fect of the electrolytes was less pronounced. The greatest capacity for immunity stimulation by polymers was exhibited by mice of the C57BL/6 line, while this capacity correspondingly lower with mice of the C57BR, C3H, and CBA lines [5]. Additionally, for the detection of the immunostimulating effect of extracts of thymus and other organs, an immunization with a dose of $10^7$ erythrocytes was carried out. A comparative study of the effect of a complex of polyadenylic-polyuridylic acid and a dextran sulfate, as well as combinations thereof, showed that at doses of $5 \times 10^8$-$10^10^8$ sheep erythrocytes there was a twofold increase in the number of antibody-forming cells, while at immunization doses of $2 \times 10^8$-$4 \times 10^6$ cells the immunological response was stimulated more than tenfold with respect to the control [8].

Thus, it can be concluded that the use of animals with a low response to sheep erythrocytes, particularly those of the C57BL/6 line, for testing immunotropic activity as well as immunization with an antigen dose of $10^8$-$10^6$ cells is a more promising method of assessing correctly the activity of a preparation and its future potential as well as increasing the coefficient of its useful selection. Additionally, the suggested experimental technique for detecting the immunotropic activity appears to be universal as it allows one to detect both immunosuppressants and immunostimulators in one experiment.

We believe that the given method can be applied to the initial testing of chemical compounds for immunological activity as well as for clarifying the side effects and the long-term effects of the administration of pharmacological agents exhibiting biological activity.

LITERATURE CITED

MECHANISM OF THE NEUROTROPIC ACTIVITY OF ANALOGS
OF γ-AMINOBUTYRIC ACID, L-Dopa, AND L-5-HYDROXYTRYPTOPHAN

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The formation and utilization of γ-aminobutyric acid (GABA) is regulated by the pyridoxal enzymes: glutamate decarboxylase (GDC; EC. 4.1.1.15) and transaminase (TGABA; EC. 6.1.1.19). The formation of dopamine and serotonin from L-Dopa and L-5-hydroxytryptophan, respectively, takes place with the pyridoxal decarboxylase of the aromatic amino acids (EC 4.1.1.26). The hydroxy acids (I-IV) discussed in this work are structurally similar to the above-mentioned amino acids and can be considered as their analogs.

A hydroxyl group replacing an amino group can interact with the pyridoxal phosphate carbonyl group to form hemiacetals. This process can compete with the natural interaction of the pyridoxal phosphate carbonyl group with the amino group of the amino acid leading to the formation of a Schiff base, which is an indispensable step in the chain of conversions essential to a biological reactions. Thus, the basis for the neurotropic activity of the hydroxy analogs could be their effect on the metabolism of the physiologically active amino acids. (See scheme on next page.)
Compound I was prepared from α-butyrolactone [1]. Compound II was prepared by reacting nitrous acid with L-glutamic acid [2, 3].

The acid III (racemate) was prepared in the following manner: The reaction of N-acetyl-glycine with 3,4-dihydroxybenzaldehyde yielded 2-methyl-4-(3,4-diacetoxybenzylidene)oxazolone [4], which was converted into 3,4-dihydroxyphenylpyruvic acid by acid hydrolysis and, in its turn, was reduced to the desired α-hydroxy acid III in the presence of Raney nickel catalyst [5].

Compound IV (racemate) was prepared from the ethyl ester of α-methylacetic acid, obtained from dimethyl sulfate and β-dimethylaminocrotonic ester [6], by a Japp-Klingemann reaction with the benzyl ether of p-aminophenol; indolization of the hydrazone obtained was carried out, without isolation, with hydrogen chloride [7]. The obtained ethyl ester of 5-benzyl-oxyindolcarboxylic-2 acid was saponified and the acid was decarboxylated by heating with activated copper in quinaldine [8]. 5-Benzyloxyindole was converted into 5-benzyloxygramine [9], which was further converted into 5-(5-benzyloxyindoly1-3)-α-hydroxy-α-carboxypropionic acid after its condensation with the diethyl ester of acetoxymalonic acid and the saponification of the carboxethoxy and acetoxy groups [10]. The acid obtained was then decarboxylated in quinoline in the presence of copper. The benzyl blocking group was removed by hydrogenation of the 5-benzyloxy derivative in the presence of palladium on barium sulfate. Acid IV was obtained as a crystallizing yellow oil (cf. [10]).

Acids I-III were obtained as sodium salts. Acid IV was dissolved in a calculated amount of sodium bicarbonate before the use.

**EXPERIMENTAL BIOLOGICAL PART**

*In vitro* investigations were carried out on the effect of the hydroxy analogs on the activity of the pyridoxal enzymes participating in the metabolism of the corresponding amino acids: compound I on the transaminase of GABA from pig kidneys (TGABA) [11]; compound II on the glutamate decarboxylase (GDC) from the acetonated powder of *Clostridium welchii* [12]; compounds III and IV on the decarboxylase of the aromatic amino acids from mouse liver [13]. The hydroxy analogs were investigated in concentrations of 10⁻³ M, and higher. Such concentrations are reached in the tissues on systematic administration of the compounds in doses causing a clear pharmacological effect.

Using a Hitachi spectrophotometer in the range 300 to 450 nm, we evaluated the adsorption spectra of pyridoxal phosphate in the presence of amino acids in combination with the hydroxy analogs. Data were recorded 1-10 min after mixing the solutions in the cells at 20°C in a 0.1 M phosphate buffer of pH 7.3. The substances were mixed in the concentrations