tract into rabbits in an attempt to produce an immune serum have failed to produce a serum which would neutralize the poison. The poison is not destroyed by freezing to —12° C. for 5 hours or by heating in a boiling water bath for 10 minutes. The extract does not produce hemolysis of human red cells or does it prevent coagulation of the blood. This is in agreement with the work reported by Yorke and Macfie on this mosquito. The extract is without effect when injected intracutaneously into guinea pigs. The nature of this toxic substance remains unknown (toxin, foreign protein, etc.) but will be the subject for future investigation.

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Phloridzin Diabetes and Vital Staining.

B. M. FRIED. (Introduced by Reid Hunt.)

From the Surgical Department of the Peter Bent Brigham Hospital, Boston.

Since the introduction in experimental pathology of vital staining of animals this method has been widely employed for the study of the physiology of the macrophage (Reticulo-Endothelial) system. In a number of instances this method has been used in conjunction with bacterial infections in the experiments with the so-called blockade of the reticulo-endothelial apparatus.

The investigation to be reported concerns itself with experiments on vital staining of animals treated with phloridzin. This substance leads to an outstanding glycosuria which, according to older observers is renal in origin. Present investigations have shown that beside the kidneys chronic phloridzin intoxication also leads to marked pathologic changes in other visceral and hematopoetic organs.

From a chemical standpoint, although the intimate mechanism of the glycosuria is not clear the loss of the function of carbohydrate oxidation is considered as being almost complete; and for practical purposes the disease is regarded as being close to diabetes mellitus (Nash[1]). It is obvious that the faulty metabolism of the carbohydrates carries with it an impairment in the combustion of fats and proteins.

Chronic phloridzin intoxication is therefore from histopathologic

[1] Nash, T. P., *Phys. Reviews*, 1927, vii, 385.

as well as from chemical standpoints a condition in which the entire metabolic apparatus is involved.

The purpose of this investigation was then to study the response of animals with this disease to vital staining.

Full grown rabbits were used in the experiments which were divided into 2 groups: 1, animals were primarily stained for a certain period of time and then were in addition "phloridzinized"; 2, animals were at first "phloridzinized" and this was later accompanied by vital staining. One per cent trypan blue diluted in physiologic sodium chloride was daily injected in the marginal ear of the rabbit's vein in the amount of 3 cc. per kilo body weight. One gm. of phloridzin Merck diluted in 1.2% of sodium carbonate or emulsified in 7 cc. of olive oil respectively was daily injected subcutaneously.

The diet of the rabbits consisted of oats and water.

Rabbits, unlike dogs, resist for a considerable length of time phloridzin intoxication: thus, some control animals received 30 gm. of the drug in a period of 30 days without any appreciable loss of weight. On the other hand, vital dye in reasonable concentrations can be administered to animals, almost indefinitely.

The present experiments have shown in the first place that when rabbits are treated simultaneously with phloridzin and vital dye they fail much more rapidly than those that receive only one substance.

Another point of interest is as follows: Older observers, and of recent investigators Fischler,[2] and Peskin[3] stated that phloridzin intoxication deprives the animal of its power to store glycogen (glycogenoprive Intoxikation). This is true when phloridzin is administered in sodium carbonate (1.2%). But when phloridzin is given in an emulsion with olive oil (1 gm. to about 7 cc. of oil) the power of the animal to store glycogen is retained, being almost equal to the normal control. But, here too, in animals sumultaneously treated with phloridzin in olive oil and vitally stained the hepatic cells show no or only slight traces of glycogen.

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[2] Fischler, F., *Beitz. z. Path. Anat. u. z. Allg. Path.*, 1927, lxxvii, 217.

[3] Peskin, A. R., *Biochem. Z.*, 1928, cii, 5.