with a 0.8° cone attached (Tokyo Keiki Co.). The kinetic parameter of hemoglobin deoxygenation (the velocity constant, k_c) upon mixing the air-saturated erythrocytes with the hydrosulfite solution[10,11] was measured by a stopped flow apparatus (Union Giken Co.).

The representative results are summarized in tables 1 and 2. The 'deformability' decreases as the cholesterol/phospholipid ratio increases. A slight but definite increase of the viscosity of the cell suspension, in particular at higher shear rates, is noticed with the cholesterol-loaded erythrocytes (table 1). In a separate experiment, decreased motion of a stearate spin label has been observed in the cholesterol-loaded erythrocyte membrane[14].

Furthermore, the velocity constant of oxyhemoglobin dissociation was reduced, as the membrane cholesterol was augmented (table 2), in spite of the similarity a) of the oxygen equilibrium curve (as expressed by P_{50} and n), b) of the cell shape (e.g., MCV and MCH, also the microscopic observation) and c) of the amounts of the allosteric effectors (such as 2,3-diphosphoglycerate, ATP and intracellular pH; though the amounts of 2,3-diphosphoglycerate decreased during the sample preparation in this case).

Increase in membrane cholesterol 1. immobilizes the acyl chain motion of the phospholipids and reduces the membrane fluidity[1-3], and 2. may change the membrane protein organization[15], therefore the erythrocyte deformability decreases. The slight increase of the suspension viscosity under higher shear rates, observed with the cholesterol-loaded cells, may be explained by the decreased ability of passive deformation to reduce the rheological resistance. The increased blood viscosity observed with hyperlipoproteinemias[16], might be due to the increased membrane cholesterol.

The retarded rate of oxygen egress from the cholesterol-loaded erythrocytes may result from the decreased rate of oxygen diffusion in the lipid portion of the membrane, since Fischkoff and Vanderkooi[17] have shown that the diffusion rate of oxygen molecules is decreased in the cholesterol-containing phospholipid vesicles.

In conclusion, an increase in erythrocyte membrane cholesterol not only affects the rheological properties such as deformability and suspension viscosity, but also reduces the rate of oxygen egress from the cells.

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Hymenolepis nana: Transfer of acquired immunity in mice through sensitized peritoneal exudate cells

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Summary. Sensitized peritoneal exudate cells from syngeneic donor Swiss albino mice infected with single and repeated doses of viable eggs of *Hymenolepis nana* produced a significant adoptive immunity in test mice when compared with animals which received non-sensitized (normal) cells. No significant difference was observed among the 2 recipient groups which received singly or repeatedly sensitized peritoneal exudate cells.

Hymenolepis nana, (the dwarf tapeworm) is known to be highly immunogenic[2], and in the mouse immunity can be detected at various intervals after primary infection[3]. Reports of passive transfer of immunity directed against *H. nana* from infected donors to uninfected animals are very few. Male C_3 B F_1/cum mice irradiated after normal expulsion of adult *H. nana* worms and injected with syngeneic normal bone marrow cells showed persistance of acquired immunity 1 day after irradiation[4]. The role of CMI was fully established when Friedberg et al.[5] demonstrated transfer of acquired immunity to X-irradiated recipients through immunized spleen cells from previously infected donors.

While the present authors were engaged in investigations involving CMI in the *H. nana* mouse model, it was found that sensitized peritoneal exudate cells from infected donors produced a measurable amount of acquired immunity in syngeneic recipients challenged with *H. nana* eggs.

Materials and methods. Collection and transfer of peritoneal exudate cells were in accordance with the method of Vardhani and Johri[6]. Approximately 26×10^4 cells were transferrred within 4 h of collection. Syngeneic female recipient Swiss albino mice, of approximately the same body weight and age (6-8 weeks and 15-20 g) were divided into 5 groups of 30 mice each. Groups a and b received cells from singly, d and c from doubly infected and e from uninfected (control) donors. These recipient groups were further divided into 3 subgroups, each challenged with a single dose of 1000 *H. nana* eggs at 7, 14 and 21 days after cell transfer. Infection of donor mice, injection of sensitized cells, and challenge were carried out according to table 1. Mice were necropsied on the 4th day (96 h) after challenge.

Table 1. Experimental protocol of infection with *Hymenolepis nana* donors and recipient mice groups

Days	Sensitizing dose of *H. nana* eggs to donor groups				
	Single infection		Repeated infection		Uninfected controls
	A	B	C	D	E
0	100	1000	50	500	-
1	-	-	50	500	-
4	Collection and transfer of peritoneal exudate cells from donors to recipient				
11	Challenge infection of 1000 eggs to each mouse of a_1, b_1, c_1, d_1 and e_1 subgroups (10 in each)				
18	Challenge infection of 1000 eggs to each mouse of a_2, b_2, c_2, d_2 and e_2 subgroups (10 in each)				
25	Challenge infection of 1000 eggs to each mouse of a_3, b_3, c_3, d_3 and e_3 subgroups (10 in each)				

Table 2. Percentage recovery of *Hymenolepis nana* cysticercoids from recipient mice each injected with approximately 26×10^4 peritoneal exudate cells and challenged with 1000 eggs at different periods after cell transfer

Recipients challenged: Days after cell transfer	Recipients with singly sensitized cells		Recipients with repeatedly sensitized cells		Recipients with nonsensitized cells
	a	b	c	d	e
7	0.8 (18.9031)*	0.40 (20.1224)*	0.84 (19.1190)*	0.65 (20.0366)*	3.60
14	0.68 (19.9880)*	0.35 (25.4582)*	0.72 (19.9881)*	0.45 (21.0312)*	3.30
21	0.54 (17.2242)*	0.20 (23.8864)*	0.60 (15.3834)*	0.30 (21.4684)*	2.90

(Figure in parentheses shows the χ^2 values). Readings are based on mean of recoveries made from 10 animals. Tabulated χ^2 value is 3.841 at 1 degree of freedom and 5% level. * Statistically significant.

The degree of immunity was assessed by cysticercoid counts according to Hunninen[2].

Results and discussion. Cysticercoid counts made from experimental and control recipient groups are shown in table 2. Experimental recipient exhibited strong adoptive immunity when compared with controls. Lowest average percentage of recovery (0.20) was recorded from experimental recipients of cells derived from donors sensitized with a single dose of 1000 eggs and challenged 21 days after cell transfer. Maximum average percentage (0.84) was recorded from recipients of repeatedly sensitized cells from donors and challenged 7 days after cell transfer. Groups a and b, i.e. the recipients of cells from singly sensitized donors, showed maximum average percentage recoveries of 0.80 and 0.40, respectively, 7 days after cell transfer. Similarly, recipient groups with cells from repeatedly sensitized donors gave maximum average percentage recoveries of 0.84 and 0.65, respectively, 7 days after cell transfer. Subsequent recoveries from all these groups gradually decreased on days 14 and 21 after cell transfer. Low counts were obtained from recipients with singly sensitized cells; however, the difference was not statistically significant when compared with their counterpart recipients of repeatedly sensitized cells. Cysticercoids obtained from recipients (with singly sensitized cells) challenged on days 7 and 14 after cell transfer (0.8, 0.68 and 0.40, 0.35) were comparable to those obtained from recipients with repeatedly sensitized cells on days 14 and 21 after cell transfer (0.72, 0.60 and 0.45, 0.30).

The results indicate that acquired immunity was transferred by sensitized peritoneal exudate cells to the recipient groups of mice in this model. This was attested by the failure of development of large numbers of challenged eggs. Experimental recipient groups a and b (singly sensitized cells) and c and d (repeatedly sensitized cells) showed a statistically significant immune response when compared to normal (control) recipients. In all experimental recipient groups, transferred sensitized cells migrated to the gut, resulting in a dense accumulation at the base of the intestinal villi; they sensitized the gut wall, which became l unsuitable for most of the eggs to survive and/or develop into cysticercoids[7]. Thus, these transferred sensitized cells added an additional immune response. Control recipients also showed migration of cells, however, devoid of any sensitizations, they allowed a large number of cysticeroids to become established. The cellular response and the predominance of polymorphonuclear neutrophils around the developing cysticeroids in the intestinal villi of mice infected with *H. nana* has already been demonstrated by Bailey[8]. Arrested development of cysticercoids may also be accounted for by degranulation of mast cells, resulting in allergic inflammation and alteration of the biochemical environment, preventing larvae from migrating to their usual site of development in the host. This may create unfavourable local conditions for the parasite[9]. Participation of macrophages in the immune response of mice infected with *H. nana* was suppressed by using antithymocyte serum (ATS); this suggests that suppressive effects are exerted early in the immune response and probably on antigen sensitive, thymus-derived T-cells (Okamoto and Koizumi[10]). The fact that maximum response was established in recipients challenged 21 days after cell transfer, showed that immune response is a gradual process which takes some time.

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