

as the blood of the restricted groups was either of the same or slightly higher concentration than the pathological groups.

All the lipemia could not be attributed to catabolism of body tissue because some of the mothers on a vitamin B deficient ration, owing to previous storage, did not decrease in body weight as much as their corresponding restricted controls.

For the past 4 years attempts were made to find a symptom complex in vitamin B deficiency produced in albino rats, as evidenced by the blood picture. The anhydremia and increase of non-sugar reducing substances in the blood were considered of interest but of relatively little clinical significance. The findings of a marked lipemia in this avitaminosis, reported in this communication, may prove of aid to the diagnostician in vitamin therapy.

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Glycogen Formation after Oral Administration of Sodium Salts of Propionic, Butyric, Valeric and Caproic Acids.

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By the use of Cori's method, the absorption of and the formation of glycogen from the salts of fatty acids of low molecular weight have been determined. Male rats previously fasted for 24 hours were used. The control and experimental animals although not litter mates were nearly of the same age and weight. The data in the table show that of the substances administered only sodium propionate affected the glycogen stores. The increments in liver glycogen resulting therefrom, although not large, are nevertheless definite, and are not to be ascribed to individual variations. The failure of butyric and caproic acids to form glycogen cannot be due to faulty absorption, since it was demonstrated that their salts were as well absorbed as sodium propionate. The data are sufficient to warrant the inclusion of propionic acid in that list of products that can be transformed into glycogen by the white rat. Butyric and caproic acids cannot be added to that list for under the conditions obtaining in this study no evidence of the formation of glycogen from these acids was secured. Further studies on the behavior of valeric acid are desirable, since it is possible that the dosage em-

ployed was too small. Until such studies are completed definite conclusions regarding the fate of this acid must be withheld.

TABLE I.

	No. of Animals	Liver Average %	Glycogen, entire body except liver Average %
Fasting controls	32	0.070 (0.036-0.093)*	0.067 (0.049-0.092)
Sodium propionate	21	0.313 (0.092-1.370)	0.077 (0.061-0.104)
" butyrate	5	0.063 (0.057-0.069)	0.054 (0.043-0.065)
" valerate	6	0.096 (0.075-0.122)	0.076 (0.064-0.091)
" caproate	8	0.084 (0.046-0.098)	0.074 (0.059-0.071)

* These are the ranges in the individual experiments.

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Results of Feeding Staphylococcus Filtrates to Monkeys.

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Attempts to produce in the lower animals symptoms resembling those in human volunteers fed with staphylococcus filtrates have not generally been successful. Barber stated that kittens, puppies and monkeys show no symptoms after ingesting large quantities of milk cultures of the toxic staphylococcus isolated by him: "or at all events, the symptoms are very slight." Unpublished work by Dack and others in the Bacteriology Laboratory of the University of Chicago has similarly given negative results. Rhesus monkeys of approximately 6 kilos have repeatedly been fed large amounts—up to 60-70 cc.—of staphylococcus filtrates of proved toxic power for man without in any instance producing the "food poisoning" symptoms observed in human volunteers. These consistently negative results led us to abandon for a time attempts to produce illness in monkeys by feeding toxic filtrates.

In March, 1931, opportunity was offered us through the courtesy of Dr. H. C. Clark, Director of Gorgas Memorial Laboratory, Panama, of repeating these experiments with a number of smaller monkeys of different species. The monkeys used were chiefly the red spi-