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Effect of Arsenicals on Liver Lipids of Rabbits.

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Attempts to determine the effect of liver injury on the amount and distribution of the liver lipids have yielded widely divergent results. Theis[1] found that the relation of phospholipid to neutral fat is quite constant for normal liver tissue and may be expressed as an equilibrium, 55 to 60% phospholipid: 45 to 40% neutral fat. However, if the liver is damaged or diseased this relation is altered. The abnormal organs seldom show any change from normal in the amount of total lipid, but the proportion of phospholipid is greatly diminished apparently because of a failure to convert neutral fat to phospholipid. Results obtained by MacLachlan[2] for white rats are at variance with those reported by Theis in two respects: (1) the proportion of total lipid present as phospholipid in normal liver tissue is considerably higher, and (2) no displacement of the normal phospholipid: neutral fat balance takes place as a result of liver injury. MacLachlan and Hodge[3] found in cocaine-fed mice which showed extensive liver injury that the neutral fat and cholesterol contents increase greatly but the phospholipid content remains strikingly constant. This clearly shows that a change in the phospholipid to neutral fat ratio of the liver lipids from normal may result from a change in the neutral fat content only.

Since arsenicals are capable of producing extensive necrosis of the liver with fatty degeneration, it was considered worthwhile to determine the effect of arsphenamine and neoarsphenamine poisoning on the amount and distribution of the liver lipids.

Fourteen young adult rabbits of both sexes were maintained on a diet of Purina rabbit chow for 2 weeks prior to the experiment. To each of 4 rabbits, 50 mg per kg of arsphenamine were administered intravenously every third day until 5 doses were given; to each of another 2 animals, 5 doses of 75 mg per kg of neoarsphenamine were administered similarly. Thus the rabbits received a total of 250 mg per kg of arsphenamine or 375 mg per kg of neoarsphenamine within 2 weeks. The remaining 8 animals served as controls.

1 Theis, E. R., *J. Biol. Chem.*, 1928, **76**, 107; 1928, **77**, 75; 1929, **82**, 327.

2 MacLachlan, P. L., Proc. Soc. Exp. Biol. and Med., 1936, **34**, 31.

3 MacLachlan, P. L., and Hodge, H. C., *J. Biol. Chem.*, 1939, **127**, 721.

A small portion of each liver was used for moisture determination. Lipid analyses were made on another portion by standard procedures, Bloor[4] and Boyd.[5]

Histological examination of the livers showed moderate to severe necrosis with fatty degeneration as a result of the arsenical treatment. However, the results of the chemical analyses of the livers (Table I) show that there are no significant changes from normal following the administration of either arsphenamine or neoarsphenamine. The neutral fat content of the treated animals, while showing more individual variation, is no greater in amount than that of the untreated animals. The constancy of the phospholipid content, moreover, does not support the idea that in liver injury neutral fat increases at the expense of phospholipid. The normal values obtained for the total lipid content and the ratio of phospholipid to neutral fat of the livers of rabbits following arsenical poisoning are in agreement with the observations of MacLachlan[2] for rats following liver injury, but stand in marked contrast to the findings of MacLachlan and Hodge[3] for cocaine-fed mice. Apparently a change in the phospholipid : neutral fat balance of the liver

TABLE I.
Liver Lipids of Rabbits Following Administration of Arsphenamine and Neoarsphenamine. (Calculated on the basis of moist weight.)

Rabbit No.*	Moisture, %	Total Lipid, %	Phospholipid, %	Neutral Fat, %	Cholesterol, %	Phospholipid : Neutral Fat† %
1-C	71.9	4.42	3.15	.928	.337	71 : 21
2-C	71.1	4.68	3.43	.873	.381	73 : 19
3-C	71.7	4.68	3.68	.666	.331	79 : 14
4-C	72.0	4.44	3.44	.669	.335	78 : 15
5-C	73.3	3.95	3.07	.462	.415	78 : 12
6-C	70.7	4.23	3.45	.487	.297	82 : 12
7-C	71.5	4.01	3.22	.516	.278	80 : 13
8-C	70.4	4.15	3.23	.569	.350	78 : 14
Avg	71.6	4.32	3.33	.646	.341	77 : 15
9-A	73.7	4.12	3.16	.361	.600	77 : 9
10-A	74.3	4.11	3.44	.237	.433	84 : 6
11-A	72.0	4.43	3.62	.372	.437	82 : 8
12-A	70.5	3.74	2.47	.995	.277	66 : 26
13-N	72.0	4.74	3.29	.960	.390	70 : 20
14-N	72.5	4.04	3.03	.645	.365	75 : 16
Avg	72.5	4.20	3.17	.595	.417	76 : 14

*C—Control; A—Arsphenamine; N—Neoarsphenamine.
†Expressed as per cent of total lipid.

[4] Bloor, W. R., *J. Biol. Chem.*, 1928, **77**, 53.
[5] Boyd, E. M., *J. Biol. Chem.*, 1931, **91**, 1.

following liver injury occurs only when there is a change (from normal) in the total lipid content of the organ.

Summary. Liver injury in rabbits resulting from the administration of arsenicals in the form of arsphenamine and neoarsphenamine cause no significant changes from normal in the amount or distribution of the liver lipids.

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Effect of Chlorination of City Water on Virus of Poliomyelitis.*

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Water was considered in early reports concerning the transmission of poliomyelitis. This method of spread seemed unlikely when later experimental evidence favored an air-borne infection entering the host through the olfactory tract. However, Kling,[1] observing European epidemics, reconsidered the question and additional evidence was accumulated incriminating water as a factor in the spread of the virus.

Poliomyelitis virus was found in human feces as early as 1912[2] and these observations have been amply confirmed. Unfortunately the technic of Sawyer[3] requiring a second monkey passage as an important criterion to verify the presence of the virus was ignored until 1938. In that year, Trask, Vignec, and Paul,[4] and Kramer, Hoskwith, and Grossman[5] improved the technic of virus isola-

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1 Kling, C., *Bull. Office internat. d'hyg. pub.*, 1928, **20**, 1779.

2 Kling, C., Petterson, A., and Wernstedt, W., *Communication Inst. méd. État*, Stockholm, 1912, **3**, 5.

3 Sawyer, W. A., *Am. J. Trop. Dis. and Prev. Med.*, 1915, **3**, 164.

4 Trask, J. D., Vignec, A. J., and Paul, J. R., PROC. SOC. EXP. BIOL. AND MED., 1938, **38**, 147.

5 Kramer, S. D., Hoskwith, B., and Grossman, L. H., *J. Exp. Med.*, 1939, **69**, 49.