TABLE I. Results of Inoculation of Chick Embryo Yolk Sac for the Demonstration of *M. tuberculosis.*

Type of specimen		No. tested	Acid fast bacilli in yolk sac Pos.	Neg.	Inoculation of yolk into guinea pigs Pos.	Neg.
Spinal fluid	Tests	13	13	0	13	0
	Controls	6	0	6	0	6
Sputum	Tests	7	7	0	7	0
	Controls	0				
Gastric washings	Tests	5	5	0	5	0
	Controls	2	0	2	0	2
Pleural fluid	Tests	6	6	0	6	0
	Controls	2	0	2	0	2
Peritoneal fluid	Tests	3	3	0	3	0
	Controls	0				
Pericardial "	Tests	1	1	0	1	0
	Controls	0				
Tissue	Tests	4	4	0	4	0
	Controls	1	0	1	0	1
Total tests		39	39	11		
Controls		11				

in cases of tuberculous meningitis, tuberculous pericardial, pleural, and peritoneal effusions. Limited experience thus far indicates its usefulness in following the effect of therapy in cases of tuberculous meningitis.

No attempts have as yet been made in this laboratory to determine whether or not the method can be adapted to assess the effect of therapeutic agents. It also may prove to be useful in determining the development of drugfastness of strains of *M. tuberculosis.*

These investigations are being continued in order to assess more accurately the usefulness of this method as a practical laboratory procedure for the more rapid etiological diagnosis of tuberculosis infections.

Conclusions. Inoculation of the yolk sac of 5- to 8-day embryonated eggs provides a rapid method for demonstrating the presence of *M. tuberculosis* in materials collected from patients with suspected tuberculous infections. Acid fast bacilli are readily demonstrated in the yolk of embryos 4 to 6 days after inoculation.

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Influence of Niacin and Priscoline on Ketonuria, Liver Glycogen and Liver Lipid.* (19702)

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It has been established that when niacin is given in relatively large amounts to diabetic rats or normal fasting rats a marked ketonuria is exhibited by many of the animals(1,2).

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TABLE I. Urinary Acetone Bodies, Liver Lipid and Glycogen in Rats Fasted for 5 Days. 12 animals in each group.

Treatment	Terminal wt, g	Urinary acetone bodies, mg/24 hr	Liver glycogen, mg %	Liver lipid, g %
Saline control	245	1.96 ± .5*	230 ± 35	7.71 ± .5
35 mg niacin daily	237	10.30 ± 1.9	463 ± 44	8.50 ± .4
20 mg priscoline daily	232	8.74 ± 2.7	90 ± 19	8.73 ± .8

* Stand. error.

Recently, it has been pointed out that priscoline also has a ketogenic effect in fasted rats (3). The question arises as to whether there may be alterations in liver fat or glycogen during this ketonuria. It has been shown that when an animal is in ketosis the liver usually has reduced glycogen levels(4), but Beringer(5) observed that the level of liver glycogen cannot always be correlated with the amount of ketone bodies formed. Also, total liver fat may be increased during a ketonuria but this is probably not obligatory(6,7).

In the present studies an attempt has been made to determine if there is any *correlation* between liver changes and the ketonuria induced by niacin or priscoline. This work has been briefly described in a preliminary note(3).

Methods. Thirty-six adult female rats of the Long-Evans strain were divided into 3 groups and fasted for 5 days. During the fasting period one group received daily intraperitoneal injections of 35 mg niacin in 7 cc physiological saline, another group 20 mg of priscoline in saline, while the third group was injected with saline alone. Urinary acetone bodies were determined for 24-hour samples on the third and fifth days of fasting by the gravimetric method of Van Slyke(8).

At the termination of the experiment the rats were anesthetized with sodium amytal and liver samples were taken for chemical and histochemical studies. Liver glycogen was determined by the method of Good, Kramer, and Somogyi(9) and histochemically by the periodic acid leuco-fuchsin method. Total liver lipid was determined by extracting homogenized tissue with alcohol-ether, evaporating the solvent and weighing the residue. Histochemically the lipid was demonstrated by an osmic acid procedure. The liver was fixed for 24 hours in Flemming's solution and then transferred to 2% osmic for 48 hours. This procedure more clearly delineated the fat than the Sudan-Black method. No counter stains were used.

Results. The saline injected group of rats excreted only small amounts of acetone bodies on the 5th day of their fast (Table I). The animals receiving niacin or priscoline, however, had increased levels of urinary ketone bodies. The priscoline group exhibited the largest variation in their ketogenic response.

There were quantitative differences in liver glycogen values in the 3 groups of rats. The glycogen values for the saline group were relatively high considering the animals had been fasted for 5 days. The niacin injected group, however, had higher levels and the priscoline injected much lower levels than the saline controls (Table I).

It was thus of interest to know what the histochemical preparations for liver glycogen would show as far as amount and distribution is concerned. The saline injected controls had normal glycogen distribution. Most of the glycogen was present in cells in the region of the central veins (Fig. 1) and with the exception of a few cells adjacent to portal triads, the rest of the liver was relatively free of glycogen. The liver glycogen of the priscoline injected rats was much less than seen in the controls and although the photomicrograph (Fig. 3) shows practically no glycogen, in some instances there was more than is represented in this picture. The niacin injected rats showed a remarkable amount of liver glycogen, similar to that found in non-fasting animals. Furthermore, the distribution of the glycogen droplets was somewhat different than that ordinarily observed since they were quite

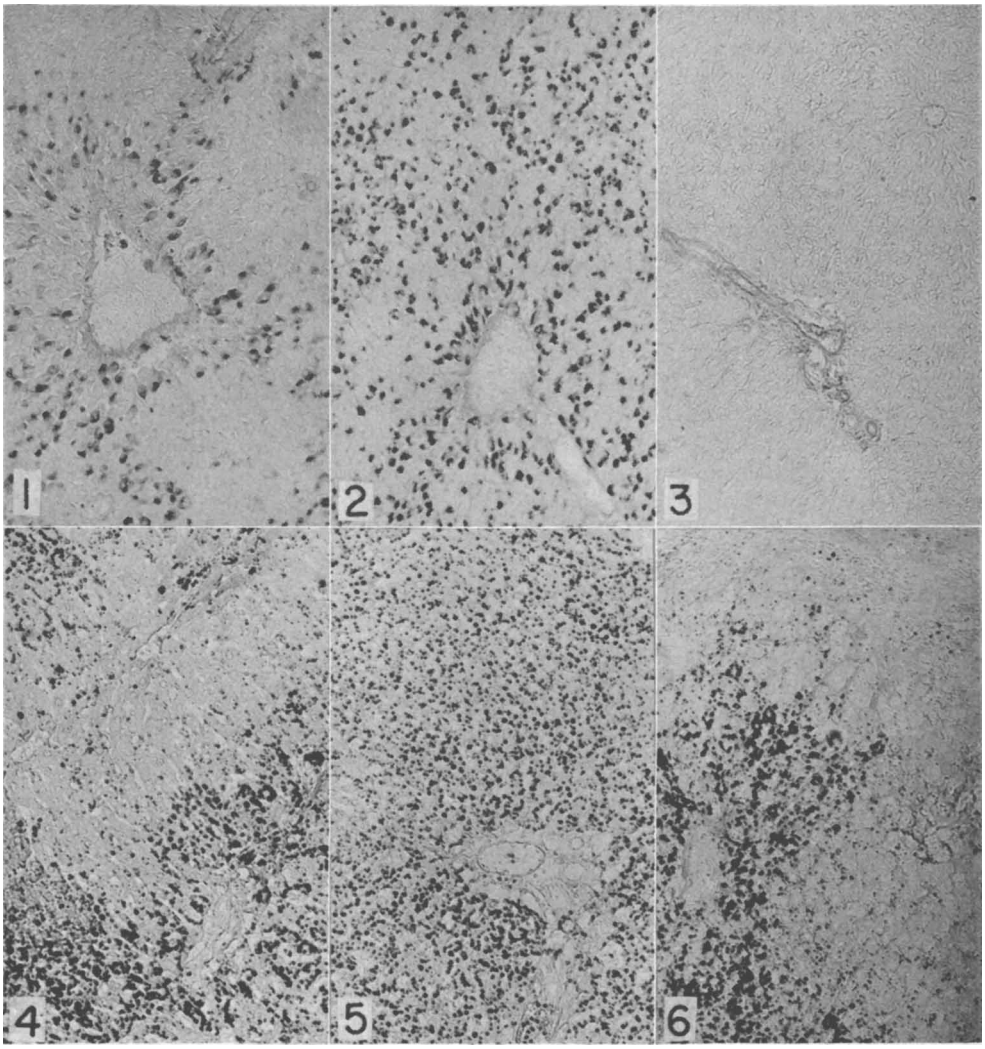

FIG. 1. Saline control, liver glycogen 154 mg %.
FIG. 2. Niacin injected, liver glycogen 560 mg %.
FIG. 3. Priscoline " " " 42 mg %.
FIG. 4. Same liver as Fig. 1, liver lipid 7.49 g %.
FIG. 5. " " " " 2, " " 7.00 g %.
FIG. 6. " " " " 3, " " 7.31 g %.
(All figures × 125.)

evenly distributed throughout the lobules (Fig. 2).

Because of the differences in the amount and distribution of liver glycogen in the 3 groups of rats, it was of particular interest to study the levels and distribution of liver fat. Chemically the amounts of total liver lipid were similar in the 3 groups of animals (Table I). Moreover, in the histochemical preparations the liver lipid in the saline and priscoline injected animals had a similar distribution. The lipid was present, chiefly in the region around the portal triads and consisted of small, medium and large intracellular droplets (Fig. 4, 6). The small droplets were generally present in the periphery of the liver cells. In the niacin injected animals the liver lipid was present as only small and medium droplets but the droplets were quite evenly dispersed over the whole liver section (Fig. 5).

In the liver preparations of each group of rats some nuclei contained osmiophilic material. Whether this was lipid is not clear because it was not possible to reproduce this picture with Sudan-Black stain. There was no microscopic evidence of liver injury in any of the animals.

The question arises as to whether there is any correlation between the described liver changes and the presence of increased levels of urinary acetone bodies. There apparently is no such correlation because a ketonuria is present, on the one hand, in niacin-injected rats which have high levels of liver glycogen and, on the other, in priscoline-injected rats which have low levels of liver glycogen. Evidence that priscoline modifies carbohydrate metabolism is brought forth by Sasson(10) who showed that the insulin requirements of some human diabetics is reduced following the administration of priscoline.

Summary. When large amounts of niacin or priscoline were given daily to rats fasted for 5 days, an excessive excretion of urinary acetone bodies was noted in most of the animals. Niacin-injected rats had higher levels of liver glycogen, priscoline-injected lower levels. Total liver lipid was not altered by either drug. There appeared to be no correlation between the amount of liver fat or liver glycogen and the presence of excess ketone bodies in the urine.

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Intramuscular Cortisone Administration to Dogs. (19703)

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Most of the experimental studies dealing with the effects of cortisone administration to dogs refer to profound changes in electrolyte and water metabolism, but there is little information available as to the glycemia and the behavior of the glucose tolerance test curves during and after prolonged injection with cortisone. Mushett, Porter, and Silber (1) reported that daily subcutaneous treatment of 3 dogs with 10 mg/kg for a period of 2-3 weeks brought about a profound polyuria and polydipsia. Davis, Bass, and Overman(2) in an extensive study were able to demonstrate the remarkable influence of cortisone on the ionic balance of normal and adrenalectomized dogs. The present study was originally an investigation of the influence of cortisone upon the carbohydrate metabolism in normal dogs. However, it was found that large doses of cortisone may produce a picture which resembles diabetes insipidus rather than diabetes mellitus.

Material and methods. Five mongrel dogs, 3 males and 2 females of a weight between 9 and 12 kg were used in this study. The animals were confined in metabolic cages and a measured amount of commercial dog food was fed. The amount of urine, the specific gravity and the amount of consumed drinking water were measured every morning. Urine