the cardiovascular system, the enzyme oxidizes the aldehyde form of plasmalogens associated with the arterial wall leading eventually to plaque formation.

This report describes the irreversible, pH-dep- endent inactivation of XO when exposed to low pH, presumably stimulating those conditions in the stomach of man.

EXPERIMENTAL PROCEDURE

Five samples of commercial homogenized milk were obtained from supermarkets in the Lansing, MI area. Activities of XO were determined by monitoring the change in absorbance at 296 nm resulting from the conversion of xanthine to uric acid (1). Aliquot of the milk samples were adjusted to pH ranging from 6.7 to 2.0 with .1N HCl and held at room temperature for 5 min. The incubation period was terminated by transfer to phosphate buffer at pH 8.2, holding for 30 to 300 min. The percentage of original activity as a function of pH exposure for a typical sample of homogenized milk is in Fig. 1. Each data point represents the average of three determinations. Enzyme activity decreased sharply between pH 5.5 and 3.2. At values lower than pH 3.2, no activity was detected in any of the milks nor

FIG. 1. Effect of pH on activity of homogenized milk xanthine oxidase.
was the enzyme reactivated by holding the pH-treated samples at pH 8.2 for up to 5 h.

Presumably, the human stomach normally maintains a pH of about 2.0 which may rise to approximately 5.0 upon the ingestion of food, followed by a return to more acidic conditions. Thus, it appears that dietary XO would be exposed to conditions causing at least a partial irreversible denaturation, the extent of which depends on the pH of the stomach at time of ingestion. Furthermore, it has not been demonstrated clearly that the enzyme-associated membrane particulate or even the relatively smaller enzyme (approximately 300,000 daltons) itself is transported across the stomach wall or intestinal mucosa. If, indeed, it were, we might expect to encounter antibody production within the host giving rise to allergenic symptoms. In this regard, tentative evidence supporting the existence of species specificity for XO was demonstrated in this laboratory by the failure of rabbit anti-bovine XO to crossreact with the cream phase of mouse milk. The presence of XO in mouse milk was verified by assay.

These limited observations contribute inconclusive evidence against the role assigned by Oster to membrane-associated XO of homogenized milk. Obviously, a definitive answer awaits further, more sophisticated studies. However, the hypothesis as originally presented is too important to the dairy industry to remain unchallenged and unverified.

REFERENCES

