was the same at all times after intragastric or intravenous administration. These results suggest that the mechanism for water absorption *in vivo* is not analogous to *in vitro* conditions. The major route for transport from the small intestine is *via* the portal venous system and only about 3% is transported *via* the lymphatics.

Summary. When tritiated water was introduced into the lumen of the intestine of rats equilibrium with body water occurred in about an hour. Collections of absorbed tritium in the lymph and body water showed that 97% was absorbed into the portal system and only 3% into the mesenteric lymph.

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Effect of Dietary Selenium on Incidence of Nutritional Encephalomalacia in Chicks.* (29570)

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Extremely low levels of dietary selenium have been shown to ameliorate a variety of conditions in animals fed tocopherol-deficient diets. These include prevention of necrotic liver degeneration(1,2), and delay in onset of creatinuria in vitamin E-deficient rats(3). In tocopherol-deficient chicks, less than 0.1 ppm prevented exudative diathesis(4-6), while higher levels reduced the incidence of muscular dystrophy(4,7). On the other hand, selenium has been reported to be ineffective in preventing encephalomalacia(8-10). Nutritional encephalomalacia in chicks is unique in that it has been related specifically to ingestion of essential fatty acid sources(11-16), rather than to intake of polyunsaturated lipids in general. In the present study, an incidence of encephalomalacia of about 50% was obtained by feeding a diet containing 4% corn oil from which tocopherols had been removed. Under these conditions, a low level of selenium added to the diet was effective in delaying the onset of encephalomalacia.

Materials and methods. One-day-old Vantress-Arbor Acres crossbred cockerels were reared in electrically heated brooders and fed the diet described by Bosshardt *et al* (17), with slight modifications(12). Corn oil was stripped of most of the tocopherols by vacuum distillation† followed by aeration at 100°C until a peroxide number of 60 was reached. The diets containing corn oil at either 4% or 8% levels were isocalorically constant with respect to the protein, vitamins and minerals. Various levels of sodium selenite were added to the vitamin mixture when included in the diet. The corn oil and vitamin mixtures were added to stock mixtures

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† Distillation Products, Inc., Rochester, N. Y.

TABLE I. Effect of Added Selenium in Diet upon Incidence of Encephalomalacia.

Exp No.	Added selenium (ppm)*	4% Corn oil Incidence	8% Corn oil Incidence
1	.0	5/11	9/11
	.13	2/10	6/10
2	.0	14/20	19/21
	.13	5/21	13/20
3	.0	5/10	7/19
	.13	2/10	8/11
	.26	—	10/21
	.52	—	12/20
	.78	—	13/20
	1.56	—	7/10
4	.0	22/40	—
	.13	15/38	—
	.26	11/41	—
Total	.0	46/81	35/51
	.13	24/79†	27/41
	.26	11/41†	—

* Levels are ppm of selenium added as sodium selenite.
† $p < .005$.

2 or 3 times per week and kept refrigerated. Diets were changed in the feed pans daily to minimize autoxidation. Vitamins A (1250 IU) and D (180 IU) were given orally each week. Birds with gross signs of encephalomalacia were sacrificed and examined for evidence of hemorrhagic and necrotic lesions in the cerebellum. Incidence of encephalomalacia was evaluated up to 29 days.

Results. In each of 4 experiments, when a tocopherol-deficient diet containing 4% corn oil was fed to the chicks, addition of 0.13 ppm of selenium resulted in a lower incidence of encephalomalacia (Table I). Altogether, 24 out of 79 chicks developed encephalomalacia with 0.13 ppm selenium added to the diet as compared with 46 out of 81 with the basal diet. Addition of 0.26 ppm of selenium to the 4% corn oil diet instead of 0.13 ppm had virtually no further effect upon the incidence. When instead a diet containing 8% corn oil was fed, the added selenium had no significant effect upon the incidence of encephalomalacia, even when included in levels up to 1.56 ppm.

The results of adding selenium on the cumulative incidence of encephalomalacia in chicks fed a tocopherol-deficient diet containing 4% corn oil is shown in Fig. 1. A low level of added selenium delays the appearance of encephalomalacia but does not prevent it.

In addition, a group of chicks were fed a diet containing 2.5% of highly purified ethyl linoleate instead of corn oil. With 0.13 ppm of selenium added, 3 out of 8 birds had encephalomalacia, as compared with 6 out of 9, when no selenium was added.

The possibility that selenomethionine is formed and can act as an antioxidant was considered(18,19). A group of chicks were fed a 4% corn oil diet in which the 0.3% of methionine, which was normally added to the basic diet, was omitted. Under these conditions, 8 out of 20 chicks had encephalomalacia. Two out of 20 were noted when 0.13 ppm of selenium was included in the diet not supplemented with methionine.

Good growth was observed in all of the experimental groups. Surviving birds fed the corn oil diets weighed over 500 g and were usually over 600 g after 4 weeks. The groups without added methionine were somewhat lighter, averaging about 500 g. No differences in weight were obtained as a result of adding selenium to the diet.

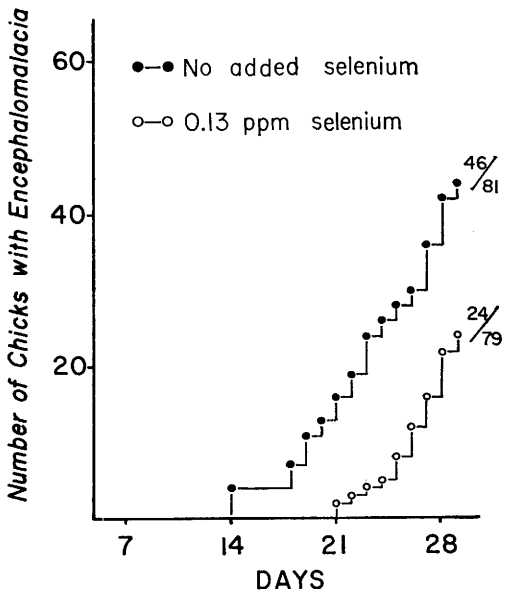

FIG. 1. Effect of adding 0.13 ppm selenium to a tocopherol-deficient diet containing 4% corn oil upon incidence of nutritional encephalomalacia in chicks.

Discussion. From these results the protective action of selenium against nutritional encephalomalacia was shown to be small but significant when the dietary lipid stresses were borderline. The failure of other investigators(8-10) to observe protection may have been due to their use of larger overwhelming levels of the stressor lipid, thereby masking whatever possible protection the added selenium might afford. In the present study, feeding an 8% corn oil diet also appeared to overwhelm the protective effect of selenium. Higher levels of selenium in the diet, up to 1.56 ppm, did not overcome the effect of feeding more corn oil.

In our studies, the omission of supplementary methionine from the diet did not prevent the effect of selenium in lowering the incidence of encephalomalacia. Possibly the present diet, which contained 18% casein and 10% gelatin, furnished enough methionine, although borderline, so that sufficient selenomethionine could be formed *in vivo* when selenium was added to the diet. The role of selenomethionine(18,19) as an *in vivo* antioxidant is not established.

Summary. Addition of 0.13 ppm of selenium as sodium selenite to a semisynthetic diet containing 4% corn oil freed of tocopherol reduced the incidence of encephalomalacia. On the other hand, when 8% corn oil was fed, selenium in levels up to 1.56 ppm had no effect on the proportion of chicks with encephalomalacia. Growth was not affected by addition of selenium.

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Penetration of the Placental Barrier by the Lactate Dehydrogenase-Elevating Virus (Riley) and its Behavior in Mouse Embryo Cultures Following Infection *in utero.* * (29571)

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There are contradictory findings on the behavior of Riley's lactate dehydrogenase elevating agent(1) (LDH agent) in tissue cultures. Although some authors have reported successful *in vitro* propagation(2,3,12), others have found the LDH agent to disappear from tissue cultures after a few days(4,5). In attempts to adapt the LDH agent to host cells

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