# ACID PHOSPHATASE (PHOSPHOMONOESTERASE II) ACTIVITY IN RAT BRAIN DURING TRAUMATIC SHOCK

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As the result of detailed investigations carried out with rapid methods of ultramicrobiochemical analysis, the topography of cerebral enzymes has been studied at length. Considerable attention was paid to the distribution of enzymes of the esterase cycle in morphologically and functionally different sections of the central nervous system. However, up to this time the role of phosphomonoesterases in the nervous tissue has not been fully elucidated. Their activity in the cerebrum during different functional states of the central nervous system has not been sufficiently investigated. Data which have been obtained relate to changes in the activity of acid and alkaline phosphatase in the whole brain of animals during stimulation and depression [12].

Recently, the localization of acid phosphatase to the subcellular fraction of the brain has been well studied [13, 15]. Similar to the distribution in the liver, the major acid phosphatase activity was detected in the cytoplasmic portions and much less in the supernatant.

According to some data [11] the catalytic activity of acid phosphatase is not altered during embryonic development of the brain, but falls in the post-embryonic period.

The importance of studying phosphomonoesterase is related to its suggested role in the metabolism of phospholipids which enter into the composition of the myelin sheath of the nerve fiber [2, 14] and into the metabolism of hexose phosphates and the transphosphorylation reaction [2].

We have studied the activity of acid phosphatase (phosphomonoesterase II) 3.1.3.2* in homogenates of rat cerebral hemisphere and brainstem during traumatic shock.

## METHODS

The work was carried out on white male rats of weight, 160-180 gm, of which 9 animals served as controls and 10 were subjected to traumatic shock. Studies on control and experimental animals were made in parallel at the same time, using identical reagents and apparatus.

Shock was produced by the method devised by E. A. Asratyan [1] by multiple traumatization of both hind legs. The development of shock was recorded by measurements of blood pressure, electroencephalogram, observation of thermoregulation and behavior. Ten minutes after the trauma the animals were decapitated and tissue from the cerebral cortex and brainstem was removed in the cold. Portions of the brain were rapidly suspended in 0.3 M sucrose and cooled in ice, cut into fragments with 0.3 M sucrose added to obtain a 10% tissue suspension. Homogenization was carried out in a glass Potter homogenator with careful cooling for 90 sec at 800-1000 rpm. The homogenate obtained was diluted 10 times with double distilled water and was frozen. After 24 h the homogenate (1 :100) was subjected to thawing and was again frozen.

Measurements of the acid phosphatase activity was made by the micro-rapid method of A. A. Pokrovskii and A. M. Shcherbakova [10] on homogenate diluted to 1 : 200. The substrate was paranitrophenylphosphate. The

* Classification and nomenclature of enzymes [2].

TABLE 1. Acid Phosphatase (Phosphomonoesterase II) 3.1.3.2 Activity in Homogenates of Cerebral Tissues and Liver of Control Rats (in microMoles/g/min)

| Brain | | Liver |
|---|---|---|
| cerebral cortex | Brainstem | |
| 0,932 | 0,326 | 2,20 |
| 0,886 | 0,268 | 2,40 |
| 0,690 | 0,338 | 1,32 |
| 0,747 | 0,175 | 2,16 |
| 0,654 | 0,350 | 1,56 |
| 0,700 | 0,373 | 1,84 |
| 0,723 | 0,431 | 2,76 |
| 0,723 | 0,303 | 2,08 |
| 0,816 | 0,303 | 1,56 |
| $M \pm m$ 0,763±0,030 | 0,318±0,024 | 1,99±0,153 |
| $\sigma$ ±0,091 | ±0,071 | ±0,46 |

TABLE 2. Effect of Traumatic Shock on Acid Phosphatase 3.1.3.2 (Phosphomonoesterase II) Activity in Homogenates of Brain Tissues of Rats (in microMoles/g/min)

| Cerebral cortex | Brainstem |
|---|---|
| 0,735 | 0,373 |
| 0,886 | 0,338 |
| 0,700 | 0,245 |
| 0,630 | 0,268 |
| 0,676 | 0,431 |
| 0,724 | 0,373 |
| 0,768 | 0,422 |
| 0,945 | 0,373 |
| 0,897 | 0,326 |
| $M \pm m$ 0,773±0,038 | 0,350±0,021 |
| $\sigma$ ±0,114 | ±0,063 |

reaction was carried out in citrate buffer, pH 5.0, at 25° for 60 min. For enzyme activation 0.1% of $MgCl_2$ solution was added. The reaction was halted by adding 0.1% NaOH. The amount of free paranitrophenol was determined on a FEK-H 57 electrophotocalorimeter using special filters and a microcuvette [8]. The results are expressed in micromoles per quantity of wet tissue.

## RESULTS

A comparative study of acid phosphatase in tissue of brain and liver in control rats showed that the enzyme activity was 2.6 times higher in liver than in the cerebral cortex and 6 times greater than in the brainstem tissue (Table 1) which is in agreement with the data obtained by other authors [9, 13]. It must be noted that magnesium ions do not produce activation of hepatic acid phosphatase. The enzyme activity in the cerebral hemisphere was 2.4 times greater than in brainstem, in accord with data indicating the predominance of activity of enzymes of carbohydrate metabolism and oxidative enzymes in the phylogenetically younger brain structures [3, 5].

In Table 2 are presented data concerning the measurement of acid phosphatase activity in homogenates of rat brain during traumatic shock. As seen from Table 2, the acid phosphatase activity of the cerebral cortex does not change during shock but increases in the brainstem by a statistically valid 10% ($P < 0.01$). We have previously found [4] that during traumatic shock the intensity of oxidative phosphorylation in mitochondria isolated from the brainstem is significantly increased. A rise in the brainstem acid phosphatase, which participates in the transphosphorylation reaction, evidently attests to its important role in the energy metabolism of the brain during shock.

According to some authors [14], acid phosphatase in the central nervous system is bound to glial structures. It is possible that the increase in its activity in brainstem tissues is a result of increased glial cell activity in the reticular formation of the brainstem during shock.

## SUMMARY

Experiments on female albino rats were used to study the influence of traumatic shock on the activity of acid phosphatase (phosphomonoesterase II) in the tissue homogenates of the brain cortex and brainstem. In control animals the activity of acid phosphatase was twice as high in the brain cortex as in the brainstem. In traumatic shock, an increased activity of the enzyme was noted in the brain stem.

## LITERATURE CITED

1. É. A. Asratyan, Essays on the etiology, pathology and therapy of traumatic shock [in Russian], Moscow (1945).
2. Classification and nomenclature of enzymes [in Russian], Moscow (1962), p. 127.
3. A. V. Palladin and B. L. Khaikina, In book: Progress in biological chemistry [in Russian], Moscow, 2 (1954), p. 27.

4. L. F. Panchenko, In book: Material from the first Conference of traumatologists-orthopedists of the Baltic Republics, Riga [in Russian] (1964), p. 366.
5. Z. D. Pigareva, Acta Univ. Carol. Med. (Praha), 8 (1962), p. 366.
6. A. A. Pokrovskii and L. G. Ponomareva, Biokhimiya, No. 2 (1962), p. 276.
7. A. A. Pokrovskii, In book: Papers of the Sectional Meetings of the Fifth International Biochemical Congress [in Russian], Moscow, 1 (1962), p. 7.
8. Idem, In book: Chemical bases of vital processes [in Russian], Moscow (1962), p. 311.
9. L. F. Pomazanskaya, Dokl. AN SSSR, 132, No. 5 (1960), p. 1197.
10. N. N. Pushkina, Biochemical methods of investigation [in Russian], Moscow (1963), p. 224.
11. L. B. Fleksner, Acta Univ. Carol. Med. (Praha), 8 (1962), p. 513.
12. E. Makovskii, K. Shelariu, S. Mikhésku et al., Ukr. biokhim. zh., No. 1 (1958), p. 18.
13. C. Allard, G. de Lamirande, and A. Cantero, Cancer Res., 17 (1957), p. 862.
14. K. P. Strickland, R. H. S. Thompson, and G. R. Webster, J. Neurol. Neurosurg. Psychiat., 19, (1956), p. 12.
15. N. Waked and S. E. Kerr, J. Histochem. Cytochem., 3 (1955), p. 75.

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**All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal.** ***Some or all of this periodical literature may well be available in English translation.*** **A complete list of the cover-to-cover English translations appears at the back of this issue.**

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