streptococcal antigens, heterophilic antibodies against human myocardial ICT antigens can be obtained, whereas antibodies against rabbit heart ICT are not formed. Yet bound immunoglobulins are found in ICT of immunized rabbits, which may be evidence of the presence of a "latent" antigen in ICT [4]. The problem of the mechanisms of formation of heterophilic antibodies against myocardial ICT in human sera is still unexplained and requires further study.

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EFFECT OF ADRENALECTOMY ON RECIPIENTS OF ALLOGENEIC LYMPHOCYTES ON INACTIVATION OF ENDOGENOUS COLONY-FORMING CELLS IN MICE

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T lymphocytes are the principal cells inducing delayed-type hypersensitivity reactions, and on interaction with allogeneic stem cells, they can inactivate them [5, 6]. It has been shown that positive correlation exists between inactivation of endogenous colony formation and transplantation immunity reactions [7, 8]. In endogenous hydrocorticism induced by bilateral adrenalectomy manifestations of transplantation immunity reactions are intensified [3], the number of endogenous colony-forming cells in the spleen is increased [4], with a shift of their differentiation toward erythropoiesis [2], and it is consequently interesting to study the killer activity of lymphocytes toward endogenous colony-forming cells in adrenalectomized recipients.

The aim of this investigation was to study the killer functions of lymph node cells directed against endogenous colony-forming cells in adrenalectomized recipients in a genetic system with one-way incompatibility: parental line — F_1 hybrid.

EXPERIMENTAL METHOD

The donors of lymph node cells were C57BL/6 mice, the recipients (CBA × C57BL/6)F_1 mice. Mice aged 3-4 months were obtained from the Stolbovaya Nursery, Academy of Medical Sciences of the USSR. The adrenals were removed through a midline skin incision in the lumbar region, with division of muscles in the right and left hypochondria and with approach to the upper poles of the kidneys in order to remove the glands. The mock operation consisted of the manipulations mentioned above with the exception of removal of the adrenals. After the operation

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TABLE 1. Effect of Adrenalectomy on Killer Activity of C57BL/6 Lymph Nodes Cells in the Phenomenon of Endogenous Colony Inactivation in (CBA × C57BL/6)F_1 Hybrids

Day of investigation	No. of expts.	No. of mice	Dose of C57BL/6 lymph node cells (× 10^6)	No. of endogenous colonies in (CBA × C57BL/6)F_1 mice (M ± m)		P	Inactivation index (%)
				adrenalectomy	mock operation		
9-th	2	25	2	17,1±0,85	—	<0,01	63
		17	—	27,2±0,82	—		
		26	2	—	9,8±1,08	>0,05	4,2
		18	—	—	10,6±1,14		
9-th	1	15	5	0,4±0,29	—		
		10	—	28,4±2,3	—	<0,01	98,6
		15	5	—	4±1,57	<0,01	43
		10	—	—	12,2±0,62		
8-th	1	15	5	1,13±0,71	—	<0,01	92,7
		10	—	13,7±1,15	—		
		15	5	—	4,46±0,83		
		10	—	—	5,7±0,9	>0,05	23,5

the mice were given a 0.85% solution of common salt to drink. The (CBA × C57BL/6)F_1 hybrids were irradiated 2 days after adrenalectomy or the mock operation in a dose of 600 R on an ÉGO-2 apparatus, and this was followed by intravenous injection of lymph node cells from the parental C57BL/6 line in a dose of 2×10^6 or 5×10^6 nucleated cells. The mice were irradiated with ^{60}Co γ-rays on the ÉGO-2 apparatus (experimental gamma-ray source) with dose rate from 200 to 250 R/min. Mice for irradiation were kept 20-30 at a time in plastic cages with perforated walls. Endogenous colony-forming cells were identified in the spleen of the mice on the 8th or 9th day after sublethal irradiation. The spleen was placed in Bouin's fixing fluid for 30 min and the colonies were counted macroscopically. The technique of preparing the suspension of lymph node cells was the same as that described previously [3]. The results were subjected to statistical analysis by Student's t test [1].

EXPERIMENTAL RESULTS

Lymph node cells from C57BL/6 mice in a dose of 2×10^6 had virtually no effect on endogenous colony formation in F_1 hybrids undergoing the mock operation (Table 1). This same dose of lymph node cells inhibited endogenous colony formation in adrenalectomized hybrids ($P < 0.01$); adrenalectomy itself, moreover, caused an increase in the number of endogenous colonies in the recipients' spleen to more than twice their number in mice undergoing the mock operation. An increase in the dose of lymph node cells from C57BL/6 mice injected to 5×10^6 was accompanied by inhibition of endogenous colony formation in F_1 hybrids undergoing the mock operation, but the degree of inhibition of endogenous colony formation was higher in the adrenalectomized recipients. When the number of endogenous colonies in the adrenalectomized recipients and recipients after the mock operation was determined on the 8th day after injection of 5×10^6 C57BL/6 lymph node cells, statistically significant inhibition of endogenous colony formation was observed at that time in the adrenalectomized recipients ($P < 0.01$), but not in mice undergoing the mock operation ($P < 0.05$). Just as in the previous experiment, with this dose of lymph node cells endogenous colonies were absent in the spleen of most experimental adrenalectomized mice. It can be tentatively suggested that the killer action of T lymphocytes on endogenous colonies was intensified in adrenalectomized recipients with endogenous hypocorticism, as a result of cooperation with the cortisol-sensitive subpopulation of T helper cells, of a change in the properties of the antigen-recognizing receptors, or an increase in the sensitivity of target cells to the killer action of T lymphocytes.

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