

Influence of Dietary Vitamin E and Selenium on Distribution of Se^{75} In the Chick.* (28203)

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The discovery that selenium could prevent necrotic liver degeneration in rats(1) and exudative diathesis in chicks(2) considerably clarified the complex nature of a Vit. E deficiency in animals. However, the relationship between selenium and Vit. E is still not clear. Scott(3) concluded that no single metabolic function can account for the effects of Vit. E in preventing deficiency diseases in the chick. On the other hand, Zalkin *et al.* (4) have proposed that both Vit. E and selenium function as antioxidants by preventing lipid peroxidation in the tissues. Failure of selenium to prevent certain deficiency signs (such as encephalomalacia) was explained on the basis of lack of sufficient deposition of selenium in certain critical tissues.

In preliminary experiments in our laboratory, a higher level of selenium appeared to be present in the brain of chicks fed Vit. E than in birds fed no Vit. E(5). The estimate was based on a chick bioassay. The experiments reported here were undertaken to determine the effect of previous dietary history with respect to Vit. E and selenium on distribution of a radioselenium dose in the tissues of young chicks.

Methods. In Exp. 1, 2 groups of New Hampshire $\times$ Delaware cross-bred male chicks were reared in conventional battery brooders for 3 weeks. One group was fed a basal diet composed mainly of torula yeast and glucose monohydrate, similar to that used by Scott *et al.*(6). The second group was fed the same diet supplemented with 20 I.U. Vit. E (alpha-tocopherol acetate) per kg. Both diets were supplemented with 1 ppm selenium as selenious acid. At 3 weeks of age, 6 chicks, randomly selected from each treatment, were moved to an isolation room and placed in small wire-floored cages with space heaters. After 24 hours of adjustment

to the new quarters, each chick was injected intraperitoneally with a single 0.5 ml dose of $\text{H}_2\text{Se}^{75}\text{O}_3$ in neutral solution containing approximately 5 μc of radioselenium (specific activity, 357 mc/g) and 0.0264 mg of selenium. Two chicks from each treatment were randomly selected at 24, 48, 168 hours after injection for analysis of the tissues. A blood sample was obtained from each by heart puncture. The chicks were then killed and various tissues were sampled, weighed and ground with distilled water in a glass homogenizer. Three ml of the tissue-water homogenate were placed in a $1\frac{1}{4} \times 12\frac{1}{2}$ cm test tube and counted in a well detector with a 2" thallium activated sodium iodide crystal.

In Exp. 2, 2 groups of chicks were reared in conventional batteries for 2 weeks. One group was fed the basal diet as used in Exp. 1. The second group was fed the same diet minus the added selenium. No Vit. E was added to either diet. Four chicks from each treatment were randomly selected at 2 weeks of age and moved to the same isolation room as used in Exp. 1. After 24 hours an oral dose of selenium, containing 10 μc of Se^{75} (same specification as that used in Exp. 1) was administered to each chick. The dose was injected directly into the crop of the chicks by using a polyethylene tube extension on a syringe. The chicks were continued on the same diets as used during the pre-experimental period. Two chicks were randomly selected at 24 hours after dosing and the remaining 2 chicks were removed after 168 hours. Again blood was removed by heart puncture and several tissues were examined for radioactivity. The tissues were handled as in the first experiment. Bone (portion of cranium) and feathers were also measured for radioactivity, but were not homogenized.

* Scientific Paper No. 2318, Washington Agri. Exp. Station, Pullman. Project 1706.

In Exp. 2, total excreta during the first 24 hours after dosing and during the next

TABLE I. Effect of Dietary Vitamin E on Distribution of Injected Se⁷⁵ in the Tissues of Chicks.

Tissue	% of dose ($\times 10^6$)/100 mg tissue					
	24 hr	Basal 48 hr	168 hr	24 hr	Basal + Vit. E 48 hr	168 hr
Liver	38.5	23.2	6.7	46.0	30.9	10.0
Pancreas	60.8	30.9	6.8	90.0	31.8	6.0
Breast muscle	2.4	2.1	1.2	2.4	2.8	1.1
Cerebrum	5.7	4.1	1.9	3.1	3.3	2.1
Cerebellum	5.5	4.2	2.1	4.0	4.8	2.2
Whole blood	72.0	51.1	29.3	67.7	47.8	30.6
Plasma	52.5	33.5	7.6	55.6	38.8	9.6

144 hours were collected from each treatment and an aliquot of each was checked for radioactivity.

Results and discussion. The level of Vit. E in the diet before and after injection with radioselenium did not affect the relative distribution of the isotope in the tissues examined (Table I). Radioactivity in liver, whole blood, and particularly the pancreas, was high 24 hours after injection. The isotope disappeared from the liver, pancreas and blood plasma more rapidly than from the other tissues. For example, radioactivity remaining in the pancreas at 168 hours was less than 10% of that observed at 24 hours, whereas between 40 and 50% of the activity was still present in breast muscle, cerebrum, cerebellum and whole blood. Results indicate a significant transfer of Se⁷⁵ from the plasma to the cellular constituents of blood

during the 168 hours following injection.

Selenium in the diet before and after dosing markedly affected distribution and retention of Se⁷⁵ (Table II). In all tissues studied, the relative concentration both at 24 and 168 hours was higher for the birds receiving the basal diet not supplemented with selenium. Almost one-half of the oral dose was retained by chicks not fed selenium, whereas only $\frac{1}{5}$ was retained by chicks receiving 1 ppm selenium in the diet (Table III). Breast muscles, cerebrum, cerebellum,

TABLE III. Effect of Dietary Selenium on Retention of Oral Dose of Se⁷⁵.

Dietary Se	% initial activity		
	Excreted*	Retained*	
	0-24 hr	24-168 hr	for 168 hr
None	43.6	8.4	48.0
1 ppm	53.0	26.6	20.4

* Determined by measuring activity in combined feces and urine.

TABLE II. Effect of Dietary Selenium on Distribution of Se⁷⁵ in Tissues of Chicks Orally Dosed With Se⁷⁵.

Tissue	% of dose ($\times 10^5$)/100 mg tissue			
	Basal		Basal + Se	
	24 hr	168 hr	24 hr	168 hr
Liver	56.7	34.0	27.5	8.8
Pancreas	38.6	14.5	31.8	5.7
Breast muscle	3.8	4.7	2.4	.9
Cerebrum	9.0	15.8	3.6	2.5
Cerebellum	10.1	15.0	5.1	2.5
Whole blood	162.5	75.6	75.4	30.2
Plasma	103.0	26.2	63.6	5.5
Thymus	31.2	13.4	23.2	3.5
Leg muscle	5.9	5.3	3.3	1.1
Gizzard muscle	9.7	7.5	6.9	2.2
Heart muscle	24.2	16.2	13.0	3.4
Spleen	52.5	53.7	31.0	6.2
Lungs	21.5	15.3	12.2	3.2
Kidney	53.2	44.3	38.3	6.7
Testes	24.1	27.6	17.7	2.5
Bone	10.4	8.6	7.3	2.3
Feathers	16.8	12.1	3.8	4.7

spleen and testes from chicks not receiving dietary selenium had higher concentrations of the isotope at 7 days than at one day after administering the dose.

At 24 hours after dosing, there was a substantial deposition of the isotope in the cerebellum of chicks not fed selenium and the concentration increased by about 50% at 168 hours. Lesions associated with encephalomalacia in chicks are primarily located in the cerebellum. Vit. E and chemical antioxidants will prevent encephalomalacia but selenium will not(3). The observation here that selenium was deposited and retained in the cerebellum suggests that the non-effectiveness of selenium in preventing encephalomalacia is not due to a lack of deposition in

the brain. The proposal of Zalkin *et al.*(4) that the function of both Vit. E and selenium can be explained on a physiological anti-oxidant basis is not supported by these data.

Summary. Three-week-old chicks fed either a Vit. E deficient or supplemented diet were intraperitoneally injected with Se^{75} , and distribution of the isotope was determined in various tissues at 24, 48, and 168 hours after injection. Similarly, 2-week-old chicks fed a selenium deficient or supplemented diet were given an oral dose of Se^{75} and the distribution of the isotope in several tissues was determined at 24 and 168 hours after administration. Vitamin E had no effect on distribution of selenium, but selenium itself had a marked effect on the uptake and retention of radioselenium. One-half of the dose was retained by selenium-deficient chicks at 7 days, whereas only $\frac{1}{5}$ of the dose was re-

tained by chicks fed 1 ppm selenium from day-of-age. Considerable selenium was deposited and retained in the cerebellum of chicks even though this element cannot prevent encephalomalacia.

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Received February 1, 1963. P.S.E.B.M., 1963, v112.

Roentgenography of the Thoracic Duct in Man by Oral Administration of Contrast Media. (28204)

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Information concerning the role of the thoracic duct in disease states of man is almost completely lacking. The present level of knowledge of this structure is comparable to that of the biliary and gastrointestinal systems prior to introduction of biliary and gastrointestinal roentgenography. A convenient oral method for identifying the thoracic duct in an X-ray film should provide new insight not only into abnormalities of the thoracic duct and intestinal lymphatics but in disease elsewhere. Recent studies suggest, for example, that alterations in the thoracic duct occur in disorders of the liver and heart and are of considerable importance(1).

Although attempts to obtain X-ray studies of the thoracic duct have been described by

others using technics other than intralymphatic injection of contrast material, none have been successful(2,3). In this report a method for obtaining such studies is described. The technic is based on the consideration that adequate emulsification and dispersion of fat soluble contrast media must first be obtained *in vitro*.

Methods. The contrast material was freshly prepared for each patient by mixing 15 g of bile salts, 60 cc of polyoxyethylene (20) sorbitan monooleate (Tween 80), 50 cc of the iodinated propyl ester of poppyseed oil (Lipiodol) and 400 cc of water in a Waring Blendor for 15 minutes. This emulsion was fed to 5 patients through nasogastric tubes. Two and one half hours after the feeding tomograms of the chest were taken in the left lateral position at the level of the midline, 1 cm to the left of the midline and 1 cm to the right of the midline.

* U.S.P.H.S. Research Career Development Award. This study was supported by U.S.P.H.S. grant.

† Cooperation of Dr. Max Poppel in these studies is gratefully appreciated.