ity: Compound E and equally-active Compound F were most efficacious in reducing the number of circulating eosinophils. Compound E aldehyde was less active, while Kendall's Compound A acetate, Kendall's Compound B and Compound E tricarballylate were still less potent. Reichstein's Substance S had no eosinopenic activity even though it was administered in larger amounts than the other compounds. Except for Substance S, the other corticoids tested showed a significant positive regression of percent fall of circulating eosinophils on the logarithm of the administered dose within the limits of the dosages tested. The slopes of the linear regressions of these corticoids were also more or less similar.

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Immobilization of *Schistosoma mansoni* Miracidia by Immune Serum.* (20523)

L. B. SENTERFIT. (Introduced by Frederik B. Bang)

From the Department of Medicine, Johns Hopkins University, Baltimore, Md.

Immobilization of ciliates and free living flagellates by antibodies is well known(1,2). This report concerns the development of an immobilizing factor for the ciliated larval stage (miracidium) of *Schistosoma mansoni* by experimentally infected animals. This factor appears in the serum at about the same time in the course of the infection as do the capacities to form a precipitate around the cercariae (infective larval stage)(3), to agglutinate the cercariae(4), and to form a "hood" around the cercariae(5). It is recognized that these 3 reactions and the one here described may be caused by the same antibody.

Material and methods. The miracidia used in the immobilization tests were obtained from the livers of infected hamsters. A suspension of the infected liver in 0.15 *M* NaCl was prepared by maceration in the Waring Blender for 30-60 seconds; this suspension was allowed to settle in a sedimentation cone for ½ hour. The sediment was suspended

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in aged tap water in a 250 ml Erlenmyer flask, placed under a strong light source and the hatching miracidia collected with a capillary pipette.

Immune† sera were obtained from golden hamsters (*Cricetus auratus*) infected with *S. mansoni* for varying periods of time and from monkeys (*M. mulatta*) infected with *S. mansoni* and (*M. phillippinensis*) infected with *S. japonica*. Controls included normal monkey, hamster, and human sera. All blood from monkeys and hamsters was drawn by cardiac puncture. All sera were heated at 56°C for ½ hour.

Serial dilutions of the serum were made in 10 x 100 mm test tubes. Five drops of a given dilution were then added to 5 drops of a water suspension of miracidia which had been placed on a paraffin ringed microscope slide. Each preparation usually contained 5 or more miracidia. The mixture was allowed to stand for 10 minutes at room temperature, and the effect of the serum was determined with the aid of a binocular dissecting microscope (60x). Any noticeable effects on the miracidia were studied further by placing a coverslip over the slide and observing at 100x and 400x under a compound microscope.

The procedure outlined in the original report(3) was used in studies on the cercarial precipitate reaction.

Results. Serial dilutions of sera were tested for immobilizing effect, as shown in Table I.

In the higher dilutions of serum it was observed that some cilia were immobilized while others on the same organism continued to beat. A series of preparations at different concentrations of serum were then studied under a Zeiss phase contrast microscope. The

TABLE I. Effect of Normal and Immune Hamster Serum on the Miracidia of *S. mansoni*.

Animal No.	Days infected	Serum dilution 1/10	1/20	1/40	1/80	1/160
72 (inf.)	161	4	4	2	1	0
29 (nor.)	0	0	0	0	0	0

4 = Complete immobilization.
3,2,1 = Partial immobilization but some cilia remain mobile.
0 = No effect.

† Sera from infected animals are referred to as immune sera.

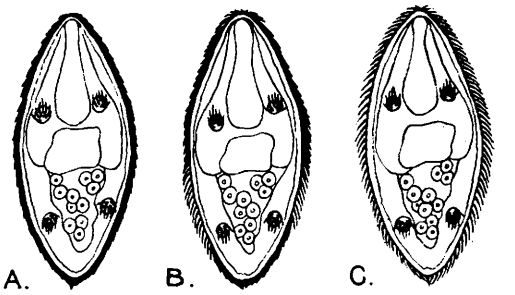

FIG. 1. Areas of immobilized cilia shown in black. A. Total immobilization (4+). B. Partial immobilization (2+). C. No immobilization (—) normal serum.

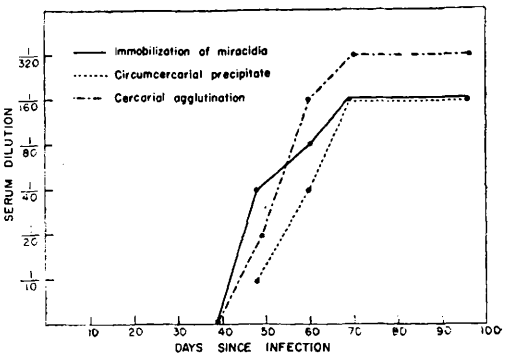

FIG. 2. Titers of *in vitro* reactions in infected monkey.

immobilized cilia were usually in the same general location on the miracidium as illustrated in Fig. 1.

Time of appearance of the immobilization antibody. A rhesus monkey infected with 500-600 *S. mansoni* cercariae was bled at approximately 10-day intervals. The sera were stored at —16°C until use. The immobilizing activity of the serum first appeared between the 39th and 48th day. It increased in titer until a maximum of 1/160 was attained at about the 75th day (Fig. 2). For comparison the titers of the cercarial precipitate reactions and the cercarial agglutination reactions are also shown. The titers of the cercarial agglutination reactions are from data previously collected by Liu and Bang(6) on these sera, but the data on the precipitate formation were obtained from new experiments.

Sera from 2 monkeys infected 5½ years previously with *S. japonica* showed a positive reaction at a dilution of 1/10. All tests utilizing normal monkey, normal ham-

ster, and normal human serum were negative, but one serum from a proven case of trichinosis in man was positive at a dilution of 1/20.

Discussion. The role of this immobilization reaction in the pathogenesis of *S. mansoni* infection is unknown. It is possible that it bears some relationship to the ray-like structures surrounding the eggs imbedded in tissue as described by Hoeppli and Li(7).

The time of appearance of the factor in the blood indicates that a possible major stimulus for its production is the initial deposition of eggs. Egg laying activity begins between the 27th and the 34th day of infection with *S. japonica*(8) and presumably during the same period in *S. mansoni* infection. The application of this reaction to studies in man remains to be made. Its possible use in the diagnosis of chronic infections is suggested by the positive reactions in monkeys infected for 5½ years.

Summary. Miracidia of *S. mansoni* were promptly immobilized when placed in heated sera of hamsters and monkeys infected with this parasite. No immobilization occurred in sera from non-infected animals. The factor developed early in the infection and was still present in the sera of monkeys which had been infected with *S. japonica* 5½ years previously. The immobilization of the miracidium appears to result from immobilization of the cilia. Cilia immobilization became less complete in high dilutions of serum and was usually confined to specific areas of the miracidium.

Since this work was done the author has become aware of an abstract by Hiyashi who reported to the Japanese Parasitology meeting in Kyoto in 1950 (Abstract 101) that he had found immobilization of miracidia from the sera of infected animals.

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Influence of Parathyroid Extract on Citric Acid of the Serum. (20524)

MAURICE V. L'HEUREUX AND GILBERT J. ROTH (Introduced by M. B. Williamson.)
From the Department of Biochemistry, Graduate School and The Stritch School of Medicine, Loyola University, Chicago, Ill.

It has long been recognized that the parathyroid glands are intimately concerned with the metabolism of calcium and phosphorus. In recent years, evidence has accumulated for considering the possibility that the active secretion may influence the metabolism of citrate as well. An early observation is that of Sjöström(1) who reported that increased parathyroid function produces a corresponding increase in the citric acid content of the blood. A rough parallelism has been observed between urinary excretion of calcium and of citrate in a patient with hyperparathyroidism and in a subject with hypoparathyroidism given parathyroid extract(2). Alwall(3) demonstrated concomitant increases in serum calcium and serum citric acid following intramuscular injection of parathyroid extract into dogs. The injection of parathyroid extract into puppies has been noted to increase the citrate content of bone(4). Repeated subcutaneous injections of citrate into puppies lead to osseous changes very similar to those produced by large doses of parathyroid extract(5). Freeman and Chang(6) observed, without exception, a decline in both serum calcium and serum citric acid levels following thyroparathyroidectomy in dogs. It thus appears that changes in the level of serum calcium referable to the involvement of the para-