Research Note

Schistosoma japonicum infection in pregnant mice

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Abstract

Ten 1-week and ten 2-weeks pregnant female NMRI mice were experimentally exposed to 70 *Schistosoma japonicum* cercariae. Ten littermice from each group were examined for worms by perfusion 4, 6 and 8 weeks post infection. Although the mothers (n = 15) were found infected with 15.5 ± 13.4 worms at perfusion 6 and 7 weeks post infection, no worms were found in any of the examined littermice, as well as no detection of faecal or tissue eggs. Litter sizes did not differ from control groups and all littermice were healthy. The present study therefore suggests that congenital infection with *S. japonicum* does not occur in percutaneously infected mice and that infection of the mother during pregnancy does not seem to affect the offspring.

Reports of the congenital transfer of schistosomiasis in cattle, goats, dogs, rabbits, guineapigs and humans are found in Japanese literature from the beginning of this century (A. Ito, personal communication). Later studies showed five of eight mice congenitally infected under experimental conditions (Sakamoto, 1958). These results, however, seem to have been ignored for several decades until Willingham *et al.* (1999) recently showed that all 26 piglets born by sows experimentally infected with *Schistosoma japonicum* during week 10–11 of gestation were born with a patent infection. Most recently, studies in sheep (M.V. Johansen, unpublished) and rabbits (B.Z. Qian, unpublished) showed that prenatal infection with *S. japonicum* may occur in these animal species. The aim of the present study was to determine if mice can be prenatally infected with *S. japonicum* in 1-week and 2-week pregnant mice.

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Thirty 8- to 9-week-old primigravidae NMRI mice were used in the experiment. Fifteen of the mice were 1-week pregnant and 15 mice were 2-weeks pregnant at the time of infection. Ten pregnant mice from each conception date were each exposed to 70 *S. japonicum* cercariae using the paddling method, where the mice were left for 30 min in beakers containing fresh water and the cercariae. The *S. japonicum* (Zhejiang strain) had been maintained in *Oncomelania hupensis hupensis* snails (Jiangsu strain). The remaining five mice from each conception date served as uninfected controls.

The littermice were randomly divided into six groups. Groups 1–3 each contained ten mice born to mothers infected while 1-week pregnant; groups 4–6 each contained ten mice born to mothers infected while 2-weeks pregnant. At the time of examination the mice were killed by an overdose of Pentobarbital and perfused in order to recover worms (Smithers & Terry, 1965). After perfusion the intestinal mesenteries were examined for residual worms under a light microscope. Groups 1 and 4 were perfused 4 weeks post infection (p.i.), groups 2 and 5 were

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perfused 6 weeks p.i. and groups 3 and 6 were perfused 8 weeks p.i.. The mothers were perfused 6 and 7 weeks p.i. and their worms were enumerated. Tissue egg counts were determined on pooled tissue samples from the littermice according to the method described by Bjørneboe & Frandsen (1979). Faecal egg counts were determined 6 weeks p.i. on pooled samples from groups 2 and 5 according to the method described by Willingham *et al*. (1998).

The mean number of worms ± SD recovered from the mother mice (n = 15) was 15.5 ± 13.4. Gestation periods lasted the normal 19–20 days and mean litter sizes ± SD for infected mice were 11.1 ± 2.1. No significant difference between infected and controls were found. No worms were found by perfusion of the littermice; nor were any tissue- or faecal-eggs recorded in the examined samples. All littermice looked healthy and there were no indications of changes in the various organs examined at autopsy.

In the present study, we found no indication that the mice had been congenitally infected with *S. japonicum*; nor was there a greater frequency of abortions, stillbirths, neonatal deaths or changes in well-being in mice born by mothers infected during pregnancy. It seems therefore that mice are, in this respect, different from pigs (Willingham *et al*., 1999), sheep (M.V. Johansen, unpublished) and rabbits (Qian Bao Zhen, unpublished), where congenital infections were confirmed and even had clinical implications.

Several parameters, including differences in the placenta structure among species, previous exposure to the parasite, the number of pregnancies, as well as the time of infection during pregnancy and the dose of cercariae used might explain the differences in the results. However, the placenta in pigs contains several tissue layers on both the maternal and fetal side separating the blood, whereas the rodent placenta contains only layers on the fetal side (Björkman & Dantzer, 1989) and would be expected to be more easily penetrated by the schistosomula. Furthermore, since the time of infection and the dose of cercariae used in this study were comparable with the experiments made by Sakamoto (1958), where both congenital infections and increased numbers of abortions were seen in mice infected with 50–200 cercariae 1–8 days before delivery, other reasons might be sought. A marked difference between the studies, however, is that Sakamoto (1958) infected the mice by subcutaneous injection into the back, whereas, in the present study, percutaneous infection was used. The recent findings of congenital infection with *S. japonicum* in pigs (Willingham *et al.* 1999) and sheep (M.V. Johansen, personal communication) were also both a result of subcutaneous injection of cercariae. It could be speculated that penetration of the skin barrier makes the schistosomula less able to penetrate the placenta barrier. If so, this might explain the rare reports of congenital infections in nature.

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