

strated in these studies that hesperidin alone reduces permeability of the connective tissue membrane. In view of the quoted evidence the possibility that hesperidin alters the absorption process by a direct effect on the membranes rather than by inhibition of hyaluronidase deserves serious consideration. Also, sight should not be lost of the possibility that hesperidin decreases the net absorption not by blocking nor diminishing the transfer of the fluid out of the peritoneal cavity, but by increasing the return flux from the circulatory system and tissue spaces into the peritoneal cavity.

Summary. 1. The effect of hyaluronidase and phosphorylated hesperidin on absorption of 0.9% NaCl solution from the peritoneal cavity of female rats was studied. 2. Intra-peritoneal administration of hyaluronidase increased and administration of phosphorylated hesperidin decreased the net fluid absorption. 3. Hyaluronidase abolished the effect of hes-

peridin when administered to hesperidin treated animals. 4. Adrenalectomy had no effect on the action of hesperidin. 5. The possibility that the hesperidin decreases the net absorption in untreated animals by direct effect on permeability of the membranes rather than by inhibition of hyaluronidase is discussed.

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Utilization of Vit. A and Carotene by Normal and Deficient Sheep.* (23819)

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For many years, the utilization of Vit. A and carotene has been extensively investigated in monogastric animals but to a limited degree in ruminants. It is well known that there is a marked specie difference regarding the relative values of carotene and types of Vit. A. Results recently reported(9) have indicated a depressed carotene utilization by the Vit. A deficient bovine. Furthermore, unpublished results from our laboratory have shown that the value of carotene for the bovine, as measured by hepatic Vit. A storage, was greatly influenced by the initial Vit. A content of the liver. Therefore, it was of interest to elucidate the suggested impairment of carotene utilization by the deficient animal as well as to establish relative biological values of types of Vit. A and carotene for sheep.

Method. Nineteen crossbred yearling wethers were divided into 2 groups of 9 and 10 animals each and pretreated with diets adequate and deficient in carotene respectively for 232 days. Four days prior to and during the experimental period all animals were fed a carotene deficient ration of barley straw and cottonseed meal. Aqueous solutions of beta carotene, Vit. A alcohol and Vit. A acetate were prepared, immediately preceding administration by the following method: One gram of the respective materials was dissolved in 20 ml chloroform; 25 ml Tween 80 were added and the chloroform was removed under vacuum at 62°C. Each remaining solution was diluted with distilled water to give a final concentration of 2 mg/ml. Trial A: Within 6 hours following liver biopsy, half of the normal and half of the deficient sheep were injected, intraruminally, with 0.6 mg of beta carotene/lb body weight. The re-

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TABLE 1. Hepatic Vit. A Storage in Normal and Deficient Sheep following Administration of Beta Carotene and Vit. A Alcohol and Acetate.

	Treatments		
	Beta carotene	Vit. A alcohol	Vit. A acetate
<i>Trial A</i>			
Normal			
No. of wethers	5	4	
Avg wt, lb	94.8	103.5	
*Avg hepatic vit. A:			
Initial content	34.82	41.08	
Final "	53.93	104.26	
Storage	19.11	63.18	
Deficient			
No. of wethers	5	5	
Avg wt, lb	88.1	84.4	
*Avg hepatic vit. A:			
Initial content	3.58	3.97	
Final "	5.57	13.19	
Storage	1.99	9.22	
<i>Trial B</i>			
No. of wethers		5	5
Avg wt, lb		84.6	81.1
*Avg hepatic vit. A:			
Initial content		9.44	9.75
Final "		28.00	17.78
Storage		18.56	8.03

* Expressed as μg vit. A/g fresh liver.

maining animals were administered with the same amount of Vit. A alcohol. Blood, for Vit. A determination, was withdrawn from the jugular vein of each animal just prior to carotene and Vit. A administration. Following treatment, blood samples were collected every 6 hours for 2 days and daily thereafter for 4 days. Liver samples were again obtained from all sheep 6 days following the carotene and Vit. A administration. Hepatic Vit. A storage, as influenced by treatment, was determined from differential liver analyses. Trial B: The 10 deficient wethers used in Trial A, in which half received beta carotene and half Vit. A alcohol, were allotted on the basis of hepatic Vit. A content into groups of 5 animals each. Vit. A alcohol was injected intraruminally at the rate of 0.6 mg/lb body weight to one group while, similarly, Vit. A acetate was administered to the second group. These injections were performed within 6 hours after the second biopsy of trial A. Blood was withdrawn for Vit. A determinations at 0, 12, 18, 24, 36, and 48 hours following treatment. Hepatic Vit. A

storage, resulting from the treatments, was determined from liver samples obtained 4.5 days following Vit. A administration. Plasma and liver Vit. A determinations were performed as described by Kimble(1) and Gallup and Hoefer(2) respectively.

Observations. Based on numerous studies with rats, the Expert Committee on Biological Standardization(3) reported that on an equal weight basis, beta carotene was equivalent to half the amount of Vit. A alcohol. However, Table I shows that normal wethers treated with Vit. A alcohol stored 3.3 times more hepatic Vit. A than similar animals treated with beta carotene.

Fig. 1 shows that Vit. A absorption commenced within 6 hours following Vit. A alcohol administration and attained maximum absorption at 12 hours. The Vit. A was not converted or absorbed from beta carotene until 18 hours, followed by a slower more sustained plasma Vit. A level as shown in Fig. 1. The difference in the rapidity of absorption may be accounted for by the possible difference in the intestinal site of Vit. A alcohol absorption and beta carotene conversion. Absorption of Vit. A alcohol has been shown to take place throughout the entire small intestine(4) whereas carotene possessed a maximum conversion in the middle of the intestine and no conversion anterior to the entrance of the bile duct(5).

Moore's(6) study indicated that rats were capable of storing only 200 i.u. of Vit. A daily from massive doses of carotene, while later work(7) showed that rats readily stored 20,-

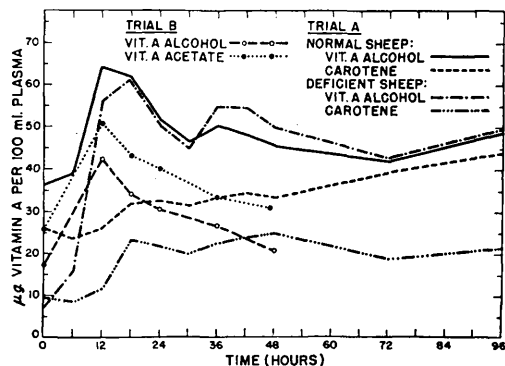


FIG. 1. Absorption rates of Vit. A as influenced by administration of beta carotene and Vit. A alcohol and acetate.

000 i.u. from a dietary Vit. A source. Therefore, wethers may perform similar to rats in that only a certain amount of carotene may be converted regardless of the magnitude of intake.

It has been shown by Lemley *et al.*(8) with rats that the efficiency of hepatic storage of Vit. A decreased with increasing levels of injected Vit. A. Therefore, a greater hepatic storage differential between beta carotene and Vit. A alcohol treatment may have existed if the efficiency of hepatic deposition had been in accord with the two respective levels and rates of absorption (Fig. 1).

The liver storage of Vit. A, that resulted from beta carotene and Vit. A alcohol administration in deficient wethers was markedly lower than in similarly treated normal animals. The increase of plasma Vit. A in the deficient wethers (Fig. 1), may account for the difference in the liver storage between the normal and deficient sheep treated with Vit. A alcohol. While the hepatic Vit. A storage was 6.8 times greater in normal than in deficient sheep treated with Vit. A alcohol, normal wethers, administered beta carotene, stored 9.1 times more hepatic Vit. A than carotene-treated deficient animals.

The decreased utilization of beta carotene by the deficient wethers was not only indicated by the low hepatic Vit. A storage but also by the low plasma Vit. A levels (Fig. 1). At no time during this study did the plasma Vit. A in the carotene deficient sheep equal the amount in normal animals. Since Vit. A alcohol absorption by deficient wethers was not depressed, these results indicate that Vit. A deficiency may possibly impair the rate of carotene conversion. Furthermore, the ratio of biological value of Vit. A alcohol to beta carotene was 3.3 in the normal animals and 4.6 in the deficient. These results are similar to those reported by Erwin *et al.*(9) where depressed carotene utilization was noted in Vit.

A deficient steers. While Week and Sevigne (10) reported higher absorption rates for the alcohol compared to the acetate form of Vit. A in humans, the same workers(11) found that Vit. A acetate was superior to Vit. A alcohol in rats.

Although Fig. 1 shows a very similar absorption rate for alcohol and acetate forms of Vit. A in wethers, the respective hepatic storage values shown in Table I differed during trial B. Hepatic storage of Vit. A was 2.3 times greater in sheep treated with Vit. A alcohol than those treated with Vit. A acetate.

Summary. Vit. A alcohol attained a maximum absorption at 12 hours post treatment, while beta carotene was absorbed at a slower more sustained rate. On the other hand, Vit. A alcohol and acetate were absorbed at comparable rates. The comparative value of Vit. A alcohol and beta carotene as measured by hepatic storage was markedly different for normal and Vit. A deficient sheep indicating that deficiency may impair carotene utilization.

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