

dying on the 17th and 18th day of gestation. In the 25 placentas examined, no larvae were found in either fetal or maternal portions. Of animals born, microscopic examinations were made on 26 from 5 litters which were stillborn or dying at ages up to 20 days, and on 8 from 6 litters killed at from one to 20 days of age. Material was taken from 7 to 38 days after infection of the mothers. In no instance were trichina larvae found in fetal tissues or fluids.

Digestion of the skinned and eviscerated carcass in a solution of hydrochloric acid and pepsin is useful only for the recovery of larvae which have become encysted in skeletal muscle. At 60 to 70 days of age, one male and one female from each of 17 litters of mothers fed 15 larvae per gram were digested. The mothers had been infected at the following times: 15, 14 (2), 12, 11 (2), 10 (2), 8, 7, 5 (2), 4, 3 (2), and one day after, and in one instance one day before, copulation. No larvae were found.

These results indicate that in the white rat trichina larvae circulating in the maternal blood are not capable of crossing the placental barrier into the fetal circulation and tissues. We have found no larvae in the sections of placentas examined. Larvae also have not been found to migrate from the peritoneal cavity of the mother rat into the amniotic sac or embryo proper. Finally, our results support the view that trichinosis is not transmitted by way of the milk or feces, as the young rats were suckled by their own mothers and were caged with them.

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Effect of Acute Infection on Iodine Number of the Phospholipid Fatty Acids.*

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There is a definite lowering of the values for the plasma lipids at the height of an acute infection as compared with the levels obtained during convalescence.¹ Fever *per se* is not responsible for the fall of the various lipid constituents of the blood plasma. Diet does not have any demonstrable influence on the results. The drop in

* This work was supported by a grant from the Medical Research Fund of the University of Minnesota.

¹ Stoesser, A. V., and McQuarrie, Irvine, *Am. J. Dis. Child.*, 1935, **49**, 658.

the total cholesterol values is due almost entirely to a marked decrease in the ester cholesterol² and this condition holds true for infants as well as children.³ The fall in the total fatty acid values is accompanied by low iodine absorption values yielding iodine numbers of the serum fatty acids which are significantly lower during the febrile period of the disease than during the afebrile period of convalescence.^{4, 5} The plasma phospholipids do not in all cases drop immediately to low levels after the onset of the acute illness. In some instances, values above the normal range have been obtained early in the disease. The behavior of the phospholipid fatty acids in relationship to the variations in the phospholipids in acute infections of children has not been recorded.

Eighteen children ranging in age from 13 months to 15 years were chosen for this study. Nine of the subjects had upper respiratory infections with acute otitis media, acute bronchitis, or cervical adenitis, while the remaining 9 had pneumonia with no complications. All the blood samples obtained during and after the height of each infection were drawn between 12 and 16 hours after a meal. The first blood sample was collected in 8 of the cases before the end of the second day of the febrile period of the disease and in the remaining 10 patients between the sixth and seventh day of the illness. The second blood sample was collected in all children between the seventh and ninth days of convalescence. Bloor's method⁶ as modified by Boyd⁷ was employed to obtain the total phospholipid content of the blood plasma and the Rosenmund-Kuhnenn method⁸ as modified by Page, Pasternak and Burt⁹ was utilized to determine the iodine absorption values of the phospholipid fatty acids.

The results are summarized in Table I.

There was with only 2 exceptions a definite fall in the plasma phospholipid values in the group of children in whom the first blood sample was obtained early in the disease. This may indicate that the rise in total phospholipids observed by Boyd¹⁰ in acute infections of the adult must occur in the child very soon after the onset of the

² Stoesser, A. V., *Proc. Soc. Exp. Biol. and Med.*, 1935, **32**, 1324.

³ Stoesser, A. V., *Proc. Soc. Exp. Biol. and Med.*, 1936, **34**, 10.

⁴ Stoesser, A. V., *Proc. Soc. Exp. Biol. and Med.*, 1935, **32**, 1326.

⁵ Stoesser, A. V., *Proc. Soc. Exp. Biol. and Med.*, 1936, **34**, 12.

⁶ Bloor, W. R., *J. Biol. Chem.*, 1929, **82**, 273.

⁷ Boyd, E. M., *J. Biol. Chem.*, 1933, **101**, 323.

⁸ Rosenmund, K. W., and Kuhnenn, W., *Z. f. unter. d. Nahr. d. Nahr. u. Genuss.*, 1923, **46**, 154.

⁹ Page, H. H., Pasternak, L., and Burt, M. L., *Biochem. Z.*, 1930, **223**, 445.

¹⁰ Boyd, E. M., *Canadian Med. Assn. J.*, 1935, **32**, 500.

TABLE I.
Iodine Numbers of the Phospholipid Fatty Acids in Infections of Upper
Respiratory Tract and in Pneumonia.

Case No.		Plasma Phospholipids		Iodine No.	
		Mg. per 100 cc. serum			
A	B	A	B	A	B
Febrile period of disease.					
1	9	95	125	146	57
2	10	85	98	146	70
3	11	159	124	88	67
4	12	181	60	105	76
5	13	124	64	131	52
6	14	111	93	153	84
7	15	76	121	161	51
8	16	72	122	147	87
	17		106		67
	18		101		62
Aver.		112	101	134	67
Afebrile period or convalescence					
1	9	175	170	120	84
2	10	180	174	115	83
3	11	116	179	51	110
4	12	136	154	77	149
5	13	182	103	116	87
6	14	184	177	120	113
7	15	162	164	109	110
8	16	121	139	102	98
	17		221		85
	18		167		86
Aver.		157	165	101	100

A—First blood sample collected second day of febrile period.

B—First blood sample collected sixth or seventh day of febrile period.

infection. The decrease in the serum phospholipids was not accompanied with a fall in the iodine absorption values. On the contrary, the iodine numbers were higher than normal at this stage of the disease. The ratio of saturated to unsaturated phospholipid fatty acids referred to by Sinclair¹¹ was certainly greatly disturbed by the acute infection, there being a relative increase in the unsaturated fatty acids probably in response to an increase in the intensity of the fat metabolism during the febrile period.

The plasma phospholipid values were consistently low in the other group of children in whom the first blood sample was obtained toward the end of the height of the disease. This condition was accompanied by a marked fall in the iodine absorption of the phospholipid fatty acids yielding low iodine numbers. The desaturation may be entirely due to the rapid utilization of the unsaturated fatty acids by the altered metabolic processes present during an infection. There is, however, an additional factor which could play a

¹¹ Sinclair, R. G., *Physiological Rev.*, 1934, **14**, 351.

rather significant rôle. The observation has been made that certain toxic substances such as cobra venom when mixed with lecithin cause it to lose its unsaturated fatty acid radicle.¹² The resulting lecithin compound contains a relatively high proportion of saturated fatty acids. The question thus arises as to whether the bacterial toxins of an acute infection could have the same influence on the plasma phospholipids. Most logical at the present time is the assumption that the intensity of the fat metabolism during an acute infection and the presence of the bacterial toxins in the blood may both be responsible for the ultimate fall in the plasma phospholipids to abnormal levels with an associated extensive loss of their unsaturated fatty acids. Should this actually be found to occur, it would give the phospholipids an important part in the phenomena of immune reactions.

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Photoelectric Spectrophotometry Applied to Studies in Fat Metabolism.*

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Recently new methods have been applied for following the rate of movement and metabolism of fats. Schoenheimer and Rittenberg¹ have published a series of papers on the use of deuterium as an indicator in fatty acid metabolism. Sinclair and coworkers² have made use of elaidin as an indicator of rate of movement and metabolism of fats in various rat tissues.

The common fats and fatty acids show very low absorption of light from 2000 to 8000 A.U., except for a weak band at 2300 A.U.⁴ However, there are 2 exceptional cases. α -Eleostearic acid of tung oil has a very strong maximum at 2700 A.U.³ Moore⁴ has shown that linseed oil fatty acids become strongly "absorptive" on

¹² Peters, John P., and Van Slyke, Donald, *Quantitative Clinical Chemistry*, Williams and Wilkins Company, Baltimore, 1931, Vol. 1.

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¹ Rittenberg, D., and Schoenheimer, R., *J. Biol. Chem.*, 1937, **117**, 485.

² McConnell, K. P., and Sinclair, R. G., *J. Biol. Chem.*, 1937, **118**, 123.

³ Dingwall, A., and Thomson, J. C., *J. Am. Chem. Soc.*, 1934, **56**, 899.

⁴ Moore, T., *Biochem. J.*, 1937, **31**, 138.