

Letters to the Editor

Goat's Milk Anemia in Retrospect

Dear Sir:

"GOAT'S milk anemia" was the designation given to a macrocytic-hyperchromatic megaloblastic anemia which was observed in infants fed a diet of goat's milk in Europe during the 1920's and 1930's.¹ In comparison with a similar type of anemia which occurred in infants fed cow's milk, goat's milk anemia was observed to develop at an earlier age, more frequently, and of greater severity. Clinical trials by György in 1934¹ demonstrated that infants with goat's milk anemia could be cured by giving them supplements of yeast, liver or liver extracts. György suggested that the missing factor in goat's milk may be the same as Castle's extrinsic factor. Experimental studies by Rominger, Meyer and Bomskov² demonstrated that hyperchromic anemia would also develop in weanling rats after they had been fed goat's milk for six days. This anemia was cured by giving them supplements of iron and liver extract, but not by iron alone. These investigators agreed with György's suggestion that goat's milk was probably deficient in the antipernicious anemia factor proposed by Castle. It is now apparent that the therapeutic success with yeast was a result of folic acid while success with liver or liver extract could have resulted from folic acid and/or vitamin B₁₂.

Investigators in the United States were unable to produce an anemia in weanling rats fed a goat's milk diet supplemented with iron and copper.^{3,4} In addition, Kohler, Elvehjem and Hart⁵ observed in 1935 that when manganese was added with iron and copper, the rats fed goat's milk failed to grow as rapidly as those fed cow's milk. The reason for the poorer growth in the animals on goat's milk was not determined at that time.

In 1953 Collins, Schreiber, Elvehjem and Hart⁶ re-investigated the nutritional value of

goat's milk and observed it to be very low in vitamin B₁₂ in comparison to cow's milk, 0.15 μg . per L. and 4.0 μg . per L. respectively.⁶ The addition of 3 μg . of vitamin B₁₂ per L. to a mineralized goat's milk diet resulted in rat growth equal to that obtained with mineralized cow's milk. Also, the supplementation of 0.5 μg . of vitamin B₁₂ per rat per day to the mineralized goat's milk diet produced a similar optimum growth, while 0.1 μg . of vitamin B₁₂ per rat per day resulted in a partial growth response.

Folic acid supplementation to mineralized goat's milk also stimulated rat growth. Fifty micrograms of folic acid per rat per day gave a limited growth response; however, when combined with 0.1 μg . of vitamin B₁₂, maximum growth was attained. Thus folic acid spared a portion of the vitamin B₁₂ requirement. In addition, the liver storage of folic acid was greatly increased by adding the small amount of vitamin B₁₂ to the folic acid supplement. Analysis of both goat's milk and cow's milk for folic acid revealed comparable low levels of from 1 to 3 μg . per L. The major difference between cow's and goat's milk in respect to the content of these two vitamins was observed to be with vitamin B₁₂ and not folic acid.

One wonders to what degree the low vitamin B₁₂ content in goat's milk may have contributed to the "goat's milk anemia" syndrome. Present day experience indicates that infants with macrocytic hyperchromic megaloblastic anemia respond more readily to therapy with folic acid than to vitamin B₁₂.⁷ In 1948 Gasser⁸ described a patient with "goat's milk anemia" who responded completely to folic acid therapy. This was a three month old infant with severe normochromic megaloblastic anemia who had been fed a goat's milk diet. Although György's hypothesis¹ that goat's milk was deficient in Castle's extrinsic factor was verified

when Collins, Harper, Schreiber and Elvehjem⁶ demonstrated a very low vitamin B₁₂ content, the major deficiency in infants with goat's milk anemia is probably folic acid. In view of the known interrelationship between vitamin B₁₂ and folic acid, the folic acid requirement may be increased in infants fed the vitamin B₁₂ low goat's milk diet. This then would explain the occurrence of the more severe goat's milk anemia which responds to folic acid therapy.

"Goat's milk anemia" is a macrocytic hyperchromic megaloblastic infant anemia which responds to folic acid therapy.⁸ Historically, goat's milk anemia is more severe than a similar cow's milk anemia. It is probable that the difference is the result of an increased folic acid requirement in such infants due to the relative vitamin B₁₂ deficiency found in goat's milk.

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Teaching and Training in Nutrition

Dear Dr. Waife:

In response to Paul György's editorial, and your request for comments, I would like to mention the lack of nutrition education at the undergraduate college level.

As a result otherwise intelligent college-educated Americans derive much of their nutrition information from the backs of breakfast-food cartons, from vitamin advertisements and from special interest advertisers. Small wonder so many of them fall prey to the yoghurt vendors, evangelists for wonder foods and (in a search for health) are taken in by alfalfa mixes and malted milk tablets sold as protein "supplements." In talking with such deluded enthusiasts, they readily admit their lack of knowledge and view nutrition con-men as genuine experts.

Surely, college is one place to provide nutrition information and to give emphasis on worldwide problems of nutrition and the whole area of nutrition research. Courses in biology and chemistry need not be the only contributors to nutrition education. Psychology, sociology and cultural anthropology can help to describe the many problems relating to food preferences and food practices.

Without insisting on a proliferation of formal undergraduate courses on nutrition (remember the old freshman courses on "hygiene"), it would seem to be appropriate to establish special training programs for college teachers interested in nutrition both as a natural science and a social science. There is adequate precedence in the summer training programs in social gerontology, sponsored by the National Institutes

