CORRESPONDENCE

Comments on Bioavailability of Iron to Rats from Nitrite and Erythorbate Cured Processed Meats

Sir: We have read the paper "Bioavailability of Iron to Rats and Nitrite and Erythorbate Cured Processed Meats" by Lee et al. (1984) with interest. This is an important subject that deserves careful study because of the huge amounts of cured meat products consumed throughout the world. We believe that the following comments are pertinent to this paper.

It appears that an error has been made in Table V. We do not believe that the rat is capable of converting 71% of the ingested iron into hemoglobin while absorbing only 27% of the ingested iron. This is inconsistent with the findings of others (Mahoney et al., 1979). Net hemoglobin iron gain should be expressed in milligrams rather than percent. We do not believe that the data in Table V warrant the statement "In this study rats utilized total heme and nonheme iron in the meat-based diets (uncured, +E, +N, and +E+N) less efficiently than the nonheme iron in the control diet (L+Fe)" (p 859, column 2, paragraph 2) since no statistically significant differences were reported among these treatments. Similarly, evidence for the statement "Not surprisingly, the animals fed the L diet used dietary iron significantly ($P < 0.05$) more efficiently than rats fed the four meat-based diets" (p 858, column 2, paragraph 4) is not presented.

Variability in this single experiment must have been rather large since a mean difference of 19% efficiency of converting ingested iron to hemoglobin was not statistically significant. Others (Mahoney et al., 1979; Park et al., 1983; Cardon et al., 1980) find that much smaller mean differences for this parameter are statistically significant. A single experiment using 36 rats divided among six treatments with the experimental variability reported in this paper is not, in our opinions, a rigorous test of the effects of curing with nitrite and erythorbate on the bioavailability of iron in cured meat.

It is well-known that anything which causes a relative tissue hypoxia results in increased hemoglobin synthesis through the erythropoietin system. On ingestion, nitrite is converted to nitric oxide, which binds strongly to hemoglobin, forming nitrosomethemoglobin. This then decreases the oxygen-carrying capacity of the blood, leading to relative tissue hypoxia. This hypoxia causes release of erythropoietin, a hormone that stimulates hemoglobin synthesis. In this way, hemoglobin concentration will increase and the oxygen-carrying capacity of the blood and tissue oxygenation are restored. There appears to be a threshold at which added dietary nitrite affects this system. Park et al. (1983) added 3.7 ppm of nitrite ion to the diet and observed no effect on the rat hematinic response. Mahoney et al. (1979) added 12.1 ppm of nitrite ion to both casein and meat diets and observed increases of 36 and 27%, respectively, in the hematinic responses of rats fed the casein and meat diets. (This was similar to the residual nitrite in the meat used to prepare the diets for their experiment 1.) Lee et al. (1984) with 33.9 ppm of dietary residual sodium nitrite (22.6 ppm of nitrite ion) found a statistically nonsignificant 33% increase in efficiency of converting ingested iron to hemoglobin iron for their nitrite added meat diet. Their failure to observe statistical significance for this great a nitrite effect was probably due to lack of sensitivity of their model since meat accounted for only 57–64% of the iron in their test diets and to the large experimental variability they encountered.

Table I. Effect of Adding Sodium Nitrite or Erythorbate during Curing on Bioavailability of Meat Iron to Anemic Rats[a]

	meat treatment				
erythorbate added, μg/g[c]	0	0	500	500	LSD 0.05/0.01[b]
sodium nitrite added, μg/g[c]	0	150	0	150	
residual nitrite ion, μg/g[d]	0	14	0	5	
diet iron, mg/kg	27.7	29.1	30.4	29.4	
initial body weight, g	77	79	78	79	NS[e]
body weight gain, g	38	43	46	45	5/7
initial hemoglobin, g/dL	5.66	5.54	5.67	5.74	NS
hemoglobin gain, g/dL	3.40	2.44	3.14	2.59	0.57/0.75
iron intake, mg	2.53	2.54	2.79	2.66	0.17/0.23
HbFe gain/Fe intake, mg/mg	0.58	0.44	0.52	0.48	0.06/0.08
liver iron, μg/g	105	125	101	104	10/14

[a] Each value is a mean of 10 rats that had been made iron deficient during a 7-day period by feeding a low-iron diet and bleeding as previously described [experiment 1, Mahoney et al. (1979)]. The rats were fed the test diets for 10 days. [b] Mean differences must equal or exceed the least significant difference value to be statistically significant at the 5 or 1% levels of probability. These data are from a larger experiment involving 160 rats divided among seven cured meat diets with varying amounts of sodium nitrite without erythorbate 7, cured meat diets with varying amounts of sodium nitrite with erythorbate added, low-iron casein diets, and low-iron casein diets supplemented with $FeSO_4$. The erythorbate data were not included in a previous paper (Mahoney et al., 1979). [c] Added to the meat emulsion during curing. [d] In the meat. [e] Not statistically significant.

Earlier, we found that adding erythorbate during meat curing did not affect the bioavailability of the meat iron whether added with or without sodium nitrite (Table I). This finding is similar to what Lee et al. (1984) observed. Both observations are inconsistent with the increased iron uptake observed with feeding ascorbic acid. This could be due to the effect of heating during the curing process, lyophilization, and subsequent grinding and mixing of the products into diets in these two studies that would be expected to oxidize the erythrobate, rendering it biologically inactive.

Registry No. Fe, 7439-89-6; erythorbic acid, 89-65-6; nitrite, 14797-65-0.

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Arthur W. Mahoney*
Deloy G. Hendricks

Department of Nutrition and Food Sciences
College of Agriculture
Utah State University
Logan, Utah 84322

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