other one in this group, became convulsive on the 77th day of the carp diet and was killed. The behavior of these 2 animals suggests the possibility that in some cats the anti-thiamine factor is either partially destroyed by intestinal action or the animals for some time obtain thiamine from a source beyond the reach of the anti-thiamine factor, either by coprophagy or refection of the sort known to occur in the rat.[4,5]

Six cats fed an exclusive diet of salt water herring also developed thiamine deficiency in exactly the same manner as those on carp. However, the cats fed perch, cat-fish, butterfish, or spots either maintained their usual weights or else gained during the course of the experiment. The feeding was discontinued after there was no longer any reasonable expectancy that thiamine deficiency would develop. These findings support the conclusion of Deutsch and Hasler[6] that the anti-thiamine factor is not found in all fishes.

Summary. Cats fed an exclusive diet of raw carp or raw herring develop all the signs of thiamine deficiency characteristic for this species.

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Influence of Pantothenic Acid Deficiency on Resistance of Mice to Experimental Poliomyelitis.*

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The relation of the nutritional status of a host to resistance in infectious diseases has received more detailed attention in recent years, largely because advances in the science of nutrition have made possible the study of the effects of an uncomplicated deficiency. Because of the intimate relation of the vitamins of the B-complex to cellular metabolism we have been investigating the influence of different levels of these vitamins on the incidence of experimental poliomyelitis. We[1] have previously reported the effect of thiamine deficiency on the resistance of Swiss mice to both Lansing strain poliomyelitis virus and Theiler's encephalomyelitis virus. In this paper similar studies are presented for pantothenic acid.

Experimental. Swiss mice (21-23 days old), bred in our laboratory, were used in all experiments and were kept in individual screen bottom cages supplied with a water bottle and an ointment jar containing the ration. Split litter technic, with consideration for sex and weight of mice, was employed. The synthetic pantothenic acid-low basal ration 503, employed in these studies, was composed of the following parts per 100: sucrose 73, vitamin-free casein 18, salt mixture 4,[2] and corn oil 5. The vitamin supplements per 100 grams of diet were: nicotinic acid 0.5 mg, riboflavin 0.3 mg, pyridoxine 0.3 mg, inositol 100 mg, para amino benzoic acid 100 mg, biotin (crude conc.) 0.01 mg, and choline 300 mg. This diet was supplemented with 0.05-0.1

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TABLE I.
Influence of the Level of Pantothenate Intake on Resistance of Mice to Poliomyelitis (Experiment 35).

No. of days after inoculation	No. of days on ration	% of mice paralyzed					
		Theilers G-D VII (0.1% conc.)			Lansing (5% conc.)		
		deficient (25)*	optimum (28)	high (18)	deficient (26)	optimum (25)	high (20)
6	20	8	18	6	27	32	40
7	20	32	32	33	35	40	45
8	20	40	57	44	38	40	55
9	20	48	86	72	42	48	60
10	20	48	93	88	46	52	60
11	20	68	96	94	46	52	65
14	20	72	100	94	50	60	70
28	20	72	100	94	54	64	70
		Theilers G-D VII (1%)					
		(10)	(18)	(10)			
7	36	0	6	20			
8	36	0	11	30			
9	36	9	22	40			
10	36	9	33	40			
11	36	9	50	50			
14	36	20	66	60			
28	36	30	72	70			

* Numerals in parentheses indicate number of mice inoculated.

ml of halibut liver oil per mouse per week, given *per os* twice weekly at the time of weighing.

On this diet, without the pantothenic acid, young mice gained slowly for about 2 weeks, then exhibited a definite weight plateau followed by gradual weight loss. The deficiency signs were a gradual loss of fur, apathy, and occasional dermatitis around the nose. Mice gained weight rapidly and appeared normal in every respect when fed ration 503 plus 2.5 mg calcium pantothenate per 100 g of diet (optimum level) or 503 plus 25 mg calcium pantothenate per 100 g of diet (high level).

The viruses employed, Lansing strain poliomyelitis and Theiler's encephalomyelitis (GD VII strain), were given by the intracerebral route using an inoculum of 0.03 ml, and in the concentrations indicated. Uninoculated control mice were maintained on the same rations to determine the effect of the diets alone, and to decide the optimum time for virus inoculation. The mice were observed twice daily after inoculation for signs of flaccid paralysis; the experiments were terminated 28 days after administration of the virus.

In Experiment 35, Table I, mice were inoculated with virus suspensions 20 days after being placed on the synthetic ration. The concentration of Theiler's virus was in this series 0.1% and Lansing 5%. The most striking differences in paralysis were observed on the tenth day following administration of virus. The results at this time in the deficient, optimum, and high diets were 12 of 25 mice (48%), 26 of 28 mice (93%), and 16 of 18 mice (88%) respectively. At the end of 28 days, 18 of 25 mice (72%) on the deficient ration, had developed Theiler's encephalomyelitis, while, of 28 mice on the optimum ration, 28 (100%) had developed the infection. The mice fed the diet high in calcium pantothenate showed a paralytic incidence of 94%. In the same experiment with Lansing strain poliomyelitis virus the results were as follows: in the deficient group 14 of 26 mice (54%) were paralyzed, in the optimum group 16 of 25 mice (64%) developed the disease, and 14 of 20 mice (70%) fed the ration containing a high level of calcium pantothenate were typically paralyzed.

The results of this experiment suggested that pantothenic acid played some role in the incidence of infection of mice to Theiler's virus, but that the vitamin had little effect when Lansing virus was employed.

In order to ascertain the effect of a prolonged pantothenic acid deficiency the remain-

TABLE II.
Influence of the Level of Pantothenate Intake on Resistance of Mice to Poliomyelitis (Experiment 37).†

No. of days after inoculation	% of mice paralyzed			
	Theilers G-D VII (10% conc.)		Lansing (5% conc.)	
	deficient (29)*	optimum (35)	deficient (28)	optimum (34)
6	3	6	0	9
7	7	26	7	9
8	31	49	14	12
9	48	69	18	15
10	52	80	25	15
11	59	91	36	18
14	59	100	43	24
19	59	100	50	38
28	59	100	50	41

* See footnote Table I.
† All mice inoculated 27 days after being placed on synthetic ration.

ing control mice from Experiment 35 were inoculated with a 1% suspension of Theiler's virus 36 days after being placed on the synthetic ration. Although the number of mice was small, the data supported our previous experience (Table I). Of 10 pantothenate-deficient mice only 3 (30%) were paralyzed at the end of 28 days, of 18 mice fed a diet optimum in pantothenic acid 13 (72%) developed the disease, while 7 of 10 mice (70%) on a high diet showed signs of infection.

In Experiment 37 (Table II), essentially a repetition of Experiment 35, the virus was administered 27 days after the basal ration was given. Both viruses were again used, the concentration of Theiler's virus being in this instance 10% and Lansing 5%. The results again indicated that the mice fed a ration devoid of calcium pantothenate and inoculated with Theiler's virus suffered a lower incidence of infection than those mice getting a diet optimum in pantothenate requirements. Seventeen of 29 mice (59%) on the deficient ration developed paralysis, while 35 of 35 mice (100%) fed the diet optimum in pantothenate showed signs of the disease. It should be noted that the virus suspension employed was 100 times and 10 times more concentrated respectively than those used in the first and second parts of the previous experiment. There was no significant difference in the incidence of paralysis when the Lansing virus was administered: 14 of 28 deficient mice (50%) were paralyzed, and 14 of 34 mice (41%) on the optimum ration developed experimental poliomyelitis.

Discussion. One might assume that an increased resistance to experimental virus infections would occur in nutritionally deficient animals on the rationale that if the virus is to be considered an intracellular parasite it would be entirely dependent on the cellular metabolism of the host. Any disturbance in the normal metabolism might therefore affect the virus contained in these cells. However, in the present experiments 2 viruses were employed each influencing nerve cells of the central nervous system. Yet the deficiency in pantothenate definitely altered the incidence of Theiler's infection and had little or no influence on the course of the disease following inoculation with Lansing virus. The possibility exists that in this deficiency there is a depletion of some metabolite necessary for the propagation of Theiler's virus but not for Lansing virus, or that there is an accumulation of a metabolite inhibitory to the former virus but not to the latter. Further studies are necessary in order to elucidate this aspect of the problem.

Conclusions. In 2 series involving a total of 348 Swiss mice, those fed a synthetic ration deficient only in calcium pantothenate exhibited a definite increased resistance to Theiler's encephalomyelitis, but little or none to the Lansing strain of poliomyelitis.