from human sources were studied in 3 types of tissue cultures. The virus recovery rates among fecal samples were of the same order regardless of the type of tissue culture used. Under the conditions of the study, monkey kidney epithelial cell cultures were superior to HeLa or testicular cultures in the primary isolation of poliomyelitis virus from oropharyngeal specimens. A comparison of tests with fecal samples in monkeys and in tissue culture indicated that the latter was as good as, if not better than, monkeys in detecting poliomyelitis virus.

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Effects of Dietary Aureomycin and Sulfasuxidine on Phosphorylation in the Liver of Rats.* (20998)

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Aureomycin has been shown to be a beneficial factor in the prevention of massive dietary necrosis in the rat(1). However, Lepper *et al.*(2) have reported that a reversible fatty infiltration of the liver frequently follows the administration of aureomycin intravenously or intraperitoneally in mice. The action of antibiotics in liver disease may be due to a direct chemical effect on the liver or to an antibacterial effect on intestinal flora. The sparing effect of antibiotics on the requirements of the rat for certain B vitamins has been shown to be mainly due to the alteration of intestinal flora(3). The supplementation of sulfasuxidine in a choline-deficient diet prevents cirrhosis(4). Both aureomycin and sulfasuxidine have been shown to delay the development of renal damage due to choline deficiency(5,6). However, no analyses were made on the phospholipide content of the liver following the administration of these antibiotics.

In view of the above findings, experiments were undertaken to ascertain if phospholipide

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metabolism was involved following the administration of aureomycin or sulfasuxidine.

Materials and methods. Male albino rats (100-110 g) were divided into 2 groups, with a control for each group maintained on a low protein-low fat(7) diet containing vitamin-free casein 5 parts, dextrin 42, sucrose 42, crisco 4, cod liver oil 1, salt mixture 4, Ruffex 2, plus the following vitamin mixture in mg per kilo of ration: dextrin 9639, thiamin 8, riboflavin 8, pyridoxine 8, nicotinic acid 241, calcium pantothenate 32, inositol 32, para-aminobenzoic acid 32. The rats of Group 1 received the low protein-low fat diet with and without supplementation of aureomycin (25 mg/rat/day for 1, 2, 4 and 8 weeks). The animals of Group 2 received the low protein-low fat diet with and without the supplementation of 5% sulfasuxidine for 5 and 7 weeks. Consumption weight of food was made daily and body weight was done every 2 days. Radioactive phosphorus was administered intraperitoneally as inorganic phosphate, containing 2-4 microcuries of P^{32}, and after 6 hours the animals were killed by decapitation. The liver was removed, rapidly weighed, and analyses made for acid soluble P, phospholipide P and nucleoprotein P and radioactivity as that previously described(8).

Results. The influence of antibiotics, aureomycin and sulfasuxidine, on lipide and nucleoprotein metabolism and phosphorus and nitrogen distribution in the liver are reported in Tables I and II. To evaluate the statistical significance of the results, the *t* test of significance(9) was applied to the differences between the mean and the results from each group. The data in Table I show that aureomycin stimulates the growth of rats maintained on a low protein-low fat diet and the *t* test showed the weight to be highly significant at 4 and 8 weeks with values of 3.7 and 6.2 respectively. The probability P for chance occurrence of these differences was <0.01. Concurrent with this increase in body weight, there was a significant increase in food consumption (*t* values of 10.9 at 4 weeks and 10.45 at 8 weeks, with a P value of <0.01). This finding is in agreement with Gyorgy *et al.*(1). However, there was very little change in the total liver lipides or the synthesis of phospholipide and nucleoprotein following the supplementation of aureomycin in the diet. Young growing rats have been shown to have an increased synthesis of phospholipides(10). The stimulation of growth following the administration of aureomycin failed to reflect an increase in the synthesis of phospholipides. After 4 weeks of supplementation of aureomycin, there was a significant drop in phospholipide synthesis when expressed as per gram of fat free tissue (*t* values of 2.99 with a P <0.01). This decrease in lipide phosphorylation may be due to a direct inhibitory effect of aureomycin (11). The ratio of the relative specific activities remained the same even if aureomycin is supplemented for as long as 8 weeks in the diet.

It is of interest that the ratio of synthesis of phospholipides to nucleoproteins is constant in the liver, irrespective of dietary fat or protein content, and is not influenced by the presence or absence of fat in the liver(12). Campbell and Kosterlitz(13) have demonstrated in the rat that the ratio of liver protein nitrogen to phospholipides remains approximately constant over various levels of dietary protein. This constant rate of synthesis of liver phospholipides appears to be changed very little by dietary intake, but may be influenced by the metabolic requirements of the body(4,14,15).

The supplementation of 5% sulfasuxidine to the low protein fat diet for 5 weeks inhibited the growth of the rats and there was a decrease in food consumption. There was a decrease in fat content of the liver following the administration of sulfasuxidine for 5 to 7 weeks. If the results were expressed as per gram of fat-free tissue, there was an increase in phospholipide synthesis. The stimulation of nucleoprotein P synthesis following the administration of sulfasuxidine is to the same extent as that of the phospholipides as shown by the ratios of the relative specific activities of the phospholipide P to that of the nucleoprotein P which is unchanged.

The mechanism of this lipotropic effect of sulfasuxidine is undetermined. The administration of 1 to 2% sulfaguanidine has been used to produce hyperplasia of the thyroid

TABLE I. Effect of Aureomycin (25 mg/Rat/Day) on Phosphorylation in Liver of Rats Maintained on Low Protein-Low Fat Diet.*

Supplement in diet	No. of rats	Body wt change, g	Food consumption, g/rat/day	Whole liver — Wt, g	Whole liver — Total lipides, g	Phospholipide P, RSA‡ /g fat free tissue	Nucleoprotein P, RSA	Phospholipide P / N	RSA‡ Phospholipide P / RSA Nucleoprotein P
					1 week				
	2	+10 ± 2	12 ± 0	4.69 ± .00	.54 ± .08	.182 ± .009	.044 ± .001	.081 ± .016	.031 ± .001	2.319 ± .406
A‡	6	+10 ± 2	13 ± 0	4.69 ± .54	.57 ± .10	.191 ± .020	.047 ± .010	.102 ± .014	.035 ± .001	1.893 ± .151
					2 weeks				
	6			5.24 ± .64	.98 ± .22	.162 ± .025	.038 ± .006	.076 ± .014	.034 ± .002	2.183 ± .343
A	6			4.88 ± .58	.63 ± .28	.157 ± .027	.037 ± .008	.066 ± .012	.037 ± .003	2.409 ± .356
					4 weeks				
	16	+17 ± 8	13 ± 1	6.98 ±1.58	1.29 ± .75	.234 ± .026	.043 ± .012	.092 ± .013	.030 ± .005	2.562 ± .294
A	24	+37 ± 7†	17 ± 1†	7.70 ± .876	1.00 ± .41	.218 ± .035	.033 ± .009†	.092 ± .014	.031 ± .005	2.388 ± .279
					8 weeks				
	12	+28 ±10	13 ± 1	7.60 ±2.27	1.82 ±1.30	.170 ± .040	.030 ± .008	.068 ± .02	.036 ± .003	2.559 ± .502
A	8	+59 ±16†	16 ± 1†	7.62 ± .71	.68 ± .14	.168 ± .019	.024 ± .004	.074 ± .013	.038 ± .003	2.299 ± .215

* Figures preceded by ± sign indicate stand. dev.
† The *t* test of significance was applied to difference between means and the P probability for chance occurrence of this difference was <0.01.
‡ A = Aureomycin; RSA = Relative specific activity.

TABLE II. Effect of 5% Sulfasuxidine on Phosphorylation in Liver of Rats Maintained on Low Protein-Low Fat Diet.*

Supplement in diet	No. of rats	Body wt change, g	Food consumption, g/rat/day	Whole liver — Wt, g	Whole liver — Total lipides, g	Phospholipide P, RSA‡ /g fat free tissue	Nucleoprotein P, RSA	Phospholipide P / N	RSA‡ Phospholipide P / RSA Nucleoprotein P
					5 weeks				
	16	+13.2± 8	11 ± 1	6.93 ±1.34	1.47 ± .79	.463 ± .190	.086 ± .038	.253 ± .094	.036 ± .016	2.084 ± .928
5% S‡	15	+14.5±12†	10 ± 1†	4.81 ± .91	.57 ± .33†	.566 ± .207	.135 ± .052†	.266 ± .108	.033 ± .014	2.410 ±1.74
					7 weeks				
	9	+ 2 ±18	11 ± 1	7.42 ±1.20	2.49 ± .84	.235 ± .027	.048 ± .007	.112 ± .011	.027 ± .002	2.107 ± .126
5% S	3	−35 ±17†	7 ± 0†	4.31 ± .77†	.47 ± .18†	.361 ± .073†	.094 ± .011†	.168 ± .015†	.026 ± .003	2.143 ± .317

* Figures preceded by ± sign indicate stand. dev.
† The *t* test of significance was applied to difference between means and the P probability for chance occurrence of this difference was <0.01.
‡ S = Sulfasuxidine; RSA = Relative specific activity.

(16). Compounds such as thiouracil, which produce hyperplasia of the thyroid, prevent experimental dietary cirrhosis(17). This lipotropic effect of sulfasuxidine apparently is not a thyroid mechanism, because hypothyroid conditions inhibit phosphorylation synthesis in the liver(8). Handler and Follis(4) concluded that the mechanism of preventing cirrhosis from the supplementation of 1% sulfasuxidine in choline deficient diet was not of thyroid or lipotropic effect as evidenced by histological changes in the thyroid and liver. The present observation of a lipotropic effect of sulfasuxidine is in agreement with its antihemorrhagic properties in choline deficiency(5), because most compounds which are lipotropic delay the development of renal damage(18).

Summary. 1. The dietary effect of antibiotics, aureomycin and sulfasuxidine, on phospholipide and nucleoprotein synthesis was studied in rat livers. 2. The addition of aureomycin (25 mg/rat/day for 4 and 8 weeks) to a low protein-low fat diet caused a marked stimulation of growth and food consumption in the rat. However, there were no significant changes in the liver lipides or the synthesis of phospholipides or nucleoproteins. 3. Addition of 5% sulfasuxidine to a low protein-low fat diet produced a significant lipotropic effect. There was a significant decrease in total lipides and an increase in lipide phosphorylation.

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