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THE INFLUENCE OF CELL TRANSFERS (MESENTERIC LYMPH NODES, BONE MARROW) FROM ANIMALS INFECTED WITH *TRICHINELLA SPIRALIS* ON THE COURSE OF TRICHINELLOSIS IN RECIPIENT MICE *

KRYSTYNA KARMAŃSKA, ROMAN PIOTROWSKI, JAROSŁAW GRABIŃSKI and ZOFIA MICHALSKA

Research Centre of Parasitology, Polish Academy of Sciences, Wrocław
Institute of Infectious and Invasive Diseases, Agricultural Academy, Wrocław
Institute of Pathology and Therapy of Animals, Agricultural Academy, Wrocław

Research on the type of imminity which plays a dicisive role in intestinal helminthoses has been going on for a long time. One of the most important manifestations of immunologic processes in parasitoses is the expulsion of intestinal parasites, resulting in "self-cure". The attention of investigators has been focused on the mechanisms of this phenomenon. In order to assess the role of cell-mediated immunity, we have carried out experiments based on transfer of cells from infected or sensitized donors to recipient mice and studied the dynamics of expulsion. Thus far, this type of investigations have been carried out mainly on a model of *Nippostrongylus brasiliensis* and *T. spiralis*.

The present paper reports observations on the influence of cell transfers on the course of trichinellosis in recipient mice, basing on parameters which were not taken into account in previously published studies.

Material and methods

1. Animals. The study material consisted of 620 male mice of the RIII $\times$ $C57B_1/F_1$ strain, weighing 20 gm (± 0.7), aged over 3 months. Throughout the course of the experiment all mice were maintained under identical conditions.

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2. X-irradiation of recipients. Normal mice were exposed to doses of 600 R by total body X-irradiation at 130 kV, 6 mA, from a distance of 30 cm with a 2.5 Al filter. For 600 R, the dose rate was 1766.5 sec., as described in previous paper (Karmańska et al., 1977).

3. Transferred cells.

Lymph node cells. Lymphocytes were obtained from mice infected 24 days earlier, in which a state of DH [1] was demonstrated by means of the macrophage migration inhibition test. Mesenteric lymph nodes secured at autopsy, after comminution, were combined with a small volume of PBS of pH 7.2 and 4°C and rubbed through a double nylon filter (Blood filters, Technochemia, Warsaw). The centrifuged cells were washed twice with PBS, counted in a hemacytometer, and their viability was tested with trypan blue. The proportion of stained cells, i.e. dead cells was 8%. The recipient mice were injected i.p. with 64×10^6 living cells.

Bone marrow cells. After removing the epiphyses of femurs, the bone marrow was blown out with a thin Pasteur pipette into PBS of 4°C, and the marrow tissue was thoroughly broken up by repeated aspiration with a thin needle and syringe. The cells were washed twice with PBS, counted, and the proportion of living cells was checked. As many as 25% of the cells were dead. The recipient mice were injected i.p. with 100×10^6 living marrow cells.

4. Immunocytoadherence test. The reaction of lymphocytes with antigen-coated erythrocytes was set up by the method adapted for studies on trichinellosis by Płonka (1974) and Machnicka (1976).

5. Other methods. The technique of the macrophage migration inhibition test and method of preparing specimens for histopathologic study, and for observation of mast cells and counting parasites in the intestines were described previously (Karmańska et al., 1977).

Results

The mice were divided into four groups. After irradiation with 600 R X rays, the mice of groups 2, 3 and 4 were injected i.p. with 64×10^6 live mesenteric lymph node cells from infected donors. Groups 3 and 4, in addition to the above, were injected also with 100×10^6 bone marrow cells from healthy mice. All mice (including group 1, which served as controls of infection) were fed perorally with 100 *T. spiralis* larvae per mouse. Groups 2 and 3 were infected 4 days, and group 4 mice 10 days after cell transfer. Mice of each group were killed 5, 10,

[1] delayed hypersensitivity.

16, 22 and 31 days p.i. (post infection), and cells from the mesenteric lymph node were used to set up the macrophage migration inhibition and immunocytoadherence tests. At the same time, sections were stained for mast cells and histopathological examination. Adult trichinellae in the intestines of the mice were also counted.

Macrophage migration inhibition test. Cells from the mesenteric lymph nodes of the control mice (group 1) killed 5 days p.i. did not inhibit migration of the peritoneal macrophages. The percentage migration compared with the test without antigen was 104.0%. A similar result was obtained 10 days p.i., when the fields of macrophage migration measured 109.2%. Sixteen days p.i. the fields of migration were somewhat smaller (96.0%), but a definite inhibition of macrophage migration was not seen until the 22nd day p.i., when the size of the fields dropped to 65.0%. The last examination on the 31st day p.i. also showed presence of DH, although the migration fields at this time were 78.7% of the size of fields in the test without antigen.

In group 2 mice, migration was observed from the beginning. 5 days p.i. migration amounted to 68.6%, at 10 days p.i. 72.0%, and at 16 days 88.2%. On the 22nd day p.i. the migration fields of macrophages dropped to 78.4%, but at 31 days increased to 94.5%.

In group 3 mice, DH was detected on the 5th day p.i., when the migration of the peritoneal macrophages was 69.4% of the control values. At subsequent examinations, the mesenteric lymph node cells also inhibited macrophage migration. At 10 days p.i. migration was 75.8%, at 16 days p.i. 79.7%, at 22 days p.i. 79.8%, and at 31 days p.i. 76.3% compared with migration in the test without antigen.

In group 4 mice, DH was detected on the 5th day p.i. (69.1%), at 10 days p.i. 65.5%, and at 16 days p.i. 73.6%. On day 22 p.i., the migration fields were smallest, amounting to only 62.9% of the fields in the test without antigen. At 31 days p.i. the migration fields increased to 87.5% (Fig. 1).

Immunocytoadherence test. In the control mice (group 1) killed on the 5th day p.i., 4.5% on average of the mesenteric lymph node cells formed rosettes. On the 10th day p.i. their proportion increased slightly to 5.0%, but highest values were noted on the 16th day p.i., when 10.2% of lymphocytes formed rosettes. At the next examination on day 22 p.i., the proportion of rosette-forming cells dropped to 9.25%, and on the 31st day p.i. to 5.5%.

In group 2 mice, on the 5th day p.i. 12.0% of the cells from the mesenteric lymph nodes produced rosettes. On the 10th day p.i., the proportion of lymphocytes reacting with erythrocytes coated with *T.*

spiralis antigen dropped to 7.7%, and then remained on about this level: 7.0% at 16 days p.i., 6.0% at 22 days p.i. On day 31 p.i. the proportion of rosette-forming cells again increased to 9.2%.

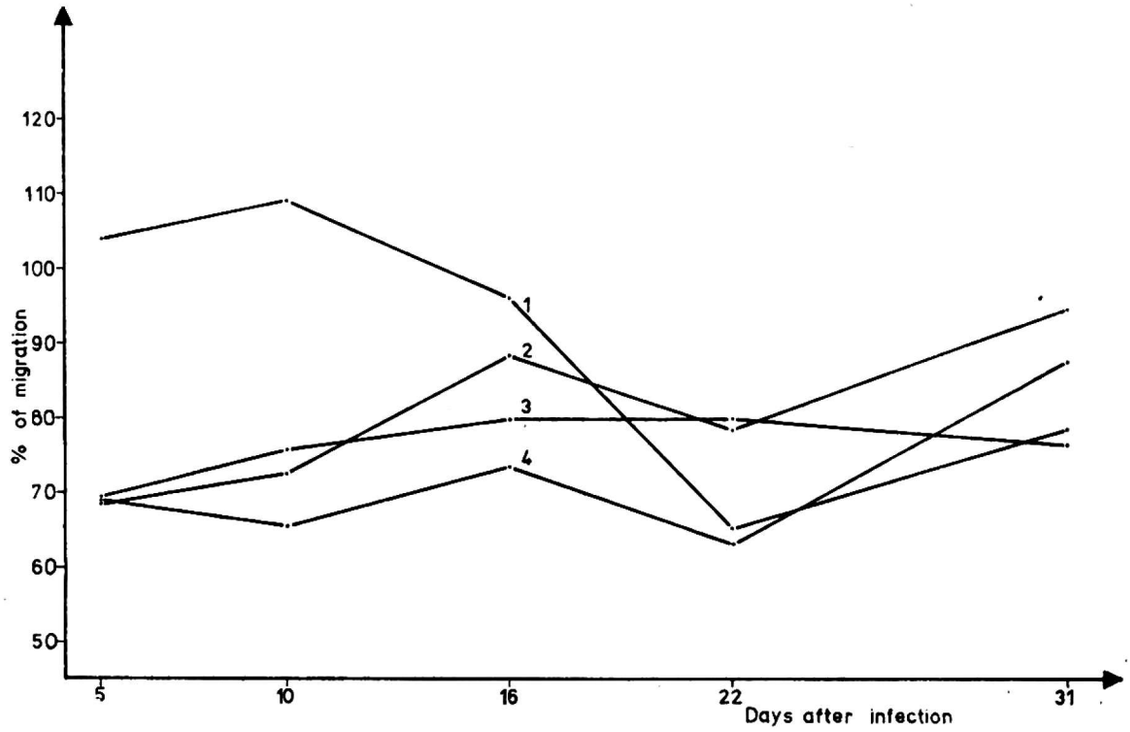

Fig. 1. Macrophage migration inhibition test
1 — group 1 (control), *2* — group 2, *3* — group 3, *4* — group 4

In group 3 mice, the percentage of rosette-forming lymphocytes from the mesenteric lymph nodes was high from the beginning. On the 5th day p.i., 11.5% of the cells reacted with erythrocytes, but on the 10th day p.i. dropped to 9.2, and on the 16th day to 8.2%. The lowest proportion of rosette-forming lymphocytes was noted on the 22nd day p.i. (6.0%), but on the 31st day p.i. increased to 9.0%.

In the group 4 mice, the behavior of rosette-forming cells from the mesenteric lymph node was different than in groups 2 and 3. High numbers of rosette-forming cells were noted on day 5 p.i. (13.2%), but decreased on day 10 p.i. (11.7%) and day 16 p.i. (11.25%). On day 22 p.i. the number of rosette-forming cells rose to 16.7%, and on day 31 p.i. dropped again to 10.0% (Fig. 2).

Mast cells in the jejunum. In the control group (group 1), the mean number of mast cells in the lamina propria of the intestinal mucosa on the 5th day p.i. was 63.2/sq mm, and on the 10th day p.i. rose to 102.1/sq mm, attaining a peak at 288.9/sq mm on the 16th day p.i. After that, the number of mast cells in the lamina propria declined to 150.7/sq mm on the 22nd day p.i., 48.4/sq mm on the 31st day p.i. Degranulation showed the same dynamics. On day 5 p.i. the mean number of degranulated cells was 27.1/sq mm on day 10 p.i. 41.0/sq mm and on day 16 p.i. 103.0/sq mm. In the mice killed on the 22nd day p.i. the mean

number of degranulated cells fell to 64.9/sq mm and on day 31 to 13.2/sq mm.

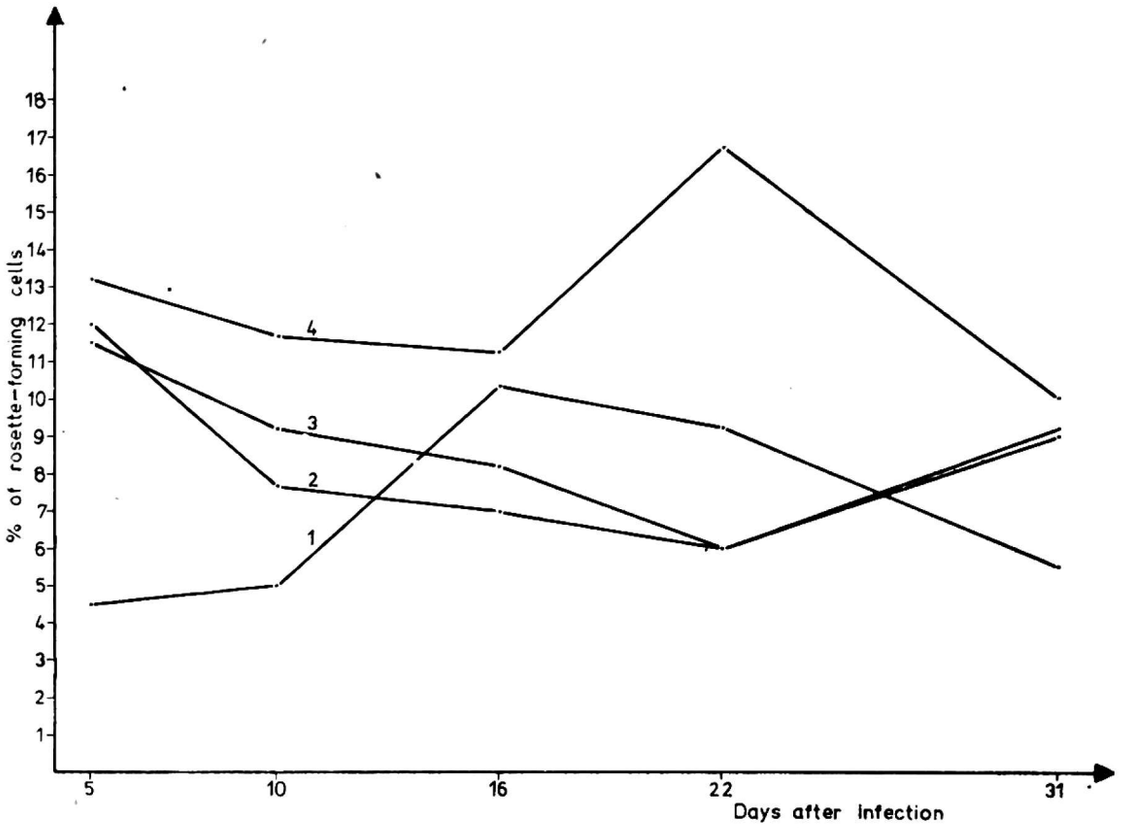

Fig. 2. Rosette-forming cells. Explanations as in Fig. 1

In the lamina propria of the intestinal mucosa of group 2 mice that were killed on the 5th day p.i. the mean number of mast cells per sq mm was 2.7, and remained at about this level throughout the experimental period: 3.8/sq mm on day 10 p.i., 2.7/sq mm on day 16 p.i., 8.8/sq mm on day 22 p.i., and 6.6/sq mm on day 31 p.i.. The numbers of degranulated intestinal mast cells were similar at all time intervals. In mice killed 5 days p.i., the numbers of degranulating intestinal mast cells was only 1.6/sq mm and on the 10th day p.i. 2.2/sq mm, on the 16th day p.i. 1.1/sq mm, on the 22nd day p.i. 5.5/sq mm, and on the 31st day p.i. 3.8/sq mm.

The results in group 3 mice were similar to those in group 2. The mean number of mast cells in the lamina propria of the intestinal mucosa in mice killed on the 5th day p.i. was 1.6/sq mm, 0.5/sq mm on the 10th day p.i., 1.1/sq mm on the 16th day p.i., 2.7/sq mm on the 22nd day, and 3.3/sq mm on the 31st day p.i. The levels of degranulated cells were also low. On the 5th day p.i. there were 1.1/sq mm degranulated cells, no degranulated cells on the 10th day p.i., and only 0.5/sq mm on the 16th day. On the 22nd day p.i. there were 1.1/sq mm, and on the 31st day 2.2/sq mm mast cells.

In the intestinal mucosa of the group 4 mice at 5 and 10 days p.i. the mean was 1.6/sq mm and on the 16th day still only 2.2/sq mm

but on day 22 p.i. rose abruptly to 136.4/sq mm. On the 31st day p.i. the number of mast cells in the intestine was still high 95.7/sq mm.

Dynamics of degranulation were similar. At first, the numbers of degranulated cells were low: 0.5/sq mm on the 5th day p.i., and 1.1/sq mm on the 10th and 16th day, and rose to 75.0/sq mm on the 22nd day p.i. On the 31st day p.i. the number of degranulated cells was 35.2/sq mm (Fig. 3).

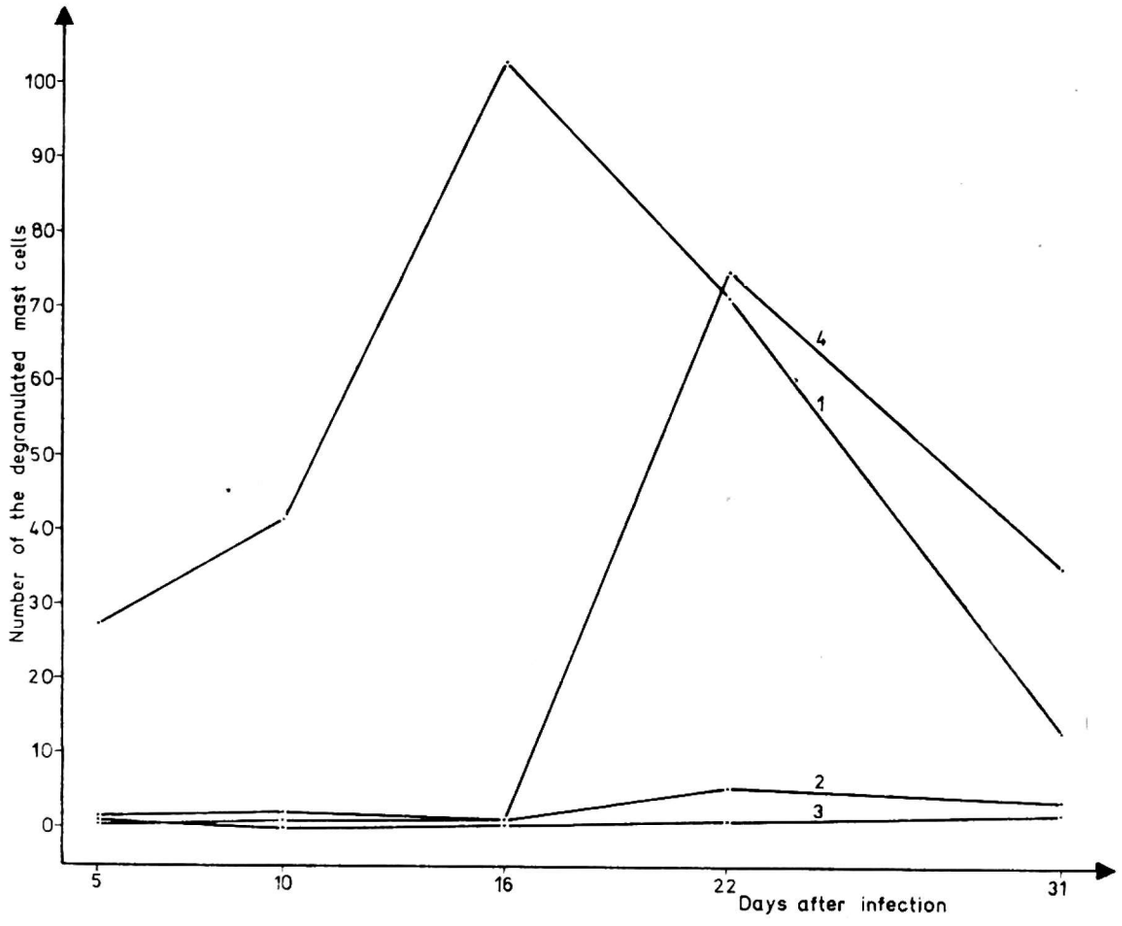

Fig. 3. Degranulated mast cells in jejunum Explanations as in Fig. 1

Histopathologic studies.

Jejunum. In group 1 mice (controls) on days 5 and 10 p.i. the stroma in the intestinal mucosa was moderately edematous and the lymph vessels were dilated. The apices of the villi were hyperemic, mucus production was somewhat increased, and infiltrates composed of histiocytes, lymphocytes and a few plasma cells were moderately numerous. The infitrates were scattered in the lamina propria of the mucous membrane of the larger intestinal villi. On the 16th day p.i. neutrophilic and eosinophilic leukocytes began to appear in the infiltrates, especially in the mucous membrane at the base of the villi. Starting on the 10th day p.i., Paneth cells began to multiply. On the subsequent days p.i. (22), inflammatory changes began to recede. Scanty inflammatory infiltrates with a greater admixture of leukocytes than before were observed in

only some of the villi of the mucous membrane, but no edema or hyperemia. From the 22nd day p.i. the Paneth cells showed no changes. On the 31st day p.i. the appearance of the walls of the jejunum in the control mice was normal.

In the group 2 mice, between days 5 and 31 p.i. the mucous membrane showed only moderate and mostly focal hyperemia. Serous edema of the villous apices was encountered 10 days p.i. The first scanty cell infiltrates composed almost exclusively of lymphocytes and histiocytes, appeared on the 5th day p.i. and persisted with unchanging intensity until the 31st day p.i. Infiltrates were observed only in the larger villi, and in some mice no infiltrates were present in the walls of the jejunum. Between days 5 and 16 p.i. the numbers of Paneth cells increased. A small number of neutrophilic and eosinophilic leukocytes appeared in the intestinal mucosa first at 31 days p.i.

In the group 3 mice, at 5 and 10 days p.i. moderate focal hyperemia and scanty lymphocytic and histiocytic infiltrates were observed in 50% of the villi. On days 16 and 22 p.i., hyperemia increased and serous edema appeared in the apices of some of the villi. Mixed infiltrates were fairly numerous at the base of the mucous membrane, and less often in its central part. The villi contained scanty infiltrates composed mainly of mononuclear cells. Between days 5 and 16 p.i. many Paneth cells were swollen and released their eosinophilic granules into the intestinal lumen. On the 31st day p.i. inflammatory changes such as edema, hyperemia and infiltration at the base of the mucous membrane diminished somewhat, but some infiltrates made up of lymphocytes and histiocytes were still seen in the lamina propria of the apices of the intestinal villi.

In the group 4 mice, 5 days p.i. fairly numerous infiltrates, composed mainly of lymphocytes and histiocytes, were observed in the lamina propria of the larger villi of the mucous membrane, but edema and hyperemia of the superficial layers of the intestinal wall appeared first on days 10 and 16 p.i., but were not pronounced. At this time the number of infiltrates was somewhat smaller than before, but they contained some leukocytes. Leukocytes were found also in the middle and basal layers of the mucous membrane. Increasing numbers of Paneth cells were observed on days 5, 10 and 16 p.i. On day 22, a few mixed infiltrates were seen only in the apical parts of the larger villi. On the 31st day p.i., infiltrates in the villi were encountered only sporadically.

Parasitologic studies. In the intestines of the control mice (group 1) trichinellae were found only at the first two examinations. The mean numbers of parasites per one mouse was 49.0 on the 5th day p.i., and

15.0 on the 10th day. No adult parasites were found after the 16th day p.i.

In the mice of groups 2, 3 and 4, parasites were present in the intestines until the end of the experimental period. In group 2, the mean number of parasites on the 5th day p.i. was 45.4, on the 10th day 38.5, 16th day 36.5, on the 22nd day 35.5 and on the last 31st day p.i., 24.0.

In the intestines of the group 3 mice, the mean number of parasites on the 5th day p.i. was 35.0. Similar numbers of trichinellae were found in the intestines of mice killed on the 10th day p.i. (34.0), 16th day p.i. (31.5) and 22nd day p.i. (31.0). At the last examination on the 31st day p.i. the number of parasites was 20.0.

In the group 4 mice killed 5 and 10 days p.i. the mean number of trichinellae was 47.0. On the 16th day this decreased to 20.5 and on the 22nd day p.i. dropped to 3.5. On the 31st day p.i. the mean number of intestinal parasites was 4.0 (Fig. 4).

Discussion

In recent years many investigators have studied the factors responsible for expulsion of adult parasites from the host's intestines, leading to "self-cure" in the course of intestinal helminthosis. Some authors attribute the main role to the mast cells (Jarrett et al., 1968; Miller, Jarrett, 1971), and others to DH (Kelly, Ogilvie, 1972; Kelly, Dineen, 1972a, b; Keller, Keist, 1972; Dineen et al., 1973a, b). A role of antibodies which damage the parasites before their expulsion is undisputed (Ogilvie, Hockley, 1968; Johnes, Ogilvie, 1971; Dineen et al., 1973a, b).

Proponents of the theory that cell-mediated immunity is responsible for expulsion of parasites from the intestines base their opinion on results obtained in recipient animals in which lymphocytes from infected or sensitized donors were injected (Larsh et al., 1964, 1966, 1969, 1970a, b, 1972, 1974; Dineen, Kelly, 1972; Kelly, Dineen, 1972; Dineen et al., 1973a, b; Ruitenberg, 1974). In these experiments, however, the transferred cells were an additional factor acting also on the intact immunologic apparatus of the recipients. It cannot be excluded that transfer of sensitized cell mobilizes elements of the defense mechanisms of the recipients. This conclusion is all the more likely because nearly all the cited studies (with the exception of the work of Larsh et al., 1974) did not include controls in which cells from healthy animals were transferred which could stimulate the immunologic system of the recipient.

The work of Kelly et al. (1973) and Dineen, Kelly (1973) on rats irradiated with 400 or 750 R before transfer (and before infection with *N. brasiliensis*) brought the first precise data. These investigators found

that the irradiated animals with 400 R (dose destroys lymphoid cells only in those animals) in which mesenteric lymph node cells were transferred from infected rats, leading to self-cure, but not in rats irradiated with 750 R. The dose of 750 R, besides destroying the cells of the lymphatic system, also destroyed bone marrow cells. Dineen and Kelly concluded that expulsion requires presence of normal cells of myeloid origin, besides sensitized lymphocytes.

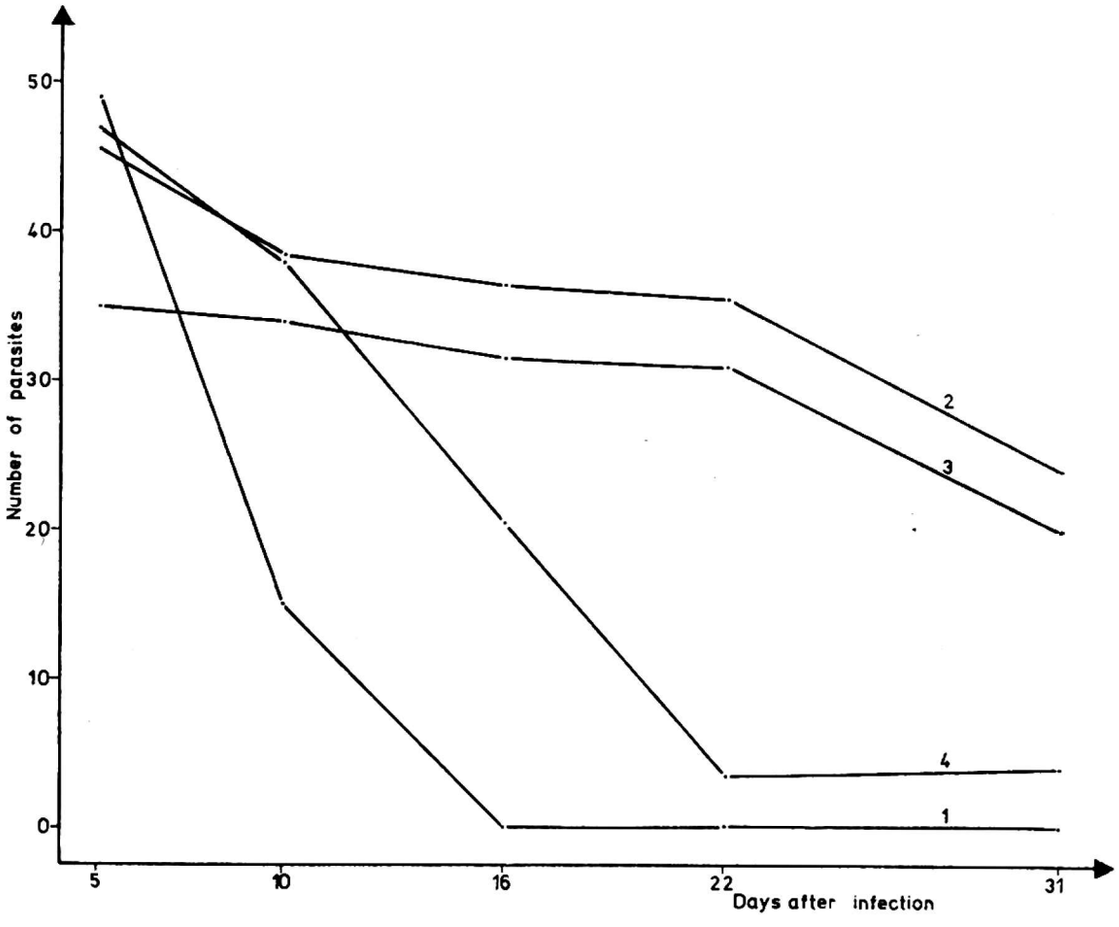

Fig. 4. Mean number of *T. spiralis* in the intestines. Explanations as in Fig. 1

These findings prompted our present study, which was designed to verify this hypothesis in *T. spiralis* infection, extending our observations on the mechanism of self-cure in trichinellosis. Most of our previous studies were concerned with the mast cells (Karmańska et al., 1973a, b, 1974; Karmańska, Michalska, 1975).

Our experiments were preceded by a study of the extent and stage of the histopathologic changes in the lymphoid organs and bone marrow of mice irradiated with varying doses of X rays. Our observations showed that the maximal dose after which mice of the RIII × $C57B_1/F_1$ strain survive was 600 R, and that the greatest damage of the lymphatic system and bone marrow was noted 3 to 20 days after irradiation.

Since it had been determined previously that RIII × $C57B_1/F_1$ mice expell adult trichinellae usually about 16 days p.i., groups 2 and 3 of mice were infected 5 days after irradiation (and 4 days after transfer),

basing on the assumption that expulsion takes place during the period of greatest damage of the defense mechanisms of the recipients. In group 4, our observations included the period of reversion to normal after irradiation. The mice were therefore infected 10 days after exposure to X rays (and after transfer).

According to the literature, the influence of cell transfers on the course of infection has been studied mainly on the basis of the dynamics of expulsion of intestinal parasites (in some studies, in addition, transfer of DH was followed by skin tests and antibody assays). We therefore designed our experiments to include detailed observations on the recipients which could measure the degree of transfer of immunity together with the cells (macrophage migration inhibition test) and the numbers of precursors of plasma cells (rosette test with erythrocytes coated with trichinella antigen), and on the other hand the behavior of mast cells. The observations were compared with the histopathologic changes (intensity and character of inflammatory infiltrates in the mucous membrane of the small intestine) and with the numbers of parasites in the intestines. In this way we obtained an accurate picture of the morphologic changes in the intestinal mucosa under the influence of *T. spiralis* infection.

By the use of this method we were able to show that the DH state, which in trichinellosis appears in the 3rd week p.i. (as reported by Kozar, Piotrowski, 1971; Piotrowski, 1975; Karmańska, Michalska, in printing) can be easily transferred to recipients. Proof of this was reported earlier by Kim (1966) and Kim et al. (1967a, b, 1970, 1974) and Larsh et al. (1974). In our experiments, proof of transfer of immunity with cells is provided by the fact that lymphocytes of the recipients as early as 5 days p.i. (9 days after transfer) inhibited migration of peritoneal macrophages.

We also noted that precursors of plasma cells were transferred, although their number diminished quickly, which is understandable because, as is known, they belong to the short-living lymphocytes. Their numbers again increased in the second phase of the experiment only in the group 4 animals, supposedly as a result of stimulation of the recipient's own immunologic mechanisms.

Histopathologic studies in all groups of experimental animals showed formation of inflammatory infiltrates, although with varying dynamics. In group 2 mice (which received only lymphocytes and were infected 4 days after transfer), the infiltrates were smaller, appeared 5 days p.i., and were composed mainly of mononuclear cells (leukocytes were not observed until the 31st day p.i.). On the other hand, in group 3 (transfer of lymphocytes and marrow cells and infection 4 days after

transfer) mixed infiltrates appeared 16-22 days p.i. In group 4 mice (which were injected with lymphocytes and bone marrow cells but infected 10 days after transfer), infiltrates at first contained lymphocytes and histiocytes, and leukocytes appeared first 10-16 days p.i.

In this situation, i.e. in the presence of DH and high levels of precursors of antibody-producing cells and inflammatory infiltrates, large numbers of trichinellae were found in the intestines of group 2 and group 3 mice until the end of the experimental period, and in mice of group 4 until the 16th days of infection.

Our results are discordant with those of Larsh et al. (1964, 1969, 1970a) and Love et al. (1976). Perhaps the difference would not have been so great if these investigators had also studied mast cells in their experiments.

It seems that the explanation of this phenomenon is probably linked with the behavior of the intestinal mast cells. In the first two groups of mice, no mobilization of mast cells as usually observed between days 10 and 20 p.i. with *T. spiralis* was noted (Karmańska et al., 1973a, 1974; Karmańska, Michalska 1975).

A similar situation was noted in group 4 mice until 16th day p.i. Hence, the cell transfers had no influence on regeneration of mast cells damaged by irradiation of the mice with 600 R. Reconstruction of mast cells, as shown in previous studies, began first after the irradiated animals had reverted to normal (i.e. about 20 days after irradiation). In group 4 mice the abrupt mobilization of intestinal mast cells 22 days p.i. is corroboratory evidence. In our experiments, the numbers of intestinal trichinellae were strictly correlated with the numbers of mast cells in the intestinal mucosa. The conclusion is suggested that mast cells (not necessarily through amines) play an important role in the process of expulsion, respectively that both phenomena are regulated by the same, hitherto unknown, factor, which causes their simultaneous occurrence.

A detailed comparison of our results with those of Larsh et al. (1964, 1969, 1970a) and Love et al. (1976) is not possible because these investigators did not study the mast cells.

In the present study, for technical reasons, a control group in which exclusively cells from healthy animals were transferred, was not included. However, earlier work by Piotrowski (1975) showed that transfer of such cells does not lower immunity to subsequent infection.

In summary, our results indicate that DH, which according to earlier studies at our laboratory (Kozar, Piotrowski, 1971; Piotrowski, 1975; Karmańska, Michalska, in printing) develops 3 weeks p.i. with *T. spiralis*, does not play a decisive role in expulsion of the parasites

from the intestines (as suggested earlier by Karmańska, Michalska, in printing).

Recent studies by Love et al. (1976) using young rats also show that expulsion depends not only on lymphocytes, and myeloid cells. May be a mast cell system insufficiently developed in young animals, is involved.

Another interesting observation is the lack of reconstitution of mast cells in animals irradiated with 600 R in which lymphocytes and marrow cells were transferred. This fact is not explained neither in the light of views that basophils are blood mast cells, nor by theory that mast cells originate from lymphocytes (Ginsburg et al., 1962, Csaba, Baráth, 1970; Ishizaka et al., 1976).

Authors' address:
50-375 Wrocław, C. Norwida 29
Poland

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WPŁYW TRANSFERÓW KOMÓRKOWYCH (WĘZŁY LIMFATYCZNE KREZKOWE, SZPIK KOSTNY) POCHODZĄCYCH OD ZWIERZĄT ZARAŻONYCH *TRICHINELLA SPIRALIS* NA PRZEBIEG WŁOŚNICY U MYSZY BIORCÓW

K. KARMAŃSKA, R. PIOTROWSKI, J. GRABIŃSKI i Z. MICHALSKA

Myszy szczepu RIII $\times$ C57B$_1$/F$_1$ o wadze około 20 g, w wieku 3 miesięcy napromieniowano dawką 600 R i podzielono na 3 grupy. Następnego dnia po napromieniowaniu zwierzętom z wszystkich trzech grup podano dootrzewnowo 64×10^6 komórek węzła chłonnego krezkowego od zarażonych dawców, u których stwierdzono obecność stanu NTP. Myszy z grupy 2 i 3 otrzymały dodatkowo 100×10^6 komórek szpiku kostnego od zdrowych dawców. Zwierzęta z grup 2 i 3 zarażono larwami włośni 4 dnia po transferze, a z grupy 4 — 10 dnia. Wszystkie grupy myszy (łącznie z grupą 1, która stanowiła kontrolę inwazji) zarażono dawką 100

larw/mysz. W 5, 10, 16, 22 i 31 dniu po zarażeniu (p.z.) zwierzęta uśmiercano i nastawiano z komórkami węzła chłonnego krezkowego odczyn zahamowania migracji makrofagów oraz test immunocytoadherencyjny. Z wycinków jelita czczego sporządzano preparaty histologiczne oraz preparaty dla obserwacji komórek tucznych. Ponadto liczono pasożyty dorosłe.

U myszy z grup 2, 3, i 4 obserwowano przeniesienie stanu NTP, komórek tworzących rozetki w teście immunocytoadherencyjnym oraz tworzenie się nacieków zapalnych w błonie śluzowej jelit. U zwierząt zarażonych 4 dnia po transferze (grupy: 2 i 3) nie obserwowano mobilizacji komórek tucznych do 31 dnia p.z., a dorosłe pasożyty przeżywały do końca obserwacji. Natomiast w grupie 4, którą zarażono 10 dnia po transferze, obserwowano wyraźną mobilizację komórek tucznych w 22 dniu p.z., w tym samym czasie stwierdzono też znacznie mniejszą ilość dorosłych włośni.