COMBINED PROTECTIVE ACTION OF NONIMMUNE SPLENOCYTES AND ANTIVIRAL ANTIBODIES DURING EXPERIMENTAL POLIOMYELITIS (TYPE II) VIRUS INFECTION IN MICE

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The role of humoral response during poliovirus infection has been thoroughly studied, whereas the mechanism of cellular protection have been scarcely investigated (*1,6*). Here evidence is given in favour of interaction of specific antiviral antibodies with nonimmune splenocytes in the resistance against poliomyelitis virus type II in mice.

BALB/c mice weighing 18—20 g were given with cyclophosphamide (CPM) (s.c., 200 mg per kg) 24 hr before intracerebral inoculation of poliomyelitis virus type II strain MEF-I (3×10^5 TCD_{50}/ml). Simultaneously the animals were inoculated either with 0.3 ml of rabbit immune serum at dilution 1 : 32 dilution (titre 1 : 320), or with $3-4 \times 10^7$/0.3 ml of nonimmune syngeneic splenocytes, or with both, into tail vein.

It can be seen from the Table that CPM pretreatment was without effect on the death rate of infected animals. Inoculation of splenocytes from syngeneic uninfected donors (group 3) or of nonspecific serum alone (group 4) was also without effect. Combined inoculation of splenocytes and subprotective doses of antibodies, hovewer, was associated with marked protective effect (group 5). Most recipients appeared to have clinical signs of neuroinfection (pareses or paralyses). The death rate was as high as the half of other groups.

Group of animals	Number of animals	Recipients infected with poliomyelitis type II treated with			Death rate (per cent)	p
		CPM	Splenocytes	Serum		
Ist	45		—	—	100	
2nd	45	+	—	—	100	
3rd	45	+	+	—	100	
4th	45	+	—	+	93.3	
5th	45	+	+	+	42.2	≤ 0.001*

* Statistical significance by Student's test.

Combined protective effect of subprotective doses of antibody and nonimmune effectors has been previously described and attributed to the relatively well studied phenomenon of antibody-dependent cytotoxicity (*2—5*). The data presented indicate that this mechanism can be regarded as one of basic components of protective action of specific immune response at early stages of poliovirus infection reducing the death rate of animals infected directly into CNS.

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