of human tissues.

Summary. Autoradiography showed specific localization of As^{73} and As^{74} in adult mouse epidermis and skin adnexa. In tissue cultures embryonic mouse epidermis and chicken fibroblasts both absorbed the radioactive arsenic. Histochemical methods so far developed have evidently not accurately demonstrated the locations of arsenic in tissues.

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Time-Response Effect of Cortisone upon Liver Glycogen in the Rat. (20602)

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The acute glycogenic effect of cortisone is known. In the present experiments cortisone acetate was administered to force-fed rats for periods up to 6 weeks. The peak level of liver glycogen was reached within 5 days and thereafter began to decline but remained at higher-than-normal levels during the times studied here.

Methods. Male rats of the Sprague-Dawley strain, having an initial weight of approximately 300 g, were force-fed a medium carbohydrate diet(1). All of the animals were force-fed for 14 days prior to beginning the injection of cortisone acetate. Cortisone acetate (microcrystalline suspension, Upjohn) was administered subcutaneously each morning. At the end of the injection period each rat was fasted for 24 hours and anesthetized with cyclopal; the liver was rapidly excised, weighed and dropped into hot KOH. Liver glycogen was determined by the use of anthrone(2).

Experiments and results. The cortisone dosage was 2, 5 and 10 mg/rat/day. At each of the 3 dosage levels, 6 to 8 rats were killed at intervals of 1, 2, 3, 5, 7, 10, 14, 21, 28 and 42 days. Liver glycogen was determined in 8 untreated rats. Rats given 10 mg of cortisone daily developed generalized infection between 4 and 6 weeks. Four of these rats died and the remaining 3 were sick and unsuitable for the determination of liver glycogen. The average values are summarized in Fig. 1. The increase was proportional to the dose of cortisone acetate. The peak level was reached by 5 days at each of the 3 dosage levels. Thereafter, there was a decline in the amount of liver glycogen but the time-response curves (Fig. 1) did not approach the values for untreated animals during the periods studied here. The values were also calculated as total glycogen per rat and as per cent glycogen in liver. Although these values did not correlate exactly with those shown in Fig. 1, the shape of the curves remained essentially the same. The concentration of glycogen in liver reached 9.26% for the group treated with 10 mg of cortisone acetate daily for 5 days.

Discussion. The decline in the time-response curve showing the effect of cortisone upon the level of liver glycogen may represent

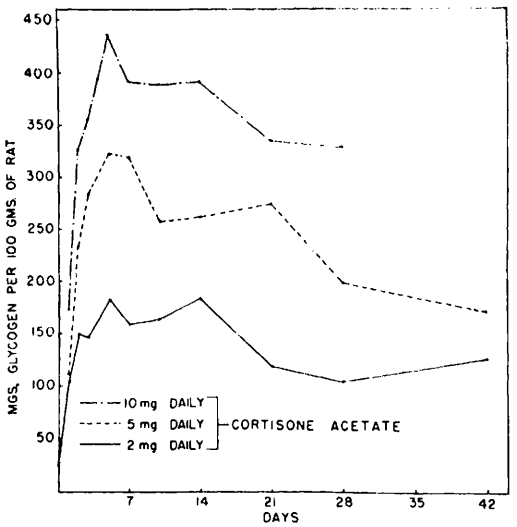

FIG. 1. Values for liver glycogen in rats treated with cortisone acetate. Each point in the curves represents avg of 6 to 8 rats. Expressed as mg of liver glycogen per 100 g of rat.

adaptation to hypercorticalism. There is a similar decline in the time-response curves showing the effect of high cortisone dosage upon urinary non-protein nitrogen and the glycosuria of rats having steroid diabetes induced by cortisone(3). This is not the only interpretation which can be made of the data. It was noted that the livers of these animals became progressively fatty especially at cortisone dosage of 5 and 10 mg daily. Baker *et al.* (4) studied similar animals having hypercorticalism induced by corticotropin and suggested that sufficient fat accumulated in the liver so that some of its functions might have been impaired. Partial impairment of hepatic participation in gluconeogenesis from protein might explain the reduction in the level of liver glycogen and also the decline in urinary nitrogen and glucose from their initial peak levels.

Summary. Male rats having an initial weight of approximately 300 g were given single daily injections of cortisone acetate in doses of 2, 5, and 10 mg/rat/day. At each of the 3 dosage levels 6 to 8 rats were killed at intervals ranging from 1 to 42 days and the amount of liver glycogen determined in each rat. The peak level was reached within 5 days and was followed by a significant decline towards, but not approaching, normal values.

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Single Dimension Chromatographic Separation of Thyroxin and Triiodothyronine.* (20603)

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The presence of the amino acid 3:5:3′ triiodothyronine in the plasma of normal and hyperthyroid subjects was recently demonstrated by Gross and Pitt-Rivers(1). These workers separated triiodothyronine from thyroxine by means of two dimentional chromatography. We have found this procedure time-consuming, and the spots produced tend to be diffuse and lack sharp definition. Described below is a method by which thyroxin‡ and triiodothyronine§ can be conveniently sepa-

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