body in the sera of young mice remains unchanged during the 4-h test period. Consequently, differences, if any, in the nature of the antibodies fed to young mice and those measured in their circulations 4 h later will have been effected in the absorptive cells of the gut rather than in the circulation.

Table 2. Antibody Activity of the Sera of Young Mice injected intravenously with Rabbit Anti-*Brucella* Serum

Interval after injection	Serum titre	Dilution of antibody in serum
5 min	Saline = 512	1/8
	Coombs = 512	1/8
4 h	Coombs = 512	1/8
	Saline = 512	1/8

It has been established that the activity of the immune serum used is due primarily to 7*S* and 18*S* complete antibodies, and possibly to 7*S* and 18*S* incomplete antibodies, whereas the activity of the sera of young mice fed with the immune serum is due to 7*S* incomplete antibodies. Consequently, the transmission of rabbit anti-*Brucella* agglutinins across the gut may involve any or several of the following possibilities:

(1) During transfer across the absorptive cells the 18*S* antibodies may be degraded into the 7*S* incomplete type. An analogous change *in vitro* would be the decomposition of 18*S* saline agglutinating Rh antibody by the action of mercaptan into 7*S* Coombs sensitizing antibody[7].

(2) A configurational rather than a degradational change from the 7*S* complete antibody into a 'more acceptable' 7*S* incomplete type may be effected by the absorptive cells. A similar explanation has been suggested to account for the observation that bovine γ-globulin, which had reached the circulation from the gut of young rats, entered the circulation in relatively larger amounts when fed a second time to other young rats[8].

(3) The absorptive cells may exhibit selection between 7*S* complete and incomplete antibodies and exclude the former from the circulation. If this were the case, such a high degree of selection between two antibodies prepared in the same species would be hard to reconcile with the much lower degree of selection exhibited between antibody molecules of different antibody activities prepared in different species[2], unless the effect of variation in the configuration of the antibody reactive portions[9] of 7*S* antibody molecules on selection overrides that of the species-specific parts.

The inability to measure the incomplete antibody activity of the immune serum used, due to the higher concentration of the complete antibody, precluded hitherto any assessment of the relative importance of these possibilities. Further investigation of the problem is desirable, especially in view of the light it might throw on the mechanism of the transmission of Rh antibodies across the human placenta.

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An Anomalous Infection of Mice with *Schistosoma mansoni*

Mice exposed percutaneously to cercariæ of *S. mansoni* develop an infection which can be well standardized. For the past three years we have exposed albino mice, kept in identical conditions of diet and housing, to about 200 cercariæ of a Venezuelan strain[1] which led to the development of a bisexual infection in the portal and mesenteric veins and the deposition of eggs in the liver, intestinal walls and occasionally in the lungs.

In one experiment on a group of ten mice, in spite of a normal bisexual development of the trematodes, no eggs could be found in any of the organs of the hosts. In this experiment all cercariæ were shed by a single snail, *Australorbis glabratus*. The total number of cercariæ shed by the snail, which died the following day, was about 2,000, much higher than the usual number encountered in the average experiment, that is, to 600–700 a day. The snail itself was infected with 5–10 miracidia.

Eight mice were autopsied 55–140 days following infection and no ova were found in any of their organs. Perfusion of the liver and the mesenteric circulation[1] revealed that the 33–83 (average 65) schistosomes counted per mouse were of normal size, many of them copulated, with a ratio of 42–63 per cent (average 52 per cent) males. Slides of the fresh livers and intestines examined under microscope showed no eggs, nor did the trypsin–pepsin digestion of the organs followed by exposure to intense light in river water liberate miracidia[2]. The reproductive organs of the schistosomes stained with 0·005 per cent methylene blue solution were normal in size and number in the males, as were the ovaries and the vitelline glands in the females. However, in the uterus at the site where normally the ovum is located an irregular mass without shell was noted. Two of the mice of this group infected ten months ago are still alive, though animals infected with the same inoculum usually do not survive more than two months.

It has already been shown that induced nutritional deficiencies in guinea pigs[3] and in mice[4] may cause variations in the sexual organs of the schistosomes, and that in certain hosts[5–8] hermaphroditism of the male occurs; but we cannot find a precedent for complete sterility in mouse, a species allowing the development of a bisexual infection with deposition of eggs. We exclude nutritional influences in the present case and we think that the sterility, presumably due to the anomaly noted in the female schistosomes originated from cercariæ shed by the same snail, may have been determined by the miracidium. It is known that the sex of the parasite is predetermined in the miracidium, which forms the cercaria[9], and it could be assumed that the deformation resulting in sterility may depend on a genetic variation in the parent miracidium, which may have been the same individual for all the (female) cercariæ of the infection.

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