In this regard, conducted a series of experiments associated with the induction of lipid peroxidation of liver microsomes enzymatic (NADP • H –dependent) mechanisms in the incubation medium, in the presence of lipids of liver microsomes and individual presence nicotine, gexametonium in the incubation medium.

The conducted work that nicotine incubation medium molar concentration of 10 -4 M and 10 -5 M ; 10 -6 M in during of the induction of enzymatic (NADP • H –dependent) mechanisms of POL leads to a decrease in lipid oxidation in the microsomes of the liver and decreasing the molar concentration of nicotine in the incubation environment, the ability of the lipids of liver microsomes to oxidize increases was showed.

The presence gexametonium (10 -4 M ; 10 -5 M ; 10 -6 M) in the incubation medium with lipids of liver microsomes, in contrast to nicotine had led to the opposite effect – to increase the ability to oxidize lipids of liver microsomes in the activation of enzymatic mechanisms POL and decreasing the molar concentration of hexametoni in the incubation environment, the ability of the lipids of liver microsomes to oxidize increased.

Thus, the presence of nicotine in the incubation medium while induction enzymatic mechanisms for POL reduces the ability of the lipids of liver microsomes oxidation and decreasing the molar concentration of nicotine in the incubation medium noted an increase in the ability of the lipids of liver microsomes to oxidize was.

Gexametonium the presence in the incubation medium resulted in opposite in respect to the nicotine effect is to increase the ability of the lipids of liver microsomes by oxidation and reduction of molar concentration gexametonium in the incubation medium led, in the case of nicotine to increase the ability of the lipids of liver microsomes to oxidize.

Summarizing the experimental data we can note the following - the presence of pharmacological agents - nicotine, gexametonium in the incubation medium under the induction of enzymatic mechanisms for POL leads to different trends of oxidation in lipids of liver microsomes.

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LIPID LIVER OXIDATION INDUCED BY NONENZYMATIC MECHANISM IN VITRO IN THE PRESENCE OF NICOTINE, HEXAMETHONIUM

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Keywords: nicotine, gexametonium, nonenzymatic mechanisms oxidation , microsomes, in vitro

Abstract. Work´s carried out to determine the ability to oxidize lipids of liver microsomes nonenzymatic mechanisms in the presence of nicotine, gexametonim were. The results showed – nicotine in an incubation medium of a molar concentration of 10-4 M increases the oxidation of lipids microsomes, molar concentration of nicotine of 10-5M, 10 -6M decrease a microsomal lipid oxidation.

Hexamethonium of the incubation medium (10-4 M, 10-5 M, 10-6 M) reduces the ability to oxidize.

Experimental work out to determine the ability of nicotine, hexamethonium to influence the oxidation of lipids liver microsomes during the activation of enzymatic (NADP • H-dependent) LPO mechanisms in vitro was carried.

Data testify to a multidirectionality of the results was received. The enzymatic mechanisms LPO of the incubation medium in the presence of nicotine induction of decreases, and the ability of lipids liver microsomes to oxidize hexamethonium was increases.

Effect of the nicotine, hexamethonium on the peroxidation of liver lipids in inducing non-enzymatic (ascorbate-dependent) mechanics was remained unresolved.

The results of the experiments that the presence of nicotine in an incubation medium of a molar concentration of 10-4 M increases the oxidation of lipids microsomes of the liver was showed.

Molar concentration of nicotine of 10-5M, 10-6M in the incubation medium decrease a microsomal lipid oxidation when lipid microsome oxidation induced by nonenzymatic mechanisms was.

Hexamethonium of the incubation medium (10-4 M, 10-5 M, 10-6 M) when inducing non-enzymatic mechanisms of LPO reduces the ability of liver liposomes to oxidize, the decrease in the molar concentration of hexametonium both,

and in the case of nicotine reduces the ability of liver microsomes to oxidize .

Thus, the activation of non-enzymatic mechanisms of lipid peroxidation in vitro in the presence of nicotine, hexamethonium leads, in the case of nicotine, to an increase in the oxidation of lipids microsomes of the liver, and in the case of hexamethonium, to a decrease.

Reducing the molar concentration of nicotine in the incubation medium leads to a decrease in lipid oxidation of liver microsomes and a decrease in the molar concentration of hexametonium in the incubation medium also leads to a decrease in the ability of liver microsomes to oxidize.

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OXIDATION OF LIPIDS LIVER MICROSOMES BY ENZYMATIC AND NON-ENZYMATIC MECHANISMS IN VITRO IN THE PRESENCE OF NICOTINE, HEXAMETHONIUM, AFTER HEAT TREATMENT OF LIVER MICROSOMES

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Keywords: nicotine, gexametonium, enzymatic mechanism oxidation , non enzymatic mechanism oxidation, microsomes, heat treatment.

Abstract. Work´s carried out to determine the ability to oxidize lipids of liver microsomes after heat treatment by nonenzymatic mechanisms and by enzymatic mechanisms in the presence of nicotine, gexametonim were. The results showed – reduces the ability of the liver microsomes to oxidize in the presence of nicotine when inducing the enzymatic mechanisms, in the presence of hexomethonium the lipids of the liver microsomes become less sensitive to oxidation. Nicotine of a molar concentration of 10-4 M leads to a slight increase in oxidative activity, and in molar concentrations of 10-5 M, 10 -6 M prevents the oxidative activity of non-enzymatic mechanisms of LPO. Hexamethonium of a molar concentration of 10 -4 M does not significantly decrease the ability of lipids of liver microsomes to oxidize, and at 10 - 5 M, 10 -6 M molar concentrations increases the ability of liver microsomes to oxidize.

Of protein components of plasma membranes hepatocyte to participate in the LPO of the liver the opportunity was assessed. It suggested that the protein formations of the membranes of the endoplasmic reticulum of hepatocytes (liver microsome‘s) may also participate in the LPO process. In the series of experiments carried out, in order to switch off the protein structures from lipid oxidation, liver microsomes membranes subjected to heat treatment at a temperature of + 80 0 C was.

Lipid microsomes of the liver subjected to heat treatment, in the presence of nicotine, hexamethonium, oxidation in vitro carried out with the induction of enzymatic (NADP • H-dependent) and non-enzymatic (ascorbate-dependent) LPO mechanisms was.

The results obtained indicate the presence of nicotine, in an incubation medium with a molar concentration of 10-4 M; 10-5 M; 10-6 M after heat treatment of liver microsomes results in a more pronounced decrease in the ability of microsome lipids to oxidize when inducing enzymatic mechanisms of LPO.

Enzymatic oxidation of microsome lipids undergoing a thermal procedure and in a hexametonium incubation medium with a molar concentration of 10-4 M; 10-5 M; 10-6 also changes the direction of oxidation- the lipids of liver microsomes in the presence of hexamethonium become less sensitive to LPO and this tendency increases with decreasing molar concentration of hexametonium in the incubation medium.

Thus, the thermal inactivation of the protein components of the membranes of the endoplasmic reticulum of hepatocytes (liver microsome’s) reduces the ability of the liver microsomes to oxidize in the presence of nicotine when inducing the enzymatic mechanisms of LPO to a greater extent than in microsomes not subjected to heat treatment.

Oxidation of microsomal lipids subjected to heat treatment in the presence of hexomethonium also changes the direction of oxidation of lipids- the lipids of the liver microsomes become less sensitive to oxidation by enzymatic mechanisms.