

EXPERIMENTAL ASPECTS OF NON-ALCOHOLIC FATTY LIVER DISEASE

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Relevance

In recent years, a significant increase in the prevalence of non-alcoholic fatty liver disease (NAFLD) has been observed among various population groups, including adolescents and young adults. This trend has made NAFLD a pressing public health problem. The liver plays a central role in regulating metabolic processes in the human body, and its dysfunction negatively affects multiple organ systems, including the cardiovascular and endocrine systems. Effective treatment of NAFLD largely depends on a clear understanding of its pathogenetic mechanisms. Experimental models are of particular importance in studying these mechanisms and in developing rational preventive and therapeutic strategies.

Objective

To develop an experimental model of non-alcoholic fatty liver disease using a hypercaloric diet enriched with animal fats and to evaluate stage-dependent changes in lipid metabolism.

Materials and Methods

The study was conducted on mature laboratory rats with an average body weight of 170.4 ± 2.4 g. The animals were maintained on a hypercaloric hepatogenic diet for 120 days. Lipid metabolism parameters were assessed on days 30, 90, and 120 of the experiment. Serum levels of total cholesterol, very-low-density lipoprotein cholesterol (VLDL-C), and high-density lipoprotein cholesterol (HDL-C) were measured. The obtained data were analyzed in comparison with those of the control group.

Results

On day 30 of the experiment, alimentary dyslipidemia was observed in rats, characterized by increased serum levels of total cholesterol and VLDL-C, along with an almost twofold decrease in HDL-C. After 90 days of dietary exposure, total cholesterol levels approached those of the control group, while VLDL-C levels decreased significantly. Prolonged exposure to the hypercaloric diet for 120 days led to the progression of hyperlipidemia, manifested by a marked increase in total cholesterol compared with control values. Persistently low VLDL-C levels observed in the second and third experimental stages were considered indicative of hepatic metabolic dysfunction and the development of steatohepatitis.

Conclusion

The developed experimental model adequately reflects the dynamics of lipid metabolism disturbances at different stages of non-alcoholic fatty liver disease. This model can be effectively used to study pathogenetic mechanisms of NAFLD and to evaluate pharmacological agents aimed at correcting metabolic disorders and improving liver function.

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