PLATELET ANTIGENS IN DOGS

S. M. Belotskii

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Four antigens specific for platelets and unconnected with antigens of red and white blood cells, were distinguished in dogs by the thromboagglutination test. Inheritance of platelet antigens in dogs is Mendelian in type and is controlled by several recessive genes.

KEY WORDS: platelet antigens; transfusion complications.

The study of the isoantigenic composition of the blood cells of dogs began in 1910, when antigens of the red cells (blood groups) were identified and the character of their inheritance was demonstrated [2]. The detailed study of dogs' blood groups was undertaken in 1951 [7]. Later, antigens of dogs' white blood cells (tissue incompatibility antigens) were isolated [5]. These investigations made it possible to use dogs as models with which to study many diseases, especially those connected with the role of immunological reactions. However, besides the isoantigens of the blood cells mentioned above, other antigens specific for platelets are also known to exist. Such antigens have been distinguished, in particular, in man [3, 4, 6].

The object of this investigation was to study platelet antigens in dogs.

EXPERIMENTAL METHOD

Altogether 224 unrelated mongrel dogs and 12 families with a total of 64 puppies were tested. Red cell antigens were determined with the aid of sera obtained by the author and also with sera provided by Dr. Vriesendorp (The Netherlands), using a micromodification of the hemagglutination test: 1 μl of red cell suspension ($250 \cdot 10^3$ cells) was mixed with an equal volume of serum, and the results were read after incubation for 15 min at 37°C under the microscope (magnification 160×). Antigens of the white blood cells were determined by a micromodification of the lymphocytotoxic test, using a kit consisting of sera obtained by the author and by other participants in the Second International Working Conference on Immunogenetics of Dogs (Portland, 1974). The thromboagglutinating sera, with the aid of which platelet antigens were isolated, were identified by the crossed thromboagglutination test: 2 μl of a platelet suspension ($250 \cdot 10^3$ cells/μl) were mixed with the volume of serum, shaken for 90 min at room temperature, and the results were read under the microscope.

EXPERIMENTAL RESULTS

Tests of the sera of 224 dogs revealed 11 samples that gave a reproducible thromboagglutination reaction. From these sera four were selected which, in the familial tests (Table 1), revealed platelet antigens present in the parents and the puppies. Adsorption of these sera (Table 2) showed that the thromboagglutinins were directed against antigens specific for platelets, but not against antigens of red or white blood cells. The results of the familial analysis and adsorption indicate that dog platelet antigens exhibit group properties.

A study of the distribution of platelet antigens 1, 4, 5, and 9 in 124 unrelated mongrel dogs showed that they were present in 25.8, 10.16, 15.32, and 18.54% of animals, respectively.

Investigation of the relationships between platelet antigens and their relationships with antigens of red and white blood cells (Table 3) showed positive correlation between the T1 antigen and DLA = B6 and C11 antigens, and between T4 antigen and DLA = A1 and B5 antigens, i.e., these factors are found together most frequently. The same was found for T1 and DED1 antigens. No marked correlation was found between the platelet

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TABLE 1. Inheritance of Platelet Antigens in Mongrel Dogs

	Antigen			
	1	4	5	9
Ratio between number of the same litter possessing the particular antigen and the total number of puppies	16/33	5/15	10/31	11/27
Number of families	4	2	4	4
Example of inheritance:				
mother	+	+		
puppies				
1	+	+		
2	+			
3	+			
4		+		
5				
6	+			
7		+		
8	+			
9				

TABLE 2. Adsorption of Thromboagglutinating Sera by Dogs' Blood Cells and Blood

Serum	Experimental conditions	Thromboagglutination
1	Without adsorption	+
	After adsorption by blood cells of dog positive with serum 1:	
	by red cells	+
	by white cells	+
	by platelets	—
9	Without adsorption	+
	After adsorption by blood cells of dog negative with serum 9:	
	by red cells	+
	by white cells	+
	by platelets	—
	After adsorption by platelets of dog negative with serum 9	+

TABLE 3. Coefficients of Correlation for 4 T Specificities ($n = 78$, $r_{95} \geq 0.23$), 14 DLA Specificities ($n = 34$, $r_{95} \geq 0.35$), and 5 DEA Specificities ($n = 65$, $r_{95} \geq 0.25$)

		Antigens of platelets			
		1	4	5	9
Tissue incompatibility antigens of DLA system	A1	—0,30	0,41	0,02	—0,15
	A2	—0,15	0,09	0,10	0,11
	A3	0,01	0,21	—0,17	0,18
	B4	—0,11	0,19	0,07	0,18
	B5	0,15	0,34	—,013	—0,33
	B6	0,31	—0,15	—0,11	0,11
	A7	—0,13	—0,10	—0,08	—0,12
	A8	—0,32	—0,15	0,20	0,11
	A9	0,13	—0,01	—0,07	—0,01
	A10	—0,33	0,05	—0,25	—0,33
	C11	0,34	—0,16	—0,04	—0,30
	B12	0,11	—0,02	—0,02	—0,34
	B13	—0,28	0,00	—0,17	—0,40
	R15	—0,19	0,12	—0,11	—0,18
Antigens of red blood cells	DEA 1—2	0,02	—0,22	0,02	—0,11
	DEB 1	0,16	—0,06	0,10	0,10
	DEC 1	—0,04	—0,10	—0,19	—0,20
	DED 1	0,24	0,12	—0,06	0,14
	DEF 1	—0,08	—0,13	0,13	—0,19
Antigens of platelets	T1				
	T4	—0,19			
	T5	—0,14	—0,04		
	T9	—0,32	—0,22	0,17	

antigens themselves either on familial analysis or on mass examination, with the exception of the relatively less frequent mutual distribution of T1 and T9 antigens.

Four antigens specific for platelets were thus distinguished in dogs. These antigens have a Mendelian type of inheritance which is under the control of several genes (Table 1). Antibodies against dogs' platelet antigens can give rise to transfusion reactions [1].

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CHARACTER OF THE STIMULATING ACTION OF ANTILYMPHOCYTIC SERUM IN THE EARLY STAGES OF RESTORATION OF HEMATOPOIESIS IN THE SPLEEN OF RADIATION CHIMERAS

K. K. Zaitseva and M. M. Nezdatnyi

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The object of this investigation, conducted on 236 BALB/c mice, was to study the effect of antilymphocytic serum (ALS) on the early stages of restoration of hematopoiesis in the spleen of radiation chimeras, having regard to the fact that ALS increases the number of macroscopic hematopoietic colonies. The results showed that this increase is connected with intensification of the first stage of restoration of hematopoiesis, i.e., with acceleration and intensification of the activation process of the reticular cells of the recipient's spleen.

KEY WORDS: radiation chimeras; spleen; antilymphocytic serum; hematopoietic colonies.

Restoration of the ability to form morphologically identifiable hematopoietic cells, when disturbed by irradiation, in the spleen of mouse radiation chimeras takes place in three stages: First, the reticular cells are activated, then microcolonies of hematologically undifferentiated blast cells are formed, and this is followed by the appearance of differentiated hematopoietic cells [1].

Antilymphocytic serum (ALS), obtained 24 h before irradiation and subsequent bone marrow transplantation, approximately doubles the number of hematopoietic colonies arising in the spleen after 8 days [2].

The object of this investigation was to study the effect of ALS on restoration of hematopoiesis in radiation chimeras.

EXPERIMENTAL METHOD

Experiments were carried out on 236 male BALB/c mice. The animals of group 1 (control) were irradiated once with γ rays on a cobalt (^{60}Co) apparatus in a dose of 750 rad with a dose rate of 80 rad/min. Each animal received an intravenous injection of 10^5 syngenetic bone marrow cells 24 h after irradiation. The mice of group 2 (experimental) were irradiated in the same dose and received an injection of the same number of myelokaryocytes. These mice also received a subcutaneous injection of rabbit antimouse ALS 24 h before the injection of bone marrow cells in a dose of 0.25 ml per mouse.

The mice of both groups were decapitated 2 h and 1, 2, 3, 4, 5, and 6 days after the transplantation of bone marrow cells and a morphological analysis was made of the cell composition of the red pulp of the spleen and the number of colony-forming units (CFU) in the spleen was counted [1].

EXPERIMENTAL RESULTS

Subcutaneous injection of ALS into the mice caused rapid death of the lymphocytes in the white pulp of the spleen, and this evidently delayed their postradiation recovery. By 48 h after injection of the ALS only the

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