mal adult testis is the result of this fact. Whether this is the only factor involved, however, seems unlikely as one of us[7] has shown elsewhere that India ink spreads over a large area in castrated male rabbits than in normal males.

Summary. A study of the amount of spreading factor present in the undescended testes of young rabbits and of the cryptorchidized testes of mature rats showed that this factor was greatly reduced. From this we concluded that the spreading factor was related to spermatogenesis.

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Influence of Diet on Intoxication with Phenol and Cyanide.

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The influence of diet on resistance to disease and to various intoxications has been recently reviewed.[1, 2] The rôle of the vitamins has been extensively studied, but relatively little attention has been paid to the proportions of protein, carbohydrate and fat in the diet. From available reports it would appear that in some conditions one of these foodstuffs possesses an advantage, and in other conditions another. Many of these studies are of doubtful value since the vitamin factor was inadequately controlled. The present work was undertaken to compare the effects of diets high in protein, carbohydrate or fat on certain intoxications for which a definite detoxication mechanism is known. This report deals with 2 such poisons—phenol, which is detoxicated in part as an ethereal sulfate and in part in combination with glycuronic acid, and cyanide, which is in part detoxicated by conversion into thiocyanate.

Plan of Study Young rats from a mixed albino and hooded Norwegian colony, weighing 60 to 70 g, were placed in separate cages and given diets varying in their content of protein, carbohydrate and fat. Littermates were divided equally in the various experimental groups. After an interval varying from 10 to 24 days on the test diet they were injected subcutaneously with a 5% aqueous solution of phenol or a 0.1% solution of NaCN freshly made. The mortality

7 Sprunt, D. H., and McDearman, S., in preparation.

1 Clausen, *Physiol. Rev.*, 1934, **14**, 309.

2 Robertson, *Medicine*, 1934, **13**, 123.

of these animals was then compared and contrasted with similar animals on a stock diet. The composition of the experimental diet is given in Table I. The stock diet had the following composition: yellow ground corn, 57.0; whole milk powder, 25.0; linseed oil meal, 12.0; crude casein. 3.7; alfalfa meal, 1.5; NaCl, 0.4, and $CaCO_3$, 0.4. The mortality of 92 animals given phenol and 194 animals given cyanide is given in Tables II and III.

TABLE I.
Experimental Diets.

	High Protein	High Carbohydrate	High Fat
Casein	18.75	5.00	5.00
Dextrose	0.30	18.70	0.12
Olive oil	0.37	0.22	—
Lard	—	—	8.00
Halibut liver oil	0.30	0.30	0.30
Wheat germ oil	0.03	0.03	0.03
Brewer's yeast powder	1.25	1.25	1.25
Mineral mixture*	1.00	1.00	1.00

* This consisted of a slight modification of the Cox-Imboden salt mixture.[3] Its composition was as follows: $CaHPO_4$, 25 parts; NaCl, 18.4 parts; $MgSO_4$ (anhydrous), 6.86; KCl, 2.88; $KHCO_3$, 44.4; Fe citrate, 2.21; $CuSO_4$ 5 H_2O—0.24; $MgSO_4$ 7 H_2O—0.03; KI, 0.015; NaF, 0.03.

TABLE II.
Mortality of Rats Given Phenol.

Exp. No.	I	II	III
Days on experimental diet	16	23	24
Dose of phenol, g/kg	0.5	0.6	0.6
Mortality (deaths/total rats)			
High protein diet (avg wt = 100 g)	1/9	3/10	2/11
High carbohydrate diet (avg wt = 131 g)	3/10	5/9	5/11
High fat diet (avg wt = 129 g)	4/9	0/8	5/11
Stock diet (avg wt = 108 g)			4/10

TABLE III.
Mortality of Rats Given Cyanide (7 mg NaCN per kg).

Exp. No.	IV	V	VI		Mortality	Avg wt
Days on exp. diet	10	10	14	Total	%	g
Mortality (deaths/total rats)						
High protein diet	7/8	7/10	8/8	22/26	85	80
High carbohydrate diet	8/10	8/10	7/8	23/28	82	105
High fat diet	4/10	6/8	5/8	15/27	56	109
Stock diet		8/11		8/11	73	105
Exp. No.	VII	VIII	IX		Mortality	Avg wt
Days on exp. diet	18	20	22	Total	%	g
Mortality (deaths/total rats)						
High protein diet	0/8	6/8	6/9	11/25	44	98
High carbohydrate diet	3/5	5/10	6/10	14/25	56	118
High fat diet	0/8	2/10	1/9	3/27	11	114
Stock diet	2/9	5/10	5/0	12/25	48	106

[3] Cox and Imboden, PROC. SOC. EXP. BIOL. AND MED., 1936, **34**, 443.

Results. In the animals poisoned with phenol no consistent difference was noted between the fat and carbohydrate groups. The animals receiving protein showed a somewhat lower mortality. It is to be noted, however, that the animals fed high protein had not gained as much weight as the other groups, and, since the dose was based on body weight, these animals therefore received a somewhat smaller dose of phenol than the animals in the other groups of the same age.

With cyanide, we have tabulated separately the animals who were on the experimental diet only 10 to 14 days. Even in this group, however, it is noticeable that the mortality of animals on a high fat diet is distinctly less than on the high carbohydrate, high protein or stock diet. When the experimental diet was continued for 18 to 22 days this difference becomes very striking—the mortality in the high fat group being only 11% as contrasted with 56% for the high carbohydrate group and 44% for the high protein group. We have no explanation to offer for the marked protection against cyanide poisoning afforded by a high fat diet.

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Influence of Diet on Resistance to Diphtherial Toxin

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In order to investigate the influence of diet on the resistance of rats to diphtherial toxin, groups of rats were placed on diets rich in protein, carbohydrate, and fat respectively. The diets and conditions of the experiment were identical with those described in the preceding paper.[1] After 17 to 21 days on the experimental diet the rats were given a subcutaneous injection of diphtherial toxin* (7 m.l.d. per gram of rat). Relatively large doses of toxin are required to kill these highly resistant animals. According to Cobbet[2] and Ramon, *et al.*,[3] the rat requires from 1250 to 1750 times as much toxin per unit of animal weight as does the guinea pig. Preliminary experi-

[1] Meyer, A. R., PROC. SOC. EXP. BIOL. AND MED., 1939, **41**, 402.

* The concentrated toxin used in these experiments was supplied by E. R. Squibb and Sons, through the courtesy of Dr. G. W. Rake.

[2] Cobbet (1899), quoted by Pettit, *Ann. de l'Inst. Pasteur*, 1919, **28**, 663.

[3] Ramon, Noureddine, and Erber, *Compt. Rendu Soc. de Biol.*, 1928, **99**, 562.