growth characteristics of either the Ra or Rv variants of strain H_{37} *M. tuberculosis.*

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Nitrogen, Sulfur, Sodium, Potassium and Chloride Metabolism in Vitamin B_1 Deficient Rats.

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Metabolic studies in vitamin B_1 deficient animals have been largely limited to studies on carbohydrate metabolism and respiration of the central nervous system and other tissues.

To our knowledge no studies on the metabolism of the usual food elements and electrolytes have been made in vitamin B_1 deficient animals.

Twenty-four male and female albino rats (Wistar strain) were used in the experiments. They were kept in groups of 4 in metabolism cages, as previously described.[1] The diet consisted of purified casein 18%, Osborne-Mendel salt mixture 4%, butterfat 8%, cod liver oil 2%, corn starch 53%, and autoclaved baker's yeast 15%. In order to be able to differentiate clearly between the effects of vitamin B_1 deficiency and of inanition, paired feeding was resorted to. The food consumed by the controls consisted of the same ingredients as that given to the experimental animals, except that unautoclaved yeast was used. They received an amount of food corresponding to that eaten by the experimental animals during the preceding week. Distilled water *ad lib.* was provided from automatic glass fountains.

Urine and feces were collected twice a week. The urine was analyzed for total nitrogen (Kjeldahl), urea,[2] ammonia (Folin), uric acid,[3] creatine and creatinine,[4] total sulfur,[5] total and inorganic sulfates (Folin), sodium and potassium,[6] and chloride (modified Volhard-Harvey method). Feces were analyzed for total nitrogen, total sulfur,[7] and chloride (open Carius method). Food was analyzed

1 Sandberg, M., and Perla, D., *J. Exp. Med.*, 1934, **60**, 395.

2 Van Slyke, D. D., and Cullen, G. E., *J. Biol. Chem.*, 1914, **19**, 211.

3 Benedict, S. R., and Franke, E., *J. Biol. Chem.*, 1922, **52**, 387.

4 Folin, O., *J. Biol. Chem.*, 1914, **17**, 469.

5 Benedict, S. R., *J. Biol. Chem.*, 1909, **6**, 363.

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7 Neumann, A., and Meinertz, J., *Z. physiol. Chem.*, 1904-5, **43**, 37.

for total nitrogen, total sulfur, sodium, potassium and chloride. In order to conserve space condensed protocols for nitrogen and sulfur metabolism only will be presented, since there is no change in the excretion of sodium, potassium and chloride.

TABLE I.
Nitrogen Metabolism in Vitamin B_1 Deficient Rats and Paired Feeding Controls. Daily Average.

	Total N							% of total urinary N excretion
	Urine mg.	% of Intake	Feces mg.	% of Intake	Intake mg.	Retention, mg.	% of Intake	Urea N
Vitamin B_1 deficient rats								
Control period								
Oct. 6-Nov. 16	97	26	57	15	374	220	59	78
Deficiency period								
Nov. 17-Dec. 28	108	56	25	12	200	67	32	79
Paired feeding controls								
Control period								
Oct. 13-Nov. 23	71	19	47	12	364	246	69	74
Inanition period								
Nov. 24-Jan. 4	59	28	25	12	210	126	60	82

TABLE II.
Sulfur Metabolism in Vitamin B_1 Deficient Rats and Paired Feeding Controls. Daily Average.

	Total S						
	Urine mg.	% of Intake	Feces mg.	% of Intake	Intake mg.	Retention, mg.	% of Intake
Vitamin B_1 deficient rats							
Control period							
Oct. 6-Nov. 16	9.6	21	8.2	18	44.9	27.1	61
Deficiency period							
Nov. 17-Dec. 28	9.9	48	4.1	19	21.1	7.1	33
Paired feeding controls							
Control period							
Oct. 13-Nov. 23	6.7	15	6.9	16	43.3	29.7	69
Inanition period							
Nov. 24-Jan. 4	6.1	24	4.2	17	24.9	14.6	59

In vitamin B_1 deficient rats there is a disturbance in protein metabolism. Urinary nitrogen excretion increases in the vitamin B_1 deficient animals from 26% of the intake during the control period to 56% during the deficiency period. The paired feeding controls show an increased urinary nitrogen excretion rising from 19 to only 28% of the intake. Apparently, the increased urinary nitrogen excretion is only in part due to inanition, and to some extent to a specific factor in vitamin B_1 deficiency. The rise in nitrogen ex-

cretion is accounted for entirely by urea nitrogen, for there is no change in the excretion of ammonia nitrogen, creatinine, creatine, or uric acid. Fecal nitrogen excretion falls with the decreased intake in the vitamin B_1 deficient animals as well as in the paired feeding controls.

Total urinary sulfur excretion shows nearly the same absolute value during the deficiency as during the control period, but expressed in percent of intake it parallels the excretion of nitrogen, rising from 21 to 48%. There is hardly any change in the total sulfates as expressed in percent of the total urinary sulfur excretion. The rise in neutral and ethereal sulfur, indicating a disturbance in endogenous sulfur metabolism, is the same as that observed in the paired feeding controls. Fecal sulfur excretion drops with the decreased intake, but expressed in percent of intake it remains unchanged.

It is apparent that the only significant disturbance in metabolism in vitamin B_1 deficient animals is in the increased urinary nitrogen and sulfur excretion which exceeds, to a considerable extent, the increased excretion of nitrogen and sulfur observed in the paired feeding controls.

Though vitamin B_1 is significant in oxidative processes in cells of the central nervous system and possibly in cellular metabolism in general, its complete depletion is not associated with disturbances in the metabolism of most of the essential elements of the diet.

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Presence of an Antagonistic Factor in Serum of Dogs Following Repeated Injections of Cortin.*

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It has been reported[1] that in the normal dog a marked decrease in the excretion of Na and usually an increase in the excretion of K occurs during a 6-hour period following the intravenous injection of 20-40 cat units of cortin; and that with repeated injections (at

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[1] Hartman, F. A., Lewis, L. A., and Toby, G., *Science*, 1937, **86**, 128.