and 24th days. Injections of 0.00025 mg of vitamin B-12 were continued from the 27th to the 40th day. During the first period of vitamin B-12 alone the red blood cell count rose from 1,850,000 to 2,220,000. During the second period with thymidine added the count increased further to 2,800,000. At the end of the third period of sub-optimal vitamin B-12 alone it had decreased to 2,600,000.

Discussion. The replacement ratio of thymidine for vitamin B-12 is about 300:1 for the growth of *L. lactis*.[6] We have seen a maximal reticulocyte response in pernicious anemia to as little as 4 μg of B-12.[7] A dose of 5 mg of thymidine should therefore be more than adequate if the ratios observed in the bacterial growth system obtain for man. (That this is so for thymine with reference to folic acid was shown by Spies and his associates.[5]) From the results we obtained, it appears that thymidine alone is incapable of sustaining blood regeneration, although it does appear to cause a slight increase in reticulocytes. Similar results were reported by Geerts and Lens using doses of 10 mg of thymidine a day for 3 days.[8] From our observations it is apparent that the response to 150 mg was no greater.

It has been suggested that crude liver extracts may contain substances that give an enhanced hematopoietic response compared to B-12, and thymidine may be one of these accessory substances.[9] From our study it appears that thymidine is not completely inert hematopoietically, but if it has an enhancing effect on B-12 activity, this effect is very slight in the dosage used in this study. In view of the ability of other desoxyribosides to substitute for B-12 in bacterial growth, the effect of mixtures of desoxyribosides on pernicious anemia should be investigated.

Summary. 1. In 3 patients with pernicious anemia, thymidine in doses of 5 to 150 mg was found to cause slight reticulocytosis, but no effect on the red blood count.

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6 Shive, W., Ravel, J. M., Eakin, R. E., *J. Am. Chem. Soc.*, 1948, **70**, 2614.

7 West, R., and Reisner, E. H., Jr., *Am. J. Med.*, 1949, **6**, 643.

8 Geerts, S. J., and Lens, J., *Nature*, to be published.

9 Jacobson, M., and Bishop, R. C., *J. Clin. Invest.*, 1949, **28**, 791.

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17286. Effect of Adrenalectomy on Eosinophil Response of Rats Infected with *Trichinella spiralis*.

JOHN E. LARSH, JR. AND JOHN NICHOLS. (Introduced by A. T. Miller, Jr.)

From the Department of Parasitology and the Department of Anatomy, University of North Carolina, Chapel Hill, N. C.

Recently there have appeared reports[1-4] that adrenal hormones depress the number of circulating eosinophils. Injection of adrenal cortical hormone will cause a lowering of the number of circulating eosinophils, or injection of adrenotrophic hormone from the pituitary will accomplish the same result if the adrenal gland is intact. In fact, this lowering of the eosinophils taken together with other changes in the body chemistry is proposed as a test for adrenal cortical insufficiency. Presumably the presence of the adrenal cortex has an inhibitory influence on the eosinophil producing bone marrow.

The eosinophil response to infection with *Trichinella spiralis* usually is striking in

1 Thorn, G. W., Forsham, P. H., Prunty, F. T., and Hills, A. G., *J. Am. Med. Assn.*, 1948, **137**, 1005.

2 Forsham, P. H., Thorn, G. W., Prunty, G., and Hills, A. G., *J. Clin. Endocrinol.*, 1948, **8**, 15.

3 Hills, A. G., Forsham, P. H., and Finch, C. A., *Blood*, 1948, **3**, 755.

4 Hellman, L., *Science*, 1949, **109**, 280.

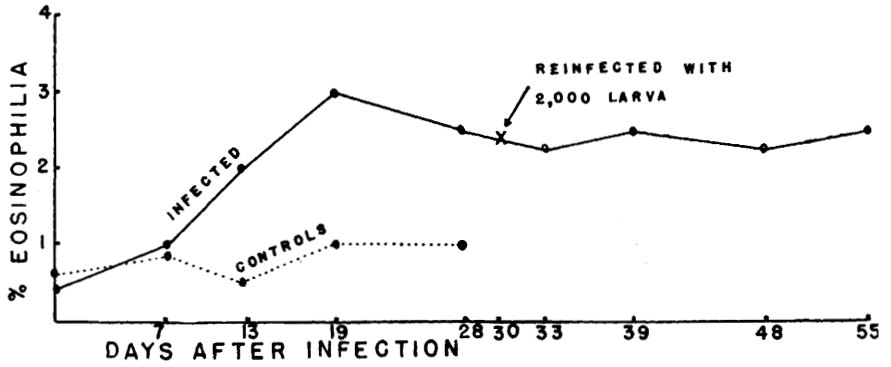

FIG. 1.
Eosinophil response of intact rats infected with *Trichinella spiralis* larvae, as compared with intact uninfected controls.

humans (Gould[5]) and has been reported rather high for mice (Hunter and Groupé,[6] Stein[7]) and rats (Someren[8]). This suggested that the eosinophil response in intact and adrenalectomized animals infected with this parasite would yield interesting results. Thus the following study was undertaken.

Experimental. Preliminary experiments were performed to check the eosinophil response of our rats to *T. spiralis* infection. These animals (Wistar strain) were raised and maintained on our stock diet in an air-conditioned room (76°F). The experimental procedures for infecting the animals with standard doses of larvae have been described elsewhere (Larsh and Kent[9]). It is important to point out that the viability of these larvae in all experiments was checked by determining in a few control animals the percentage development of adult worms 5 days post-infection. The blood for eosinophil counts was obtained from the tail, stained with Giemsa stain in the usual manner, and examined under oil immersion. These counts were done at approximately weekly intervals.

Sixteen rats (2.5 months old) were used to determine the eosinophil response. Three received 500 larvae; three, 1000 larvae; three, 1500 larvae; three, 2500 larvae; and 4 were kept as uninfected controls. The infected animals were reinfected with 2000 larvae 30 days after initial infection. Since the eosinophil response was similar for all of the infected animals, the results were plotted as the same (Fig. 1). The graph shows very little eosinophilia produced in these animals, as contrasted to the 20% (in 16 days) mentioned above (Someren[8]). Our results, however, compare favorably with those of Beahm and Downs.[10] It is unlikely that our results were due to a strain factor in the rats in that random counts of another rat strain infected as above revealed about the same findings. It is possible that our strain of *T. spiralis* is a poor stimulator of eosinophilia. There is some support for this in that our white mice (2.5 months old), likewise, showed a poor eosinophil response. These were infected with various doses of *T. spiralis* larvae; 3 with 50 larvae; 3, 100 larvae; 3, 300 larvae; and 3, 400 larvae. All of these showed about the same level of eosinophilia, which reached a maximum of 10% after one month. The 4 non-infected controls averaged about 3%. The response of the infected mice was considerably lower than that (25-33% within 2 weeks) reported by the above mentioned workers.[6,7]

Despite the poor eosinophil response described above, it was decided to determine

[5] Gould, S. E., Trichinosis, 1945, C. C. Thomas Co., Springfield, Ill.

[6] Hunter, G. W., III, and Groupé, V., *J. Parasitology, (supp.)*, 1939, **25**, 33.

[7] Stein, K. F., *Anat. Rec.* 1949, **103**, 508; PROC. SOC. EXP. BIOL. AND MED., 1949, **71**, 225.

[8] Someren, V. D., *J. Helminth.*, 1938, **16**, 83.

[9] Larsh, J. E., Jr., and Kent, D. E., *J. Parasitology*, 1949, **35**, 45.

[10] Beahm, E. H., and Downs, C. M., *J. Parasitology*, 1939, **25**, 405.

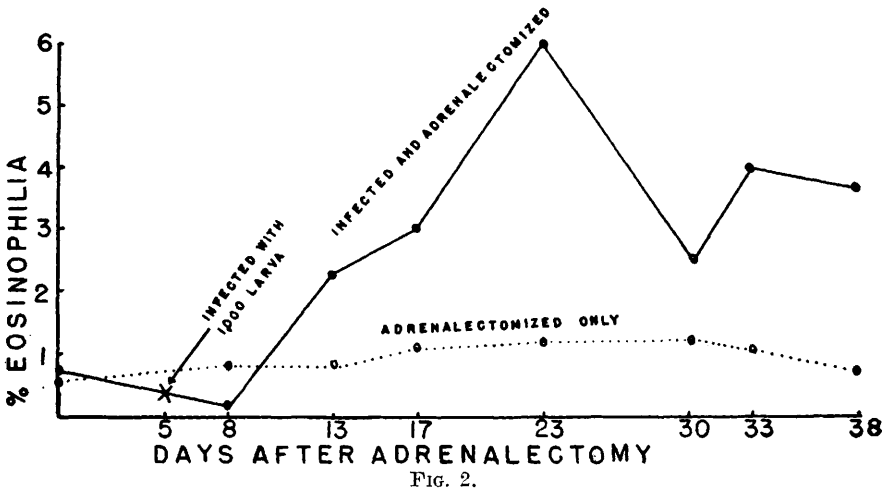

FIG. 2.

Eosinophil response of rats infected with *Trichinella spiralis* larvae 5 days after adrenalectomy, as compared with uninfected adrenalectomized controls.

the effect on this response of adrenalectomy. This work was limited to rats. Nineteen (2.5 months old) were adrenalectomized in a one stage operation, and kept on a potassium-low diet (Nichols[11]). They were given drinking water with 0.8% NaCl and 0.1% $NaHCO_3$, and remained in apparent good condition throughout the experiment. The completeness of adrenalectomy was verified at autopsy. Eleven of these rats were infected with 1000 *T. spiralis* larvae 5 days after adrenalectomy, the remaining 8 were kept as uninfected controls. The eosinophil counts of all of the animals are shown in Fig. 2. While the counts are somewhat higher than those shown in Fig. 1 for intact rats, the difference is too slight to draw definite conclusions. Perhaps it would be worthwhile to repeat this experiment with an animal which has a significantly higher granulocyte count than the rat.

Summary. (1) White mice and rats failed to show a high eosinophilia following initial infection with various doses of *T. spiralis*. (2) Rats also failed to elicit a striking response following reinfection. (3) Adrenalectomy influenced only slightly the number of circulating eosinophils in infected rats.

[11] Nichols, J., *Arch. Path.*, 1948, **45**, 717.

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17287. Local Sweating in Man Induced by Intradermal Epinephrine.

RALPH R. SONNENSCHEIN. (Introduced by M. I. Grossman.)
From Department of Clinical Science, University of Illinois, College of Medicine, Chicago, Ill.

The possibility of an adrenergic innervation of the sweat glands was suggested recently by Haimovici,[1] who reported that intravenous injection of neosynephrine was followed by an increase in palmar sweating; this response was inhibited by dibenamine (N,N-dibenzyl-β-chloroethylamine hydrochloride). In preliminary experiments[2] in our laboratory, intradermal injection of neosynephrine into the palm failed to alter the pattern of spontaneous sweating. Patton[3] measured electrical potentials of the cat's paw, as an index of sweat

[1] Haimovici, H., PROC. SOC. EXP. BIOL. AND MED., 1948, **68**, 40.

[2] Janowitz, H., Sonnenschein, R. R., and Grossman, M. I., unpublished observations.