In conclusion, the results reported here show that: a) The ferrihemoglobin reductase activity increases largely on incubation with IPP, and in particular 30 days old erythrocytes show, after incubation, a velocity of reduction which is twice that of fresh cells. However, this value is 3 times lower than that obtained by incubation of fresh erythrocytes, implying that by ageing the reductase systems are irreversibly modified. It is not yet clear where this block is localized, since the reduction as a function of time of ferrihemoglobin reductase activity of IPP incubated red cells depends not only on the intrinsic reductase system but also on the entire chain of enzymes and cofactors interposed between the substrates applied and methemoglobin. b) The oxygen affinity is lowered on incubation with IPP; the value of $p^1/_2$ obtained after rejuvenation is, however, the same for blood of different age. c) By incubation with IPP, the increased osmotic fragility of outdated blood tends towards the normal range, even though it is not fully restored.

Antigenic correlation between rat thymus and superior cervical ganglion[1]

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Summary. By using rabbit anti-rat thymocyte and anti-rat superior cervical ganglion sera in cytotoxicity, immunofluorescence and absorption assays, it has been shown that surface membranes of rat thymocytes and cervical ganglion cells (i.e. peripheral nervous tissue cells) contain common antigenic determinants.

The brain-thymus antigen[2] has been described in various tissues of the rat[3,4]. The subject of this paper is to demonstrate antigenic correlation between the rat thymus and the rat superior cervical ganglion.

Materials and methods. The sympathetic superior cervical ganglia were isolated from Wistar rats perfused with saline. The fraction which predominantly contained disrupted ganglion cell membranes and fibrillar structures was employed for the production of anti-cervical ganglion (anti-CG) serum in rabbits[5]. Rabbit anti-CG sera used in this experiment were absorbed with rat erythrocytes, liver-cell membranes, glutaraldehyde-insolubilized serum proteins, and kidney homogenate as previously described[6]. Absorbed anti-CG sera when tested against the corresponding antigen exhibited antibody titers between 1:512 and 1:2048 in complement-fixation reaction[7] using 5 50% haemolytic units of guinea-pig complement, and produced 3 precipitin lines in double diffusion in 0.8% agarose[8].

Thymocytes were separated from the thymus of saline-perfused Wistar rats and purified on Isopaque-Ficoll[9]. Anti-thymocyte serum (ATS) was prepared in rabbits[9] and absorbed in an identical manner as described above

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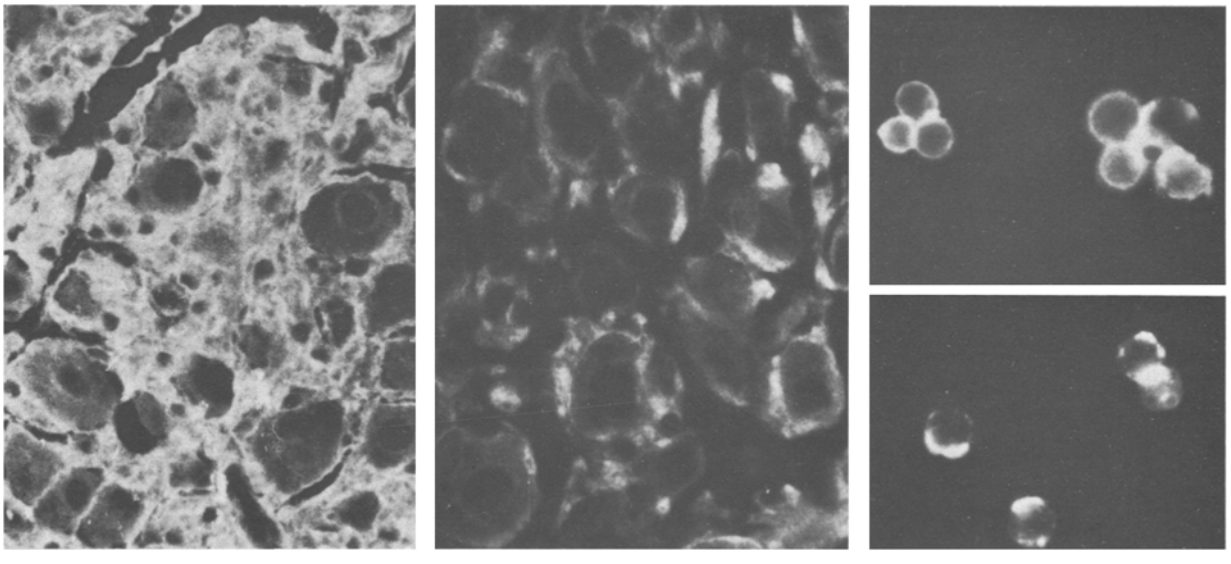

Immunofluorescence microphotographs. *a* A section of the rat superior cervical ganglion (×300) exposed to anti-rat cervical ganglion serum and fluorescein-conjugate. *b* A section of the rat cervical ganglion (×400) treated with anti-rat thymocyte serum and conjugate. *c* 2 groups of rat thymocytes (×800) treated with anti-rat thymocyte serum and conjugate. *d* 4 specifically fluorescing rat thymocytes (×800) exposed to anti-rat cervical ganglion serum and conjugate.

Cytotoxic activity and binding capacity of rabbit anti-rat cervical ganglion and anti-rat thymocyte sera for rat thymocytes and B-lymphocytes

Cells used in test	Cells used for absorption	Cytotoxic index (%) Antiserum dilution (log_2) 1	2	3	4	5	6	7	Percent of fluorescein-positive cells
		Anti-rat cervical ganglion serum							
Thymocytes	None	82.6	62.4	27.1	10.5	3.0	0	0	58.2
	Thymocytes	3.4	0	0	0	0	0	0	0.2
B-lymphocytes	None	3.1	1.9	2.4	0	0	0	0	0.3
	Thymocytes	1.4	0	0	0	0	0	0	0.2
		Anti-rat thymocyte serum							
Thymocytes	None	99.1	97.9	97.1	91.0	47.5	16.4	3.8	99.6
	Thymocytes	2.4	0	0	0	0	0	0	0.2
B-lymphocytes	None	58.2	46.8	24.5	6.7	0	0	0	60.3
	Thymocytes	2.8	0.5	0	0	0	0	0	2.1

Each figure represents mean value of 3 independently performed experiments. At least 1000 lymphocytes/preparation were counted in cytotoxicity and immunofluorescence assays.

for anti-CG serum. The cytotoxic activity[10] of anti-CG and ATS was tested against rat thymocytes and B-lymphocytes. B-cells were obtained from the femoral bone marrow of 6-week-old rats thymectomized at birth and irradiated at the age of 2 weeks with 700 R. Paraffin sections[11] of the rat cervical ganglion and viable thymocytes[9] were employed in the indirect fluorescent staining[12] using a sheep fluorescein-conjugated anti-rabbit IgG serum[13]. Several controls were set up in order to distinguish clearly specific green fluorescence from nonspecific fluorescence[14].

Results and discussion. The cytotoxic activity and binding capacity of rabbit anti-rat cervical ganglion and anti-rat thymocyte sera are shown in the table. Anti-CG serum was cytotoxic for thymocytes at a 1:8 dilution, and induced specific fluorescence of about 58% of thymocytes, whereas this antiserum was completely inactive for B-lymphcytes. On the other hand, ATS killed or stained specifically about 100% of thymocytes and about 60% of B-lymphocytes. Absorption of anti-CG and ATS with 10^9 thymocytes/ml of antiserum removed all of antibody activity to thymocytes and B-lymphocytes.

The cervical ganglion sections exposed to anti-CG (figure, a) or ATS (figure, b) and fluorescein-conjugate exhibited specific fluorescence of neuronal membranes, whereas neuronal cytoplasm and nuclei remained unstained. This fluorescence was significantly stronger with tissue sections treated with anti-CG serum. As for thymocytes, ATS induced a bright specific fluorescence of the ring-type (figure, c). Thymocytes exposed to anti-CG serum displayed a specific staining of irregular shape, most of fluorescein-conjugate molecules being concentrated on a portion of the surface membrane (figure, d). All control tissue sections and thymocytes preparations were negative.

These results show the presence of common membrane antigens on the surface of rat thymocytes and superior cervical ganglion cells, thus indicating that the thymus and the peripheral nervous tissue share at least some antigenic determinants[2].

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Electrical stimulation induces clot retraction after previous in vitro platelet aggregation

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Summary. The spontaneous clot retraction of platelet-rich plasma is inhibited by previous in vitro ADP-induced platelet aggregation. The electrical stimulation of the clot always restores a maximal clot retraction, even after a prolonged previous in vitro platelet aggregation.

Earlier work showed that previous in vitro platelet aggregation inhibits spontaneous clot retraction[2]; the degree of inhibition is related to the degree of aggregation. However, if the aggregation is followed by a rapid and complete disaggregation, subsequent spontaneous clot retraction is not inhibited[2].

These facts suggested that platelets have not to be aggregated before or during clotting in order to support a normal clot retraction. It was supposed that the inhibition of clot retraction by previous platelet aggregation could be due to the lack of a random distribution of the

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