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Vitamin A-Carotene Deficiency **Affects Serum Protein and** Utilization of Carotene by Steers

A close relationship between serum proteins and vitamin A-carotene utilization or tissue storage, or both, has been shown (1, 2). Limited data (3) indicate that the cow's carotene requirement is markedly increased after a prolonged suboptimum carotene intake. Therefore, this study was undertaken to determine whether vitamin A-carotene deficiency would influence the serum protein fractions or alter the utilization of dietary carotene by the beef animal.

Eleven 800-lb steers were pretreated as follows: six animals were fed a grainstraw diet (carotene-free), and five steers were fed a grain-dehydrated alfalfa ration, until the former group exhibited

vitamin-A deficiency symptoms (about 120 days). All animals were then fed a carotene-free diet for the 14-day experimental period. Pure carotene (10 percent alpha and 90 percent beta in an aqueous solution with Tween 80, 4) was administered by means of a stomach tube to all steers (15 mg of carotene/100 lb of body weight) on alternate days for the first 10 days of the experimental period.

Table 1 shows the plasma vitamin A and carotene and the serum protein fractions (5) of the normal and deficient animals before and after carotene administration. As expected, the plasma vitamin A and carotene increased after administration of carotene to the deficient steers. The normal steers exhibited a decreased plasma level of both carotene and vitamin A, the latter changing only slightly. Prior to carotene treatment, the percentage of beta globulin in the serum of the deficient steers was significantly higher than that in the normal steers. In the pretreated, vitamin A-deficient animals, serum albumin increased (average, 5.6 percent) while alpha and beta globulin decreased significantly (3 and 5.8 percent, respectively) after the 10 days of carotene administration. Although the remaining fractions differ, none were statistically significant. The total serum protein was not affected by either vitamin A deficiency or carotene administration. No correlation was found between the change in blood vitamin A and carotene and the change that occurred among the serum proteins. Al-

Table 1. Summary of results.

	Plasma		Serum*				Liver	
Day	Vita- min A (µg/ 100 ml)	Caro- tene (µg/ 100 ml)	Albu- min (%)	α-Glob- ulin (%)	β-Glob- ulin (%)	γ-Glob- ulin (%)	Vita- min A (µg/g)	Caro- tene (µg/g)
			Normal st	teers (avera	ige of five.	steers)		
0	65	240	41.2	17.6	16.6	24.6	23.41	7.36
2	60	263						
4	42	222						
6	48	232						
8	52	202						
10	53	150	44.5	15.4	15.7	24.4		
14							38.55	6.73
		1	Deficient	steers (aver	rage of six	steers)		
0	2	0	39.4	17.6	21.7	21.3	1.57	2.21
2	10	11						
4	26	22						
6	26	43						
8	28	51						
10	29	28	45.0	14.6	15.9	24.5		
14							0.58	0.54

* Each figure represents an average obtained from figures for four steers.

though work with human serum (1) has shown that beta globulin binds about 50 percent of the blood carotene, the beta globulin was markedly greater in the carotene-deficient steers in this study.

All animals were subjected to liver biopsy (6) at 0 and 14 days of the experimental period. The samples were analyzed for vitamin A and carotene (7). Table 1 shows the liver storage of vitamin A and carotene, as affected by pretreatment, before and after oral administration of carotene. The results of the liver analyses were extremely consistent within animals with the same pretreatment. Under the conditions of this experiment, there was no increase in the liver carotene content of the deficient or normal steers as a result of carotene administration. However, there was a large increase in the liver storage of vitamin A in the normal steers following carotene administration. There was no increase in the liver vitamin A content of the carotene-deficient animals. This marked difference in vitamin A deposition in the liver may suggest a lack of conversion of carotene to vitamin A in the deficient steers. However, the fact that plasma vitamin A increased in the deficient animals does not appear to uphold this hypothesis. On the other hand, these results may indicate extrahepatic utilization or storage of the vitamin A formed in the deficient animals, or both. This explanation is supported by results with rats (8), in which a higher dosage of vitamin A was required for liver deposition in deficient rats than was required to alter the blood level of the vitamin (9).

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