the handicaps under which he has worked. Much of the money thus spent has been wasted, since much of the work of the part-time health officer is not in prevention of disease, but in cleaning up outbreaks of diseases.

It is found also that wherever a full-time, active, competent county health officer is appointed he lowers the infant mortality promptly and speedily accelerates the diminution of the death rate from tuberculosis. He engages in effective measures for the education of the public in health matters and generally succeeds in a striking manner in increasing the span of life of those who reside in the community which he serves.

At the present time 10 Kansas counties are operating full-time health units.

THE INFLUENCE OF VITAMIN DEFICIENCIES ON SUSCEPTIBILITY TO CERTAIN POISONS

By MAURICE I. SMITH, Pharmacologist, W. T. MCCLOSKY, Assistant Pharmacologist, and E. G. HENDRICK, Laboratory Assistant, Division of Pharmacology, Hygienic Laboratory, United States Public Health Service

In the course of some work on the relation of dietary deficiencies to tuberculosis resistance it was noted that vitamin A deficiency increased the susceptibility of the tubercle-infected white rat to the intraperitoneal injection of tuberculin (1). Briefly, it was found that while in the adequately nourished rat infected with the tubercle bacillus, tuberculin shock occurred only rarely following the intraperitoneal injection of old tuberculin, similar treatment of rats maintained on a diet deficient in vitamin A, though otherwise adequately constituted, resulted in a high percentage of fatal tuberculin shock. It was scarcely possible to offer an explanation for this phenomenon in view of our limited knowledge concerning the nature of the tuberculin reaction. In spite of the enormous amount of work on tuberculin hypersensitiveness in the experimentally infected animal, but little is as yet definitely known about its mechanism, beyond the fact that it is of a different order from general protein hypersensitiveness or anaphylaxis (2), (3), (4).

The suggestion had been made that the general tuberculin reaction in the tuberculous animal is due to the reaction of the hypersensitive tuberculous organism to toxic substances liberated within the tubercle, under the influence of parenterally introduced tuberculin. The experimental work of Krause (5), Selter and Turner (6), Assermann (7), and others would seem to furnish a basis for such an hypothesis. If this view can be accepted as correct, we would be forced to conclude that the tissues of the tubercle-infected rat, which, under normal conditions of nutrition, are quite resistant to

tubercle toxin, are rendered susceptible to this toxin when deprived of the fat soluble A food accessory.

The relatively high degree of resistance of the adequately nourished rat is not alone limited to tubercle toxin. It has long been known that this animal is but little susceptible to anaphylactic shock, which has been recently pointed out anew by Parker and Parker (8). Coca, Russel, and Baughman (9) found a high resistance in the rat to diphtheria toxin, and Voegtlin and Dyer (10) have found the rat highly resistant to traumatic shock and to the shock-producing poison histamine. The influence of vitamin deficiencies upon the natural resistance of the rat in the conditions enumerated is unknown, beyond our observation with regard to an increased susceptibility to tuberculin (1) and the recent statement by Werkman, Baldwin, and Nelson (11) to the effect that vitamin deficiencies decrease its resistance to diphtheria toxin.

It seemed that further useful information upon our problem at hand would be gained from a study such as we have undertaken here, viz, the alteration of normal resistance of the rat to certain well-defined pharmacologic agents brought about by means of vitamin-deficient diets.

There is another aspect that presents itself in connection with these studies. We believe that information on the behavior of certain poisons in the avitaminous organism, if altered in some definite manner through the deficiency, should throw some light on the nature of avitaminosis. While considerable data have accumulated in recent years on the pathology of avitaminosis, the problem of altered physiologic function of organs and tissues in the avitaminous organism has only begun to receive attention, and but little is as yet known of the mode of action of the food accessory substances in the animal body. The obvious difficulty that such studies present is the fact that in our present state of imperfect knowledge of the chemistry of the vitamins, observations on their physiologic or pharmacologic action can be only of an indirect nature. Nevertheless, some important contributions in this field have already been made. Thus Baldwin, Cook, and Nelson's (12) studies on the blood pressure of avitaminous rats indicate a markedly disturbed function of the cardiovascular apparatus caused by vitamin B deficiency, and to a lesser extent by vitamin A deficiency. This altered function of the cardiovascular apparatus appears to be beyond recognition by histologic or even electrocardiographic examination of the myocardium, as is shown in the work of Baude and Deglaud (13).

Van Leeuwen and Verzar (14) examined the reactions to some of the autonomic drugs, of tissues and organs in avitaminosis, and found no deviation from the normal. Their work, however, was limited to vitamin B deficiency, the experiments having been carried out for

the most part upon pigeons subsisting on polished rice, a diet which is, of course, deficient in many ways other than in vitamins.

More recently, Alpern (15) perfused the isolated wing of pigeons subsisting on polished rice and obtained a much-reduced reaction to epinephrine and $BaCl_2$ as compared with the normal. He correlates some of his findings with McCarrison's observation of suprarenal hypertrophy in vitamin B deficiency.

EXPERIMENTAL

The work reported herein has been carried out exclusively upon the albino rat, bred and raised in the laboratory under standard and uniform conditions. The diets employed in this study were as follows:

Substance	Adequate	A-deficient	B-deficient[1]
Casein [2]	18.0	18.0	18.0
Salt mixture 185 [3]	4.0	4.0	4.0
Dried brewers' yeast	5.0	5.0	0.0
Olive oil	8.0	10.0	8.0
Cod-liver oil	2.0	0.0	2.0
Starch	63.0	63.0	63.0
	100.0	100.0	100.0

[1] From some work on the nutritive properties of brewers' yeast which will be published shortly (Pub. Health Rep. (1926), 41, 201.—Ed.) it appears that dried brewers' yeast furnishes besides vitamin B another heretofore unrecognized dietary factor essential in the nutrition of the rat when maintained on a synthetic diet as used herein. The ration referred to as "B-deficient" is therefore deficient in this unrecognized factor as well as in vitamin B. Nevertheless, the term "B-deficient" is employed in conformity with common usage.

[2] Purified by the method of McCollum (16).

[3] Formula as given by McCollum and Davis (17).

The general plan followed has been that of restricting the animals to the respective diets from the time of weaning, which was usually at the age of about three to four weeks, and at a body weight of about 30 to 40 grams. The animals on the adequate diet gained at the rate of about 15 grams per week, and were used for the toxicity tests after being on the diet for four to six weeks.

The animals on the A-deficient diet usually continued to gain at a variable rate for four to six weeks, then began to decline. The animals of this group were not used for the toxicity tests until there was definite and permanent cessation of growth, readily recognizable eye lesions, and other general manifestations of vitamin A deficiency.

Because of the rapid deterioration of young animals on the B-deficient diet it was found feasible to allow them to gain a certain degree of maturity on the adequate diet, and then to be restricted to the B-deficient diet. Within four to six weeks on the B-deficient diet considerable decline in weight occurred, and symptoms of the deficiency were clearly manifest, at which time the animals were subjected to toxicity tests.

The details of the plan pursued in this work are further illustrated by the three accompanying typical charts, which are self-explanatory and require no further comment.

The toxicity tests were carried out upon the three groups of animals with a variety of pharmacological agents the actions of which are more or less well known. All the tests were carried out under identical conditions. The substances were always administered in aqueous solution, the dilutions being such that the total volume injected did not exceed 1 c. c., and usually not more than 0.5 c. c. All the injections were made slowly into one of the saphenous veins, no anesthetic being employed. It was sought to determine the maximum tolerated dose and the minimum lethal dose of a variety of substances in the three groups of animals in order to ascertain whether a deficiency in one or the other of the well-known food accessories would manifest itself in an altered susceptibility to some one chemical substance or group of chemical substances.

The substances used to determine whether vitamin deficiency resulted in an alteration of susceptibility included—

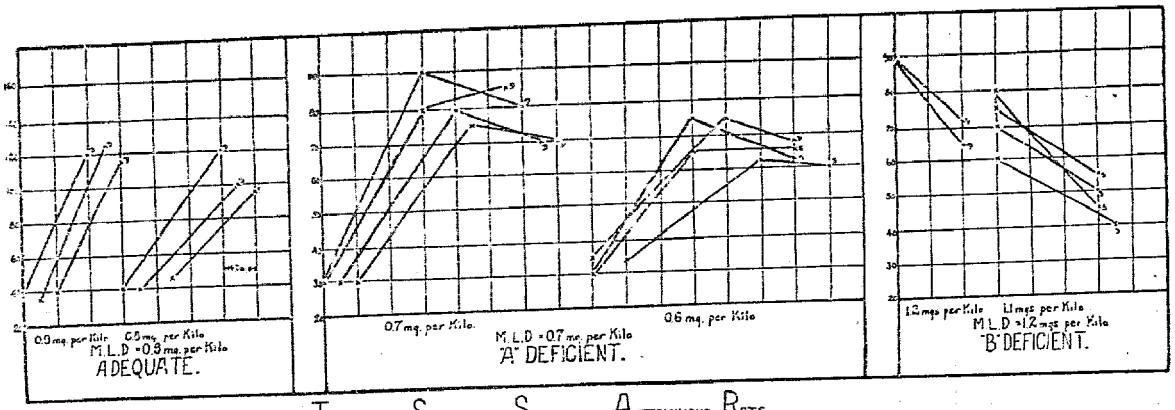
1. Central nervous system stimulants (strychnine, cocaine, atropine).
2. Central nervous system depressants (chloral hydrate, morphine).
3. Cardiac stimulants (crystalline strophanthin).
4. Autonomic drugs (atropine, pilocarpine, ergotoxine).
5. Capillary drugs and substances affecting cell permeability (histamine, pituitary principle, $CaCl_2$).
6. General protoplasmic poisons (quinine).
7. Miscellaneous (apomorphine, apocodeine, arsenic).

The results of this study are given in the following series of tables. The minimum lethal dose (M. L. D.) is the lowest dose which kills at least 50 per cent of the animals.

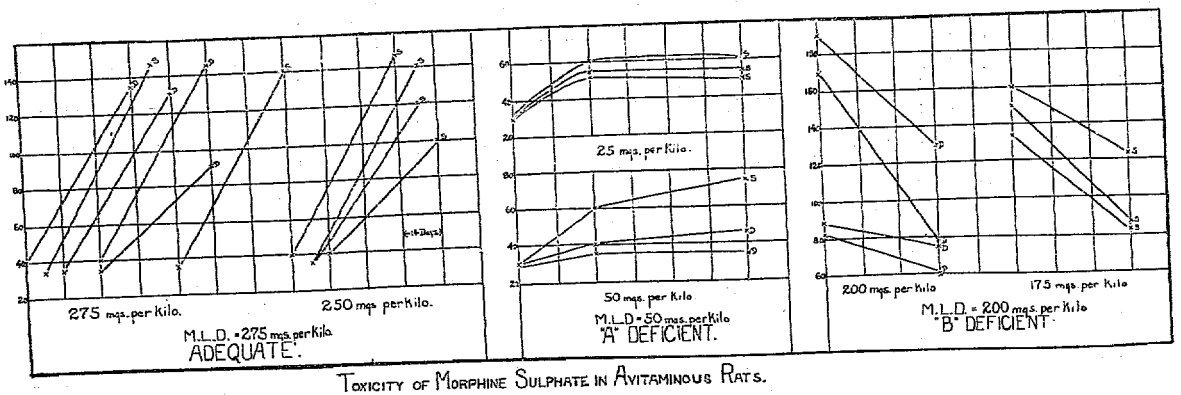
TABLE 1—*Toxicity of strychnine sulphate*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result [1]	Number of animals used	Result [1]	Number of animals used	Result [1]
1.2			2	++		+
1.1			3	−−−		
1.0	1	+	3	−−−		
0.9	3	+++			1	+
0.8	3	+−−				
0.7					4	++++
0.6					4	+−−−
M. L. D.	0.9 mg. per kilo		1.2 mg. per kilo		0.7 mg. per kilo.	

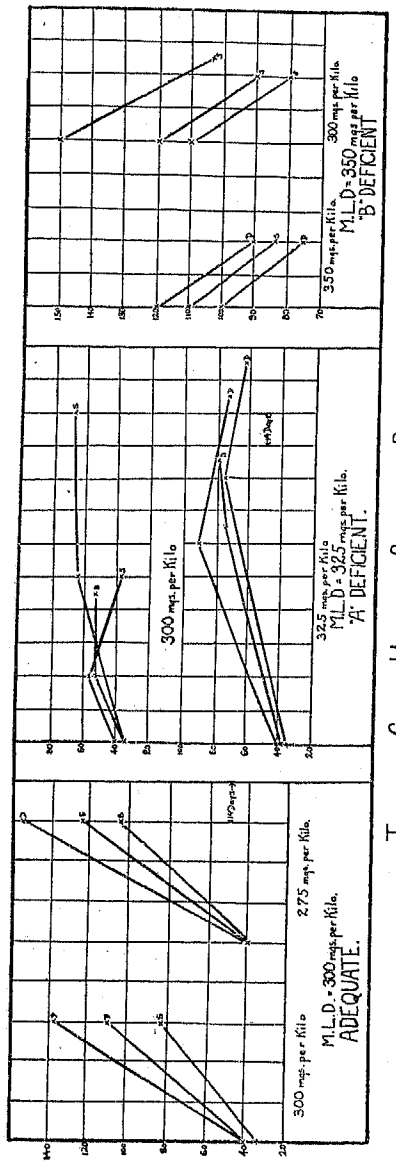
[1] + indicates death; − recovery.



TOXICITY OF STRYCHNINE SULPHATE IN AVITAMINOUS RATS.



TOXICITY OF MORPHINE SULPHATE IN AVITAMINOUS RATS.



TOXICITY OF CHLORAL HYDRATE IN AVITAMINOUS RATS.

TABLE 2.—*Toxicity of cocaine hydrochloride*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
15.0	3	+++	4	++++		
10.0	3	++−	3	−−−	2	++
8.0	4	−−−−	3	−−−	3	++−
6.0					3	−−−
4.0					3	−−−
M. L. D.	10.0 mg. per kilo		15.0 mg. per kilo		8.0 mg. per kilo	

TABLE 3.—*Toxicity of atropine sulphate*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
125.0	2	++	3	+++	2	++
100.0	3	+−−	3	+−−	2	++
75.0	4	+−−−	4	+−−−	3	++−
60.0					3	+−−
40.0					2	−−
M. L. D.	125 mg. per kilo		125 mg. per kilo		75 mg. per kilo	

TABLE 4.—*Toxicity of crystalline strophanthin (ouabaïn)*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
25.0			5	++++−		
20.0	2	++	6	++++−−		
18.0	6	++++−−	5	+++−−	3	+++
16.0	5	+++−−	3	−−−		
14.0	3	−−−			5	+++++
12.0					4	++++
10.0					6	++−−−−
8.0					5	+−−−−
M. L. D.	16.0 mg. per kilo		18.0 mg. per kilo		12.0 mg. per kilo	

TABLE 5.—*Toxicity of morphine sulphate*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
300	3	+++	3	+++	3	+++
275	6	++++−+	2	++	3	+++
250	4	−−−−	3	+++		
225	4	−−−+			2	++
200			4	++++	3	+++
175			3	−−−	3	+++
150			3	−−−		
100					3	+++
75					3	++−
50					3	++−
25					3	−−−
M. L. D.	275 mg. per kilo		200 mg. per kilo		50 mg. per kilo	

TABLE 6.—*Toxicity of chloral hydrate*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
350			3	++−	1	+
325					3	+++
300	3	++−	3	−−−	3	−−−
275	3	+−−	3	−−−	3	−−+
250	3	+−−				
225	3	−−−				
M. L. D.	360 mg. per kilo		350 mg. per kilo		325 mg. per kilo	

TABLE 7.—*Toxicity of pilocarpine hydrochloride*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
150	3	+++			3	+++
125	3	++−			3	+−−
100	3	−−+			3	−−−
75	3	−−−	3	++−		
50			5	+++−−		
40			4	−−−−		
M. L. D.	125 mg. per kilo		50 mg. per kilo		125 mg. per kilo	

TABLE 8.—*Toxicity of ergotoxine phosphate*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Results	Number of animals used	Results	Number of animals used	Results
40	3	++−				
30	3	−−−	1	+		
20	4	+−−−	4	+++−	3	++−
15	3	−−−	3	−−−	3	+++
10	4	−−−−	4	−−−−	3	++−
8					3	++−
6					3	++−
4					3	−−+
M. L. D.	40 mg. per kilo		20 mg. per kilo		6 mg. per kilo	

TABLE 9.—*Toxicity of histamine phosphate*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Results	Number of animals used	Results	Number of animals used	Results
600	5	+++−−	3	+++		
500	7	−−−++−−	4	+++−	4	+++−
400	4	−−−−	3	−−−	5	+++−−
300					4	−−−−
M. L. D.	600 mg. per kilo		500 mg. per kilo		400 mg. per kilo	

TABLE 10.—*Toxicity of pituitary active principle (standard infundibular powder (18))*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
200	3	+++	6	++++−−	3	++−
175			4	++−−		
150	3	−−−	6	++−−−−	4	+−−−
100	3	+−−	4	−−−−	4	+−−−
80	6	++−−−−			5	+−−−−
M. L. D.	200 mg. per kilo		200 mg. per kilo		200 mg. per kilo	

TABLE 11.—*Toxicity of calcium chloride*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
150			3	+++	3	++−
125	2	++	3	+++	3	++−
100	3	−−−	4	++−−	3	−−−
75	3	−−−	3	+−−		
M. L. D.	125 mg. per kilo		100 mg. per kilo		125 mg. per kilo	

TABLE 12.—*Toxicity of quinine dihydrochloride*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
75	2	++				
50	3	++−	4	++−−	3	++−
40	3	+−−	3	+−−	3	−−−
30	3	−−−	4	−−−−		
M. L. D.	50 mg. per kilo		50 mg. per kilo		50 mg. per kilo	

TABLE 13.—*Toxicity of arsenoxide* [1]

Dose, c. c. M /100 per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
10.0	7	+++++++	7	+++++++	6	++++++−
7.5	8	++++++++−	7	++++++−−	6	−−−−−−
5.0						
M. L. D.	7.5 c. c. per kilo [1]		7.5 c. c. per kilo		10.0 c. c. per kilo	

[1] This was a preparation made by Dr. J. M. Johnson in this laboratory. According to numerous experiments with this preparation by Miss H. Dyer of this laboratory, the M. L. D. for the normal rat is 7.5 to 10.0 c. c. M/100 per kilo.

TABLE 14.—*Toxicity of apomorphine hydrochloride*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
50	3	++−	2	++	3	+++
40	2	+−				
30	3	+−−	3	−−−	3	+−−
20					3	−−−
M. L. D.	50 mg. per kilo		50 mg. per kilo		50 mg. per kilo	

TABLE 15.—*Toxicity of apocodeine hydrochloride*

Dose, mg. per kilo	Adequate		−B		−A	
	Number of animals used	Result	Number of animals used	Result	Number of animals used	Result
20	2	++	3	+++	1	+
15	3	−−−	5	+−−−−	2	++
10	3	−−−	5	+−−−−	3	+−−
8					3	−−−
M. L. D.	20 mg. per kilo		20 mg. per kilo		15 mg. per kilo	

The results detailed in the foregoing tables may now be summarized so as to show the relative toxicity of the substances studied for the three groups of animals. If the susceptibility of the group of animals on the adequate diet to the several poisons examined be expressed as 100 per cent, then the relative susceptibilities of the two groups on the vitamin deficient diets may be expressed as follows:

Substance	Vitamin B deficient	Vitamin A deficient
Strychnine sulphate	75	130
Cocaine hydrochloride	66	125
Atropine sulphate	100	165
Ouabain	90	133
Morphine sulphate	137	550
Chloral hydrate	86	92
Pilocarpine hydrochloride	250	100
Ergotoxine phosphate	200	600
Histamine	120	150
Pituitary principle	100	100
Calcium chloride	125	100
Quinine dihydrochloride	100	100
Arsenoxide	100	75
Apomorphine hydrochloride	100	100
Apocodeine hydrochloride	100	133

In discussing these results we are fully aware of the fact, that, in some instances, the number of animals used for the determination of the minimum lethal dose is inadequate for arriving at anything but approximate figures. However, the purpose of the work was to

establish gross differences and not slight deviations from the normal. Keeping this fact in mind we believe that these figures clearly indicate an enormously increased susceptibility of the vitamin A deficient group to the alkaloids morphine and ergotoxine, and a greatly increased susceptibility of the B-deficient group to pilocarpine and to ergotoxine. It will be noted that central nervous system stimulants generally, such as strychnine, cocaine, and atropine, as well as ouabaïn and apocodeine, which also appear to produce in the rat, symptoms predominantly referable to the central nervous system, are all appreciably more toxic to the vitamin A deficient animal than to the adequately nourished control. The resistance of the vitamin B deficient animal to these poisons, on the other hand, seems to be either unchanged or actually somewhat increased. The other substances examined, with the exception of histamine, appear to affect alike the adequately nourished and the vitamin-deficient animals, histamine being definitely more toxic to the vitamin A deficient animal than to the control. The susceptibility of vitamin-deficient animals to apomorphine is unchanged, in spite of its close resemblance chemically to morphine.

DISCUSSION

If we attempt to classify the results obtained in this study on the basis of pharmacological action as related to altered susceptibility induced by vitamin deficiencies, we find that no generalizations are possible. Thus the two central nervous system depressants, morphine and chloral hydrate, show a wide difference in effects, vitamin A deficiency increasing the susceptibility of the animal to the one more than fivefold, but not at all to the other. On the other hand, the susceptibility to morphine and ergotoxine, two substances of widely different pharmacological action, is altered in nearly the same manner by this deficiency.

Examination of the influence of vitamin deficiency upon the toxicity of substances for which the rat normally enjoys a natural immunity shows that here too there is lack of uniformity. Thus, both deficiencies, and more especially vitamin A deficiency, increase the susceptibility of the experimental animal to histamine; and they are without appreciable effect upon susceptibility to pituitary active principle,[1] while ouabaïn toxicity is somewhat increased by A deficiency and diminished by B deficiency.

Do these experiments throw any light on the nature of vitamin action in the animal organism? The lowered blood pressure in avitaminosis noted by Baldwin, Cook, and Nelson (12) is ascribed by them to a weakened myocardium. The fact that neither vitamin A nor

[1] It should be added, however, that some recent observations on the toxicity of the active principle of pituitary on intravenous injection in laboratory animals indicate that the rabbit and cat are at least as tolerant as the rat (200 mgs. per kilo is tolerated by both species), and that the guinea pig apparently is the only animal showing a high susceptibility to this substance, 10 mg. per kilo being fatal.

vitamin B deficiency alters to any great extent the susceptibility of the experimental animal to either chloral hydrate or ouabaïn, the one a cardiac depressant, the other a stimulant of the myocardium, would indicate that the cause of the lowered blood pressure must be looked for elsewhere in the cardio-vascular apparatus. The greatly increased susceptibility to ergotoxine in the case of both deficiencies points to an altered function of the autonomic division of the central nervous system. The assumption that vitamin deficiencies damage the sympathetic mechanism controlling vascular tone would appear to explain the observed facts satisfactorily. The decreased resistance of the B-deficient animal to pilocarpine and that of the A-deficient animal to the several nerve poisons would indicate that the impairment of the nervous system, though perhaps most marked in the autonomic division, is more or less general. The greatly increased susceptibility of the A-deficient animal to morphine in particular suggests a much weakened respiratory center. Sluggish circulation and weakened respiratory center would account satisfactorily for the frequent occurrence of pulmonary congestion and lung disease in rats on vitamin A deficient diet.

If it were permissible to draw conclusions from reasoning by analogy we would venture to suggest that the action of tuberculin in the tuberculous organism is on the autonomic mechanism controlling cardio-vascular tone, and possibly to some extent also on the capillaries.

It is, of course, possible that the ability of the tissues of the vitamin-deficient animal to detoxify certain poisons may be reduced. This appears likely from a consideration of the relative toxicity of morphine and apomorphine in the avitaminous animal. Morphine is normally detoxified probably largely through oxidation. The indications are from some recent studies on the subject that cellular oxidation is reduced in avitaminosis (19), (20). We would reserve for future study the question of detoxification in avitaminosis.

SUMMARY AND CONCLUSIONS

A study was made of the toxicity of a number of pharmacologic agents in vitamin-deficient rats.

Increased susceptibility to pilocarpine and ergotoxine was observed in vitamin B deficient animals.

Rats on vitamin A deficient diet showed a much lowered resistance to ergotoxine and to morphine. Definite though slight, increase in susceptibility was also noted to histamine, ouabain, and to the alkaloids strychnine, atropine, cocaine, and apocodeine.

The bearing of these findings on the mechanism of vitamin action in the animal organism is discussed. A possible mode of action of tuberculin in the tuberculous animal is also pointed out.

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PUBLIC HEALTH ENGINEERING ABSTRACTS

Progress in the Purification of Water Supplies. Norman J. Howard, Bacteriologist in Charge of Water Purification, Toronto, Ontario. *Contract Record*, vol. 39, No. 52, December 30, 1925. Pages 133–138. (Abstracted by Rudolph E. Thompson.)

Progress in water purification during 1925 is reviewed, the phases of the subject dealt with being double filtration, slow sand and rapid sand filtration, sedimentation and coagulation, algal growths, pipe incrustation, softening, ultra-violet ray treatment, sodium iodide treatment and goiter, water standards, *B. coli* test, and removal of taste from chlorinated waters. The method of superchlorination and dechlorination has recently been experimented with at Toronto as a means of correcting the latter difficulty, and this process will be tried on a large scale in the near future. Employment of double filtration to cope with the ever increasing pollution is extending.

Relation Between Stream Pollution and Extent of Sewage Treatment Required. J. K. Hoskins. *American City*, vol. 34, No. 3, March, 1926. Pages 254–256. (Abstracted by H. N. Old.)

There is briefly discussed the relationship between stream pollution and sewage treatment in connection with public water supplies