TABLE I.
Blood Vitamin A Absorption Curves in Infants with (Cases 5 to 8) and without (Cases 1 to 4) Eczema.

Case No.	Age (mo)	Sex	Diagnosis	Vit. A blood levels (blue units) in hrs 0	3	6	9	12
1	54	M	Neurodermatitis of popliteal spaces	5	250	200	100	25
2	3	F	Cleft palate	5	100	300	100	
3	18	M	Acute mastoiditis	25	100	30	30	
4	1	F	Impetigo contagiosa	5	75	50	75	5
Mean				10	131	145	76	15
5	14	M	Infantile eczema	13	25	50	40	10
6	11	M	Infantile eczema	11	66	41	10	5
7	7	F	Infantile eczema	8	12	19	16	7
8	5	M	Infantile eczema	5	50	25	12.5	5
Mean				9	38	34	20	7

It appeared possible that the respiratory infections and malnutrition could be accounted for on the basis of vitamin A deficiency resulting from a defect in intestinal absorption (frequently seen in patients with cystic fibrosis of the pancreas). Vitamin A absorption tests, therefore, were carried out in these 4 infants and in 4 control cases.

Methods. The procedures used were those described by Clausen and McCoord[1] and by May and McCreary.[2] Vitamin A blood levels were determined before and at stated intervals after the ingestion of oleum percomorphum. The dose of fish oil concentrate varied according to body weight (0.1 cc of oleum percomorphum representing 6,000 units of vitamin A per pound of body weight). The vitamin A content of the blood was estimated by a modification of the Price-Carr reaction as described by Clausen and McCoord.[1]

Results. Table I shows the results obtained. The vitamin A blood levels in the infants with eczema (cases 5 to 8) were relatively flat as compared to those obtained in the control cases (cases 1 to 4). The mean values for the two groups are indicated in the table and show the striking differences observed.

Conclusion. In 4 infants with intractable eczema associated with retarded development and malnutrition, the absorption of vitamin A from the intestinal tract was found to be impaired.

1 Clausen, S. W., and McCoord, A. B., *J. Pediatrics*, 1938, **13**, 635.

2 May, C. D., and McCreary, J. F., *J. Pediatrics*, 1941, **18**, 200.

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Lipids of Muscle and Brain in Rats Deprived of Tocopherol.

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The changes in chemical composition of tissues associated with vitamin E deficiency include alterations in the amounts of the lipid components. In rabbits with muscular dystrophy Morgulis, Wilder, Spencer, and Eppstein[1] found pronounced increases in the total lipids and especially in cholesterol, and the increasing proportion of cholesterol esters ap-

1 Morgulis, S., Wilder, V. M., Spencer, H. C., and Eppstein, S. H., *J. Biol. Chem.*, 1938, **124**, 755.

TABLE I.
Average Amounts of Lipid in Rat Muscle and Brain as Per Cent of Dry Substance.

Numbers and kinds of animal	Cholesterol				Cholesterol in total lipids,
	Free	Total	% free	Total lipids	%
Muscle					
Normal (12)	.29 (.23-.37)	.34 (.25-.42)	84	7.7 (2.9-15.4)	4.5
Control (7)	.22 (.20-.25)	.22 (.20-.25)	100	6.9 (5.7-8.6)	3.4
E-deficient:					
Moderate (7)	.35 (.26-.56)	.38 (.27-.57)	87	9.1 (7.6-11.6)	4.2
Severe (6)	.56 (.46-.79)	.55 (.48-.74)	100+	10.9 (8.4-15.3)	5.2
Brain					
Normal (15)	4.00 (1.83-7.96)	5.83 (4.54-8.10)	61	36.9 (27.6-47.2)	15.0
Control (7)	4.32 (2.86-6.99)	6.66 (5.43-8.33)	65	37.0 (29.9-47.8)	18.3
E-deficient:					
Moderate (8)	7.47 (6.14-8.18)	7.96 (6.88-10.20)	95	53.3 (44.4-61.1)	15.0
Severe (6)	7.01 (6.16-7.67)	7.51 (7.04-8.90)	93	26.3 (12.4-39.5)	35.1

peared to be a specific characteristic of dystrophic muscle. In nursling rats the muscular manifestations of vitamin E deficiency are equally acute but in half-grown and older rats they appear more gradually. In such animals creatine and chloride in muscle are altered as in the typical acute dystrophy of rabbits, and it was therefore of interest to know whether the lipid changes in muscle and brain of rats were similar to those that take place in rabbits.

The vitamin E deficient rats were maintained from weaning on the following diet: casein 18, sucrose 45.5, lard 22, salt mixture 2.5, yeast 10, and cod liver oil 2%. Control animals were fed the same diet, with wheat germ oil substituted for 5% of the lard, and normal animals from a stock diet of dog chow were also included. All of the animals were full-grown or nearly so; those that had been on the diet longest showed the most pronounced external signs of the deficiency.

The animals were killed with sodium amytal. The entire brain was mixed thoroughly. The right gastrocnemius muscle was minced in a tissue mincer;[2] minced muscle gave significantly higher cholesterol values than identical samples diced with scissors, probably due to more complete extraction. Part or all of the ground tissue was then transferred to weighed 10 ml glass-stoppered cylinders for drying and subsequent extraction, according to the method of Sturges and Knudson.[3] Free and total cholesterol were determined in duplicate on aliquot portions of the extract by the micromethod of Schoenheimer and Sperry.[4]* For precipitation of cholesterol, aqueous digitonin as here used was found to be more satisfactory than the alcoholic solution.[3,5] Occasionally, with either solution, there was incomplete precipitation or precipitation of interfering substances as evidenced by turbidity; results of analyses in which this occurred are omitted. Total lipids were determined by drying an aliquot of the extract at 90° to constant weight.

As seen in Table I, total and free cholesterol in muscle were significantly increased in dystrophy but the proportions were not greatly altered. Total lipids gradually increased as dystrophy progressed but not

2 Seevers, M. H., and Shideman, F. E., *Science*, 1941, **94**, 351.

3 Sturges, S., and Knudson, A., *J. Biol. Chem.*, 1938, **126**, 543.

4 Schoenheimer, R., and Sperry, W. M., *J. Biol. Chem.*, 1934, **106**, 745; Sperry, W. M., *Am. J. Clin. Path.*, Tech. Suppl., 1938, **2**, 91.

* We are indebted to Dr. Morgulis for the loan of the Wratten filter required.

5 Kelsey, F. E., *J. Biol. Chem.*, 1939, **127**, 15.

quite as rapidly as cholesterol, such that in severe dystrophy the proportion of cholesterol in the total lipids was slightly but perhaps not significantly increased. In brain tissue of dystrophic animals cholesterol, both free and total, markedly increased, especially the free—such that the proportion of cholesterol esters was insignificant. Total lipids increased in moderate dystrophy; they were subnormal in amount in severe dystrophy and the significance of the high proportion of cholesterol in total lipids is not understood. These results are in general similar to those obtained in rabbits,[1] with 2 or 3 exceptions. One of these is the marked decline in the total lipids in the brain of severely dystrophic rats just mentioned. The course of the disease in rats and in rabbits is dissimilar and the chronic condition in which rats, for months, miserably drag themselves around to secure food and drink, is very different from the complete prostration of rabbits. The brain tissue of normal and control rabbits contains practically no esterified cholesterol, whereas in normal and control rats, about a third of the total cholesterol was esterified. Finally, the differences in muscle cholesterol as between normal and control animals may be related to the high fat diet of the latter. Furthermore, some dog biscuits are not a rich source of vitamin E[6] and the control animals were more generously supplied with it than the normal animals. The significance of these lipid changes will not be understood until the role of vitamin E in oxidative processes[7] is clarified.

Summary. In rats on a vitamin E-deficient diet the cholesterol content of muscle was significantly increased, the total lipid less markedly. In brain tissue the cholesterol content was markedly increased, especially that of the free cholesterol, such that cholesterol esters represented about 5% of the total; in control animals this figure was 35%. These changes generally parallel those reported for rabbit tissue but are not as striking, perhaps because in grown rats dystrophy develops slowly. An understanding of these changes must await further knowledge of the functional activity of vitamin E.

6 Mason, K. E., *J. Nutrition*, 1942, **23**, 71.

7 Houchin, O. B., *J. Biol. Chem.*, 1942, **146**, 313; Kaunitz, H., and Pappenheimer, A. W., *Am. J. Physiol.*, 1943, **138**, 328.

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Occurrence of Premature Ovulation in the Domestic Fowl Following Administration of Progesterone.*

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During the course of tests undertaken to determine the possible value of steroids in controlling the release of pituitary gonadotrophins in fowl, it was unexpectedly found that progesterone, injected under appropriate conditions, regularly caused the premature ovulation of ovarian follicles in laying hens.

Progesterone is known to induce ovulation in amphibia,[1,2,3] but we are not aware that a similar result has been obtained previously either in birds or in normal mammals. Pearl and Surface[4] concluded that the administra-

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1 Shapiro, H. A., *Chem. Indus.*, 1936, **55**, 1031.

2 Zwarenstein, H., *Nature*, 1937, **139**, 112.

3 Langan, W. B., PROC. SOC. EXP. BIOL. AND MED., 1941, **47**, 59.

4 Pearl, R., and Surface, F. M., *J. Biol. Chem.*, 1914, **19**, 263.