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Ineffectiveness of Sulfanilamide in the Treatment of Trichiniasis in Rats.

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Since the demonstration that sulfanilamide is an effective therapeutic agent in the treatment of infections caused by the coccal group of bacteria, many tests have been made of its possible value in the treatment of other diseases of varied etiology, particularly those for which no specific drug is known. So far, sulfanilamide has not been found of value in infections caused by bacilli, filterable viruses,[1] or animal parasites.[2] In view of the belief that the drug may act to increase host-resistance and to neutralize toxins, it was thought worthwhile to test its value in the treatment of trichiniasis, a disease in which the symptoms are mainly the result of toxemia, and for which there is no known effective drug treatment.

Two experiments were performed in which full-grown rats were infected by stomach tube with known numbers of *Trichinella spiralis* larvae, obtained free from infected rat muscle by artificial digestion.[3] In each experiment, one group of rats were given treatment with sulfanilamide (para-amino-benzene-sulfonamide), while another group were kept as controls. The dosage of sulfanilamide was the same as that employed by Gross and Cooper[4] for the treatment of pneumococcal infections in rats, namely, 125 mg per day. The drug was ground to a fine powder, suspended in a 10% solution of gum acacia, and given to the animals by stomach tube. Details of administration are presented in Table I. Usually, treatment was begun on the day after infection, at a time when the adult worms were developing in the intestine, and was continued during the period that the young larvae were developing in the muscles.

From 1 to 2 months after infection, all the animals were killed, their carcasses were digested individually, and a count was made of the total number of larvae which had developed in the muscles. The average amount of infection, as measured in this way, was approxi-

1 Dickerson, V. C., and Whitney, L. F., PROC. SOC. EXP. BIOL. AND MED., 1938, **38**, 263.

2 Keil, E., *Arch. f. Schiffs. u. Trop. Hyg.*, 1936, **40**, 400.

3 For methods see McCoy, O. R., *Am. J. Hyg.*, 1931, **14**, 484.

4 Gross, P., and Cooper, F. B., PROC. SOC. EXP. BIOL. AND MED., 1937, **36**, 225.

TABLE I.
Rats Infected with *Trichinella spiralis* and Treated with Sulfanilamide.

Group	No. of rats	No. of larvæ fed	Treatment with sulfanilamide	Avg No. of larvæ in muscles
Experiment 1				
Treated	4	2,000	125 mg for 12 days beginning the 6th day after infection	110,900
"	4	2,000	125 mg for 16 days beginning the day after infection	99,800
Control	8	2,000	None	93,200
Experiment 2				
Treated	9	3,000	125 mg for 12 days beginning the day after infection	165,600
Control	8	3,000	None	236,600

mately the same in the treated rats as in the control animals (Table I). In other treated rats (not listed in the table), killed at intervals after the beginning of treatment, no lethal effect was observed either on the adult worms in the intestine or on the developing larvae in the muscles. Also, as judged by the amount of weight lost during the course of the infection, the drug had no effect on the severity of the symptoms. In fact, the treated rats lost considerably more weight than the control animals, and also more than other rats which were given the same amount of drug but were not infected with *Trichinella*. No evidence was found to suggest, therefore, that sulfanilamide had any value in the treatment of experimental trichiniasis in rats.

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Human Ascitic Fluid as a Blood Substitute in Experimental Secondary Shock.

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The value of a substitute for whole blood for clinical and experimental use is self-evident. Various solutions have been utilized, such as blood plasma,[1, 2] gum-saline,[3, 4] gelatin-saline,[5, 6] hemoglobin-

[1] Guthrie, C. C., and Pike, F. H., *Am. J. Physiol.*, 1907, **18**, 14.

[2] Richet, C., and Brodin, P., *Compt. Rend. Acad. Sci.*, 1917, **167**, 55.

[3] Morawitz, P., *Beitr. Chem. Physiol. Path.*, 1906, **7**, 153.

[4] Robertson, O. H., and Bock, A. B., *Rep. Med. Res. Council, Lond.*, 1918, No. 25, 213.

[5] Hogan, J. J., *J. A. M. A.*, 1915, **64**, 721.

[6] Clark, G. W., *J. Immunol.*, 1918, **3**, 147.