

## THE INFLUENCE OF PYRACETAM ON THROMBOCYTOPOIESIS IN RATS

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A number of effects of Pyracetam other than its antihypoxic effect and influence on memory and process of learning have been discussed recently. It is known that it stimulates erythropoiesis, improves red blood cell deformability (2,6), but it also suppresses the "in vitro" platelet activation and aggregation and the release of platelets f.4 and beta-thromboglobulin from granules (4,7).

This study aims at investigating the influence of Pyracetam on thrombocytopoiesis in experiments "in vivo" especially as these things have not been studied.

We observed 33 experimental and 35 control female white "Wistar" rats, weighing 170-180 g. The experimental ones were treated for 3 days, twice a day i.p. with 200 mg/kg of Pyramem (Pharmachim, Sofia) dissolved in distilled water. The platelets were counted according to phase-contrast chamber method (1). Seventy-two hours before the end of the experiment all the rats were injected with  $10\mu\text{Ci}$

<sup>75</sup>Selenomethionine i.p., in order to define the percentage of its incorporation in the newly formed platelets (8) - this is an index reflecting the rate of thrombocytopoiesis. The study of megakaryocytes (MKC) upon bone marrow smears, coloured after Gimsa's method has been done using terms of three-degree scale. The data were analysed according to variation analysis methods using the t-criterion of Student-Fisher.

Fig.1 shows that the number of platelets in the rats treated with Pyracetam decreases by 17,11 % ( $p < 0,001$ ) against the control group.

The incorporation of <sup>75</sup>Selenomethionine in the newly formed platelets of the experimental and control animals does not differ substantially. The total number of megakaryocytes in 200 myelokaryocytes tends to decrease by 9,02 % in the Pyracetam treated rats. The partial megakaryocytogram shows a decrease of almost all forms of MKC - especially of the diploid megakaryocytoblasts, basophilic and polychromatophilic MKC (in contrast to them oxyphilic MKC tend to increase by 28,57 % (fig.2). Our data show that the established junctional depressing changes in platelets after treatment with Pyracetam are accompanied also by a depression of thrombocyto-

poiesis and megakaryocytopoiesis. Probably, the decreased influx of cells from the precursory fund of cells of MKC caused the lower number of MKC in the experimental rats. We would explain the decrease of platelets in peripheral blood with the possible decreased pinching of the latter by grown oxyphilic MKC - this probably causes their increase. The thrombopoietin which regulates the pinching of the platelets of MKC has been described (3,5). As to the intimate mechanism of Pyracetam influence on this cell system - we find it difficult to give an answer to this question. These changes in the activity of plasma thrombopoietin might lie behind these alterations and that will be the aim of our forthcoming investigations.

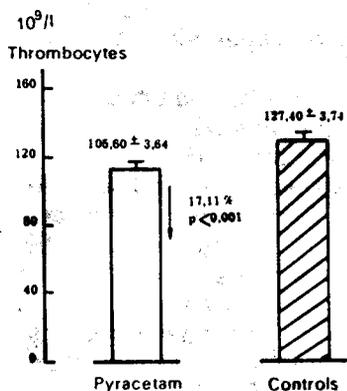


Fig. 1. The data are presented as mean ± SE

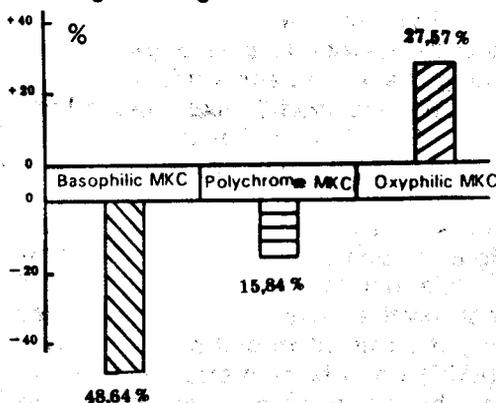


Fig. 2. The data are presented as a per cent deviation to control levels accepted as 0. The sign (+) means increase but the sign (-) diminution

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