TABLE I.
Protective Effect on Chicks Afforded by Soy Bean Oil and Non-saponifiable Matter of Soy Bean Oil. Results Taken at 28 Days.

Supplement to basal diet	No. of birds	No. developing definite paralysis	No. dying without paralysis being observed
None	30	16	6
Ether solution of non-saponifiable matter of soy bean oil corresponding to 15% of original oil	28	0	1
10% of soy bean oil	25	0	1

with 5% sulfuric acid, then with water until neutral, concentrated and used to supplement the diet described below.

The basal diet consisted of corn starch, 65%; fat-extracted whole sardine meal,† 25; rice bran extract,[3] 7; whey adsorbate,[4] 2; alfalfa hexane extract,[5] equivalent to 0.5% of alfalfa meal; cod liver oil, 1. In some experiments a purified antihemorrhagic concentrate, kindly supplied by Dr. H. J. Almquist, replaced the alfalfa hexane extract without perceptible alteration of the results. Single-comb White Leghorn chicks were placed on this diet at hatching. On the basal diet, paralytic symptoms, accompanied by loss of weight and shortly followed in most cases by death, appeared at 20 days of age. By the 28th day, the results summarized in Table I were obtained.

The chicks on the supplemented diets appeared normal, but were definitely below normal weight. This observation of the protective effect of the non-saponifiable fraction of soy bean oil is in accord with the results of Goettsch and Pappenheimer.[2]

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Non-Transmissibility *in utero* of Trichinosis in the Rat.

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For several weeks after the maturation of ingested larvae of *Trichinella spiralis* in the intestine, those of the next generation enter the circulating blood of the host. Although the presence of

† Furnished by the F. E. Booth Co., San Francisco, by the courtesy of Mr. T. D. Sanford.

3 Lepkovsky, S., and Jukes, T. H., *J. Biol. Chem.*, 1935, **111**, 119.

4 Lepkovsky, S., and Jukes, T. H., *J. Biol. Chem.*, 1936, **114**, 109; Jukes, T. H., *J. Biol. Chem.*, 1937, **117**, 11.

5 Almquist, H. J., *J. Biol. Chem.*, 1936, **114**, 241.

migrating larvae in various organs, tissues and body fluids has been observed repeatedly, studies of pregnant hosts, such as the rat, guinea pig, rabbit, hog and man, have indicated that intrauterine transmission of trichinosis from mother to fetus does not occur. Roth,[1] however, recently reported the recovery by digestion of a few larvae in 2 out of 5 litters of guinea pigs born of mothers infected during pregnancy. Previous investigations have dealt with comparatively small groups of animals, and little importance has been attached to the stage of gestation at which mother animals have been infected.

Young adult white rats weighing from 180 to 280 gm. were fed by stomach tube the larvae of *Trichinella spiralis* obtained by digestion of infected rats in artificial gastric juice.[2] The time of inoculation varied from one day before, to 15 days after, copulation. The presence of infection in all mother rats was confirmed by subsequent microscopic examinations of their diaphragms and other skeletal muscles. Since migration of young larvae begins about one week after infection and usually continues for 2 weeks, larvae were migrating in various rats during all except the earliest stages in the maturation of the placenta and of fetal development of muscle. Attempts to obtain copulation of female rats 2 to 7 days after infection were unsuccessful. Twenty-one females were fed 15 larvae per gm. of body weight, an amount sufficient to cause an infection of moderate severity. To 2 groups of 3 rats each were fed 35 and 40 larvae respectively per gm. of body weight. Most of the offspring of rats fed 15 larvae per gm. were of usual size and weight, and developed normally. The few which did not appear perfectly healthy were included among those examined for trichinosis. Most of the young of heavily infected rats died. Those living several days soon became underweight, scrawny and feeble as compared to normal rats of similar age. None was observed to have diarrhea.

To determine whether migrating larvae entered fetal tissues, microscopic examinations were made of fresh teased and pressed preparations and of sediments from centrifuged washings of serous cavities. Also several sections from each of 6 to 10 serial blocks were prepared from each young rat examined; in this way practically all organs and tissues were searched. Sections were made of the 9 fetuses and placentas from one moderately infected mother killed on the 21st day of gestation. Similar material was examined from the 8 fetuses and placentas of each of 2 heavily infected mothers

[1] Roth, H., *Acta Path. et Microbiol. Scand.*, 1935, **12**, 203.

[2] McCoy, O. R., *Am. J. Hyg.*, 1931, **14**, 484.

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 days 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.