8603 P

A New Preparation Protecting Against Rat Liver Necrosis.

J. C. FORBES AND R. C. NEALE. (Introduced by H. B. Haag.)
From the Department of Biochemistry, Medical College of Virginia, Richmond.

A preparation has been obtained from hog liver which exerts a marked protective action against liver necrosis produced in rats by carbon tetrachloride inhalation. Also preliminary experiments indicate that the substance definitely lessens the cirrhotic changes in the liver resulting from chronic carbon tetrachloride poisoning.

Preparation of material: To a concentrated aqueous extract of hog liver (about 29° Baumé), sufficient alcohol was added to make an 80% alcoholic solution and the solution filtered. The material precipitated from the filtrate by an excess of silver nitrate was treated with concentrated hydrochloric acid and the precipitated silver chloride removed. The supernatant fluid was neutralized until alkaline to congo red but acid to litmus, and evaporated under reduced pressure to a small volume. Inorganic salts were removed by the addition of alcohol to 80%, and filtration. The alcohol was evaporated off under reduced pressure and the residual liquid diluted with distilled water so that 1 cc. was equivalent to approximately 60 gm. of fresh liver.

The subcutaneous injection of this material into rats not only greatly reduced the degree of necrosis and fatty degeneration produced in acute poisoning, but also appears to speed up the regenerative processes. The mechanism of the protective action is not known but experiments show that the beneficial effect cannot be ascribed to the small amount of glucose present in the relatively impure preparation.

In the experiments with acute carbon tetrachloride poisoning, rats weighing approximately 125 gm. each were given daily subcutaneous injections of one cc. of the protective material, beginning 5 or 6 days prior to the day of acute poisoning. An equal number of rats were used as controls. Treated and control rats were subjected to inhalation of carbon tetrachloride. The rats were placed in a cage at the bottom of a large box, relatively air tight, with glass top and side. Carbon tetrachloride-laden air was forced into the box through a small opening near the top until the rats were semiconscious. They were kept in this state for periods varying from 2 to 2½ hours.

About 18 hours after the administration of the carbon tetra-

chloride, a control and a treated rat were killed. Thereafter, at least one control and one treated animal were killed each day over a period of 5 or 6 days. Immediately after the animals were killed, a portion of each liver was placed in formalin solution and later sectioned for microscopic studies.

Sections from the livers of the control rats showed extreme fatty degeneration and necrosis while the livers of the protected animals, with very few exceptions, were only slightly damaged. Even in these rats, the amount of degeneration did not approach that of the least damaged corresponding controls. Over 50 pairs of rats have been used in these acute poisoning experiments.

8604 C

Non-Effect of Estrogenic Hormones on Mammary Gland of Hypophysectomized Guinea Pig.*

E. T. GOMEZ AND C. W. TURNER.

From the Department of Dairy Husbandry, Missouri Agricultural Experiment Station.†

It was reported[1] that the mammary gland duct system of immature hypophysectomized albino rats failed to develop when dosages of 25 to 500 I. U. per day of the estrogenic hormone (progynon-B) were injected. To determine whether our results represented a species difference or applied generally, the study has been extended to other species, including the guinea pig, mouse, rabbit and cat. The present paper will present the observations with guinea pigs.

Since our work was begun two conflicting reports upon the subject have appeared. Nelson[2] reported mammary development in four male hypophysectomized guinea pigs, treated with 40 R. U. of oestrone daily, comparable to that observed in normal guinea pigs. It is not clear from his report whether he refers to the growth of an extended duct system or to a lobule-alveolar system, but as estrogenic hormone normally stimulates the growth of the lat-

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1 Reece, R. P., Turner, C. W., and Hill, R. T., PROC. SOC. EXP. BIOL. AND MED., 1936, **34**, 204.

2 Nelson, W. O., PROC. SOC. EXP. BIOL. AND MED., 1935, **33**, 222.