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SUMMARY**EFFECTS OF DIFFERENT DIETARY FATTY ACIDS SUPPLEMENTS UPON LIPID PEROXIDES PRODUCTION IN RAT TISSUES WITH DIFFERENT PHOSPHOLIPID COMPOSITION**

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Research was conducted upon experimentally induced hyperlipidemic animals by means of a custom tailored atherogenic diet. Cell susceptibility to nonenzyme induced oxidative stress appears to be influenced by membrane fatty acid composition. This study examines the degree of lipid peroxidation in rats with experimentally induced, moderate hyperlipidemia and evaluates lipid peroxidation products incubated in the presence and absence of peroxidation initiator. The idea was to determine whether differences in lipid peroxidation in steady-state and induced lipid peroxidation are a result of a different fatty acid supplementation and to compare the production of lipid peroxides in tissue with different phospholipids structure and different metabolic activity.

Adult *Wistar* strain rats of female gender were exposed to atherogenic diet for a period of 160 days, before randomization into 6 dietary groups with different intragastral oil supplementation. Lipid peroxidation products were measured in 2.5 % (w/v) of fresh liver homogenates (Tris-HCl, pH 7.4), by the assay of thiobarbituric acid reactive substances (TBARS) formation using the procedures described by Okhawa (1979), including modifications (1989) in three different experimental conditions: steady-state (which corresponds to concentration of lipid peroxides *in vivo*), spontaneous and metal stimulated lipid peroxidation.

Results were expressed as nmol TBARS per g of liver or heart tissue homogenate, calculated from the absorbency at 532 nm, using TEP as external standard. This study shows that a prolonged atherogenic dietary treatment causes moderate hypercholesterolemia and enhanced hypertriglyceridemia (+48.5% and +163.0, $p < 0.001$, respectively). Despite the lowering effects of the lipoprotein profiles, resulting from a fatty acid supplementation, the latter expressed oxidative susceptibility in the presence of metal-promoted lipid peroxidation measured in the end of each supplementation period.

When liver homogenates were exposed to Fe^{2+} and ascorbic acid – induced oxidative stress, lipid peroxidation (LPO) was enhanced in group treated with corn oil (ω -6) an fish oil (ω -6, + 60.7 %, $p < 0.001$ and ω -6, +

31.0 %, $p < 0.001$, respectively), but not in group receiving soybean oil. The achieved results supports the hypothesis that the process of lipid peroxidation is not always in correlation with the number of double bonds in fatty acids esterified in phospholipid molecules.

Discrepancies in analyzed references of the effects of unsaturated dietary fatty acids in phospholipids upon lipid peroxides production (LPO), was the reason to focus this research on the comparison of lipid peroxides production in tissues with different degree of unsaturation of their structural lipids and different "aerobic" metabolic activities. The achieved results revealed an enhancement in the production of lipid peroxides (TBARS formation) in the steady-state levels measured in liver tissue in comparison to heart muscle tissue. When homogenates were triggered to peroxidation initiator (Fe^{2+} -ascorbic acid induced oxidative stress), the heart tissue was much more sensitive to its effect. Consequently, it can be concluded that the content of LPO products is not the same in the different tissues. As a whole, it can be noted that the content of lipid peroxides is considerably higher in the organs and organelles characterized with intensive oxygen uptake, as well as by the high content of unsaturated lipids.