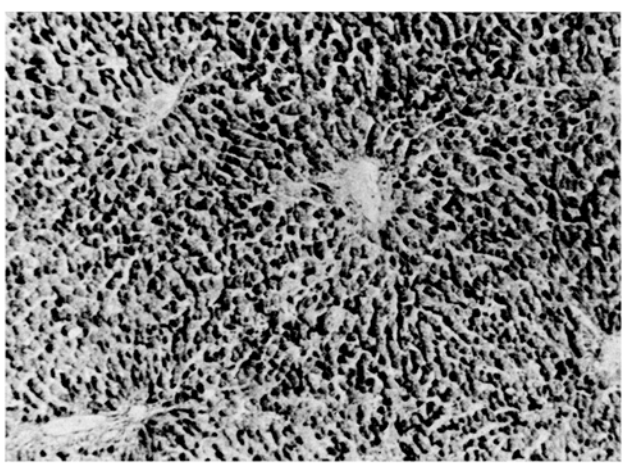
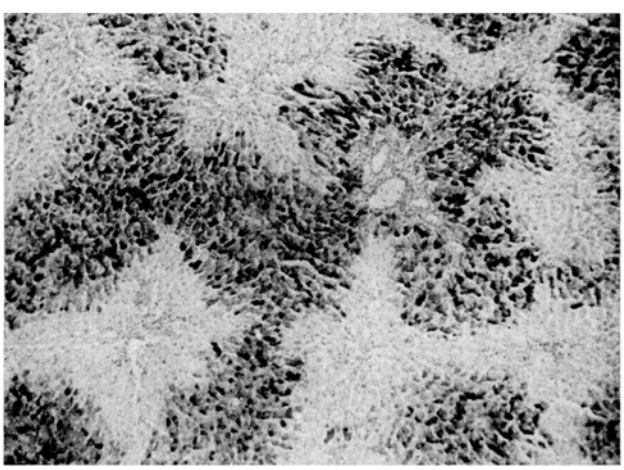
Effects of Hypophysectomy and Thyroidectomy on the Glycogen Depletion in Livers of Rats Treated with Thioacetamide

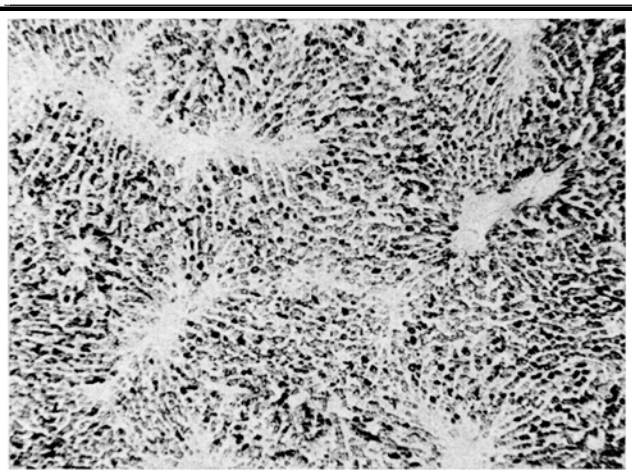
Hepatotoxic agents, when given to rats, produce changes in liver glycogen storage, which are more conspicuously

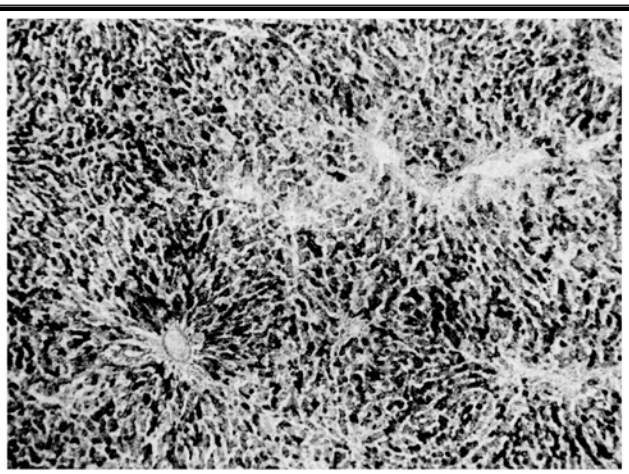
thioacetamide, carbon tetrachloride, DAB and 3-Me-DAB, all of which show cirrhogenic and tumorigenic activity in the liver.

It is a well-established fact that chronic terminal lesions (cirrhosis and tumor) induced in the liver of rats by a series of drugs are influenced by the animal's hormonal









Hypophysectomized and thyroidectomized male rats of the inbred August strain, weighing 200 g were treated for 15 days with daily injections of thioacetamide (10 mg/100 g of body weight). The animals were then killed and their livers removed for histological examination. Intact control animals were also treated as above and examined within the same period.

Figure 1 shows the uniform distribution of glycogen in the liver parenchyma of a normal, well fed rat. Figure 2 shows the typical glycogen distribution pattern in a TA-treated liver of an intact animal. There is complete disappearance of glycogen from the parenchymal cells around the central veins.

Hypophysectomized (Fig.3) or thyroidectomized (Fig.4) rats, when treated with TA for 15 days, failed to show centrolobular glycogen depletion.

These histological observations clearly indicate that changes in the animal hormonal imbalance influence not only the terminal stages of drug-induced liver lesions, as has been demonstrated by others, but also prevent the early manifestations of the treatment, like centrolobular glycogen depletion.

The fact that thyroidectomy alone can suppress the TA-glycogen effect seems to indicate that thyroxin is the key hormone involved in the complex mechanism responsible for such effect. Further investigations will attempt to clarify this point.

Zusammenfassung. Die Verarmung von Glykogen in der Leber als Ausdruck der toxischen Wirkung von Thioacetamid kann durch Hypophysektomie oder Thyroidektomie verhindert werden.

J. P. GUIMARÃES and M. C. SANTOS MOTA

Divisão de Patologia, Instituto Oswaldo Cruz, Rio de Janeiro (Brazil), January 30, 1961.

Leucine Aminopeptidase in the Foetal Kidney of the Rat

Leucine aminopeptidase is a widely distributed enzyme, taking part in protein degradation and perhaps also in synthesis. Its existence in large amounts in the kidney of numerous animal species has long been described. Histochemically it was localized by NACHLAS et al.[1] (1957) using *L*-leucyl-2-naphtylamide, and later (1960) using *L*-leucyl-4-methoxy-2-naphthylamide as substrate[2].

Leucine aminopeptidase of the adult rat kidney is noted to be most active in the juxtamedullary portion of the cortex, while the outer cortical zone is less active and the medulla is negative. The presence and distribution of leucine aminopeptidase in the foetal kidney of the rat had not been studied and was the object of the present investigation. The enzyme was demonstrated by the method of NACHLAS et al. as published in 1957. Altogether 30 kidneys of foetal and young rats were studied.

Figure 1 shows a section from the kidney of an 18-day embryo, the incubation time being 10 min. The cortex is still narrow and almost completely without enzymatic activity. Here and there, near the juxtamedullary portion, however, some groups of the tubuli have a faint enzymatic activity. Figure 2 shows a section from the kidney of a small rat, 4 days after delivery. Its enzymatic activity is clearly very much greater and localized over the greatest part of the narrow cortex. To make a comparison possible, Figure 3 shows a section from the kidney of a 2 months old rat after a similar staining procedure. It shows a high activity in the juxtamedullary part of the cortex and some staining also in the outer tubular portions of the cortex. The white spots represent the localization of the glomerula.

[1] M. M. NACHLAS, D. T. CRAWFORD, and A. M. SELIGMAN, J. Histochem. *5*, 264 (1957).

[2] M. M. NACHLAS, B. MONIS, D. ROSENBLATT, and A. M. SELIGMAN, J. biophysic. biochem. Cytol. *7*, 261 (1960).

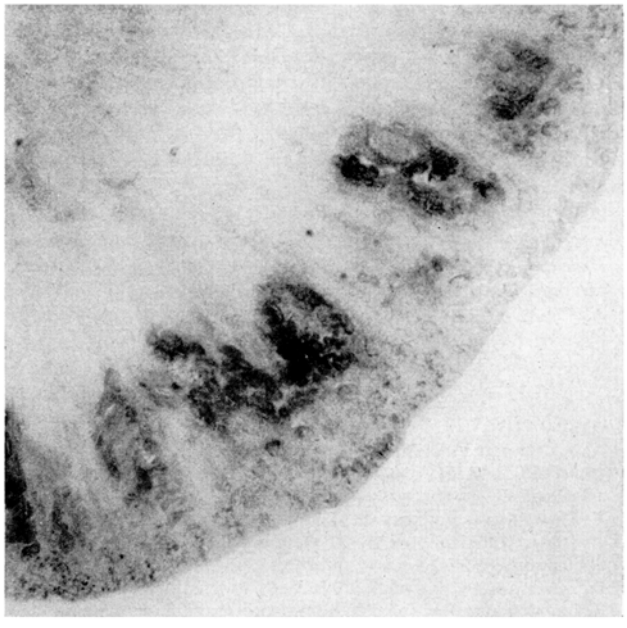


Fig. 1

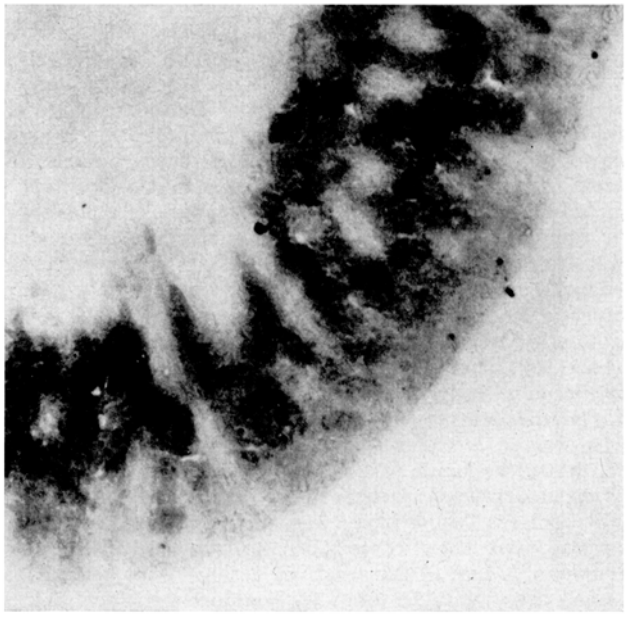


Fig. 2