TABLE I.
Effect of Homologous and Heterologous Spleen Pulp on Mitosis in Intestinal Epithelium.

Group	No. mice	Mitotic counts: Group avg ± S.D.	Mitotic counts: Range
I. Control *dba*	5	1.34 ± 0.250	(0.86–2.10)
II. Control Rockland	5	1.33 ± 0.267	(0.68–1.80)
III. Rockland spleen in Rockland	4	1.23 ± 0.295	(0.90–2.04)
IV. Rockland spleen in *dba*	5	1.55 ± 0.350	(0.82–2.46)
V. *dba* spleen in *dba*	2	1.21 ± 0.236	(0.80–1.74)

significance level 0.1%).

Discussion. The stimulation of mitotic activity obtained with heterologous tissue in these experiments tends to confirm the results of Breuhaus and McJunkin(4) using intraperitoneal injections of macerated rat kidney in normal and unilaterally nephrectomized rats.

Our results would indicate that a foreign tissue can influence mitotic activity as judged by the reaction of the epithelium in the crypts of Lieberkühn in the small intestine of the mouse, whereas a homologous tissue may not do so, as illustrated by the lack of significance of the data from *dba* mice receiving *dba* tissue. The borderline significance level for homologous tissue in Rockland mice, however, may possibly be attributed to the fact that this is a heterozygous strain and there would be a greater degree of difference between tissues in animals of this stock than between those in animals of the highly inbred *dba* line 1 strain.

Summary. The subcutaneous injection of spleen pulp from heterozygous Rockland mice into homozygous *dba* line 1 mice caused a significant increase in the mitotic activity in the epithelium of the crypts of Lieberkühn, whereas the injection of the spleen pulp from *dba* mice into mice of the same strain was without significant effect, and the injection of Rockland spleen into Rockland mice caused a mitotic decrease of borderline significance.

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Choline Deficiency in the Hamster. (17502)

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In a previous communication(1) it was noted that there is a correlation between the occurrence of fatty livers in choline deficient animals and the ability of the livers of normal animals of a given species to oxidize choline. Thus, rats,(2), mice,(3) and dogs(4) develop fatty livers on choline deficient diets and their livers possess an active choline oxidase.(5) In contrast, the guinea pig does not develop a fatty liver while living on a choline deficient diet(1) and the livers of this species are devoid of choline oxidase activity.(5) In the present work it has been found that the liver of the normal hamster shows only moderate choline oxidase activity and that liver fat accumulation in this species is less than that

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4. Best, C. H., Ferguson, G. C., and Hershey, J. M., *J. Physiol.*, 1943, v79, 94.

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TABLE I.
Composition of Diets.

	1 %	2 %
Casein	10.0	6.0
Salts†	5.0	5.0
Lard	20.0	25.0
Cotton oil	10.0	5.0
Sucrose	38.0	13.5
Cod liver oil	1.0	1.0
Cystine	0.3	0.3
Cholesterol	0.5	0.5
Inositol	0.1	0.1
Glycocyamine	0.5	0.5
Peanut meal	—	30.0
Arginine	1.0	—
Liver "L"*	3.0	3.0

* Wilson's Liver Fraction "L" obtained through the courtesy of Dr. C. E. Graham.

in rats on identical choline deficient rations.

Experimental. Liver Fat Studies. The animals were Syrian golden hamsters from a local colony of stock originally obtained from the General Biological Supply House, Chicago, Ill. They were housed in individual metal cages and offered the experimental rations *ad libitum* with water supplied at all times. The feeding technic was identical with that previously described for guinea pigs.(1) The basal diets are summarized in Table I. Each diet contained, in addition, per kilo of diet, thiamine 10 mg, riboflavin 10 mg, pyridoxine 10 mg, calcium pantothenate 50 mg, niacin 100 mg, *p*-aminobenzoic acid 50 mg, biotin 2 mg, folic acid 5 mg, α-tocopherol 200 mg, 2-methyl-1, 4-naphthoquinone 10 mg. The feeding experiments were continued for 35 days. At this time the animals were sacrificed by decapitation and liver samples analyzed for total lipids by a method previously described.(6) The results of these experiments are summarized in Table II.

The failure to obtain fatty livers in the hamsters on choline-deficient diet 1 is referable to the apparent inadequacy of this "synthetic" ration for this species. Food consumption was lower than in the animals on diet 2 and during the course of the experiment there was a net loss in weight. Fatty livers are not to be expected under these circumstances despite the choline deficiency.(7-9) No explanation is at hand for the modest fat accumulation observed when diet 1 was supplemented with choline. In contrast, all hamsters ingesting diet 2, which contained alcohol-extracted peanut meal were found to possess fatty livers and in the group shown in the table the lipid content varied from 11.9 to 19.1%. In another group of 14 hamsters fed the same diet, liver fat was found to vary from 9.4 to 28.1% with a mean of 15.3%. In general, there was a positive correlation between food consumption, growth rate and liver fat.

TABLE II.
Liver Lipids of Hamsters and Rats on Choline-Deficient Diets.

Group	Species	No. animals	Diet	Initial wt, g	Wt changes, g	Food intake, g/day	Liver lipids % wet wt
1	Hamster	6	1	96	—12	4.8	6.9
2	"	6	1 + choline	93	— 7	5.2	9.4
3	Rat	6	1	100	23	6.1	13.9
4	"	5	1 + choline	97	25	6.3	4.9
5	Hamster	7	2	101	8	6.9	15.5
6	"	6	2 + choline*	102	7	6.7	7.5
7	"	7	2 + methionine†	97	7	7.1	5.7
8	Rat	6	2	103	17	6.8	24.7
9	"	5	2 + choline*				
10	"	6	2 + methionine†	99	23	6.9	7.6

* Present as 0.6% of diet.
† Present as 0.8% of diet.

Choline Oxidase Studies. The animals were sacrificed by decapitation; liver and kidney

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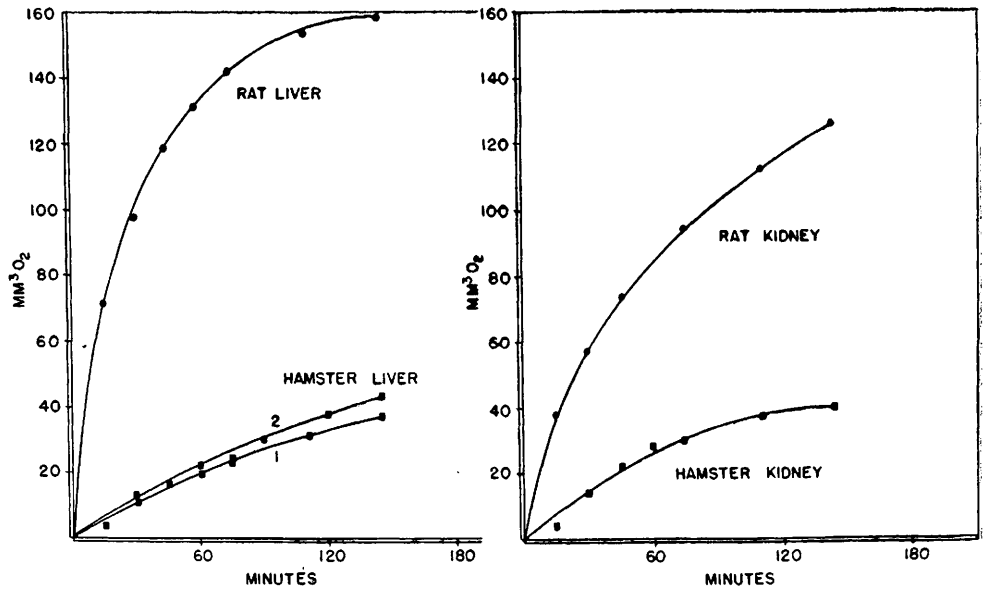

FIG. 1.

Choline oxidase activity of rat and hamster livers and kidneys. The substrate in each case was 1 mg of choline except for hamster liver where curves 1 and 2 represent the oxidation of 1 and 2 mg of choline, respectively. Theoretical oxygen uptake for 2 atoms of oxygen per choline molecule is 160 mm³.

were prepared by grinding in a mortar with sand and squeezing through muslin. Each Warburg vessel contained 0.5 ml of tissue suspension, diluted to 2 ml with M/20 phosphate buffer at pH 7.8. The choline chloride was dissolved in similar buffer and 1.0 mg of choline in 0.1 ml of buffer was added to the appropriate vessels at the expense of an equivalent volume of buffer. The vessels were then incubated at 37° in the conventional apparatus. The results are summarized in Fig. 1. In each case the control respiration in the absence of choline was subtracted before the data were plotted. It may be noteworthy that the basal respiration was of the same order of magnitude in rat and hamster. From the data shown in Fig. 1, it is apparent that hamster livers and kidneys do possess choline oxidase activity but this is markedly less than the activity of comparable rat tissues.

Because of the relatively poor growth and appetite of the hamsters on the peanut meal rations, as compared with the rats, it is not possible to determine whether the hamster is as susceptible to choline deficiency as the rat. It remains true, however, that, to date, the livers of all species which develop fatty livers on choline deficient rations have been found to possess choline oxidase activity in greater or lesser degree.

Summary. Young hamsters develop fatty livers when fed choline deficient rations containing peanut meal, although the mean increment in liver fat is only half that found in rats on the same diet. This may be correlated with the finding that hamster livers and kidneys possess markedly less choline oxidase activity than do the livers and kidneys of rats or may be due solely to the relatively poor appetite and growth of hamsters on these rations.

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