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RESEARCH ARTICLE

Changes in Biochemical Indicators During Alcoholic Intoxication

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Abstract: This study dynamically investigated the biochemical and physiological indicators of liver function and the overall metabolic state in rats under conditions of chronic ethanol intoxication. During the experiment, we assessed the activity of liver damage-indicating enzymes in the blood serum - ALT, AST, GGT, and alkaline phosphatase - as well as changes in the levels of albumin, total protein, glucose, triglycerides, and cholesterol. The results showed that chronic ethanol intoxication disrupts the structural-functional integrity of hepatocytes, leading to cytolysis, cholestasis, disorders of lipid and carbohydrate metabolism, and a decrease in the synthetic function of the liver. Furthermore, a decrease in body weight and an increase in the liver index in the experimental animals confirmed the escalating hepatotoxic effect of ethanol.

Key words: Ethanol, ALT, AST, GGT, AP, biochemical indicators.

INTRODUCTION

Chronic alcohol intoxication is one of the most pressing issues in modern medicine and experimental biology, causing profound structural and functional changes in the body, particularly in the liver. As the liver is the primary organ where ethanol metabolism occurs, it is the first to be exposed to its toxic effects. Ethanol and its metabolite, acetaldehyde, damage the membranes, mitochondria, and enzyme systems of hepatocytes, leading to the development of cytolysis, cholestasis, oxidative stress, impaired synthetic processes, and metabolic imbalance. Therefore, studying the dynamic changes in biochemical indicators that characterize liver function under conditions of long-term alcohol exposure is of great scientific and practical importance [1,2,3,4,5,6].

It is well-established that one of the most important laboratory indicators of liver damage is an increase in the serum activity of alanine aminotransferase (ALT) and aspartate aminotransferase (AST). Furthermore, an increase in the activity of excretory enzymes such as gamma-glutamyltransferase (GGT) and alkaline phosphatase (ALP) signifies cholestatic processes and damage to the hepatobiliary system. Chronic exposure to ethanol not only alters enzyme activity but also significantly affects protein, fat, and carbohydrate metabolism: the synthesis of albumin and total protein declines, glucose levels decrease, and triglyceride and cholesterol levels rise. Such changes indicate that the synthetic, metabolic, and detoxification functions of liver cells are impaired. From this perspective, a comprehensive assessment of the dynamic changes in biochemical and physiological parameters under conditions of chronic ethanol intoxication is crucial for a deeper understanding of the pathogenesis of alcohol-related liver damage, as well as for developing

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methods for its early diagnosis and correction [7,8,9,10]

METHODS

The study utilized 60 adult white outbred male rats weighing 180-220 g. In the first stage of the study, a model of chronic alcohol intoxication was induced by administering an ethanol solution into the stomachs of the rats for 7, 14, 21, and 28 days. The remaining rats formed the intact group (Group 1), and they were administered the same volume of distilled water into the stomach. On the 7th, 14th, 21st, and 28th days of the study, blood was collected from the animals, and biochemical parameters were determined.

RESULTS AND DISCUSSION

In chronic alcohol intoxication, the activity of several biochemical indicators of protein, fat, and carbohydrate metabolism - AlT, AsT, AP, and GGT - was studied. Pathological processes indicating liver damage are cytolysis, cholestasis, toxic damage to hepatocytes, insufficiency of synthetic processes in hepatocytes, decreased inactivation of toxic compounds, and inflammatory syndromes. The pathophysiological basis of cytolysis syndrome is the disruption of the integrity of the hepatocyte plasma membrane and its organelles, as well as the development of hyperfermentemia.

The results of our studies showed that exposure to ethanol disrupts the integrity of the hepatocyte membrane, leading to an increase in liver enzymes in the blood serum. It is well-known that ALT and AST activity are crucial indicators in the diagnosis of liver diseases. By day 28, transaminase activity had significantly increased. ALT enzyme activity increased 1.87-fold compared to the levels in intact rats, while the increase in AST activity was 1.73-fold. In this group of rats, we can see that GGT enzyme activity increased 4.7-fold. It was determined that the activity of alkaline phosphatase increased to a lesser extent than other enzymes, by 1.20-fold.

Transaminase activity began to change as early as the first 7 days of the study. Specifically, on days 7, 14, 21, and 28 of the study, ALT activity increased by 2.2%, 26.9%, 53.8%, and 86.6%, respectively, compared to the levels in the intact group. Meanwhile, AST enzyme activity was observed to increase by 4.0% on day 7, and in the subsequent periods on days 14, 21, and 28, it increased by 19.8%, 54.8%, and 73.5%, respectively (Figures 1, 2). 53.8% and 86.6%, and the activity of the AsAT enzyme increased by 4.0% on the 7th day, and in subsequent periods by 19.8% on the 14th, 21st, and 28th days;

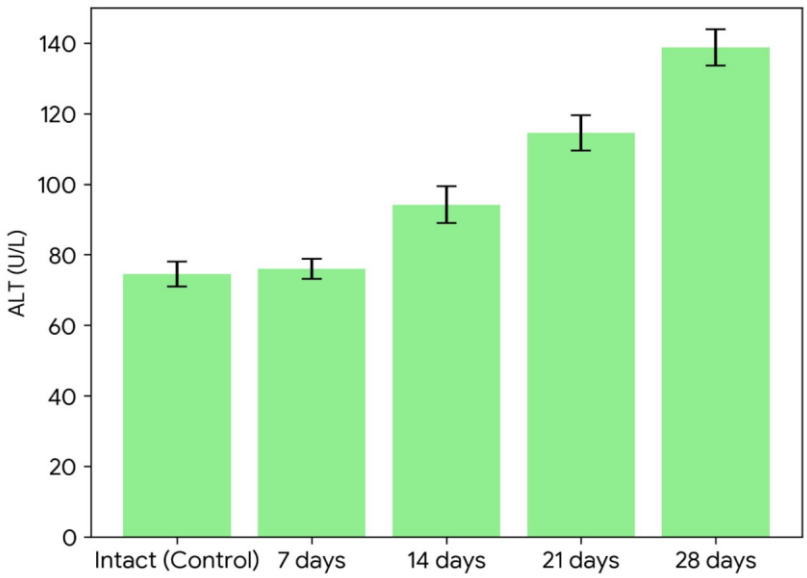

Figure 1. Dynamics of ALT activity in blood serum under conditions of alcohol poisoning.

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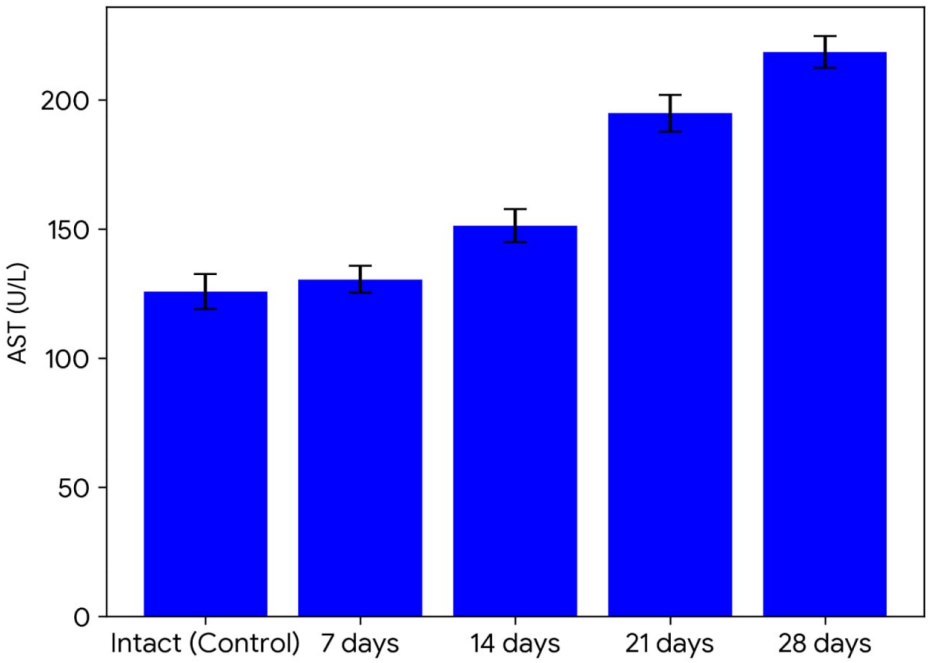

Figure 2. Dynamics of AST activity in blood serum under conditions of alcohol poisoning.

The GGT level also progressively increased over time: while it decreased by 11.1% at the 7-day mark, it increased by 100.0%, 254.4%, and 366.7% on days 14, 21, and 28, respectively, compared to the intact group. AP activity showed an increase from the very beginning of the study: on days 7, 14, 21, and 28, it increased by 18.98%, 13.02%, 31.9%, and 47.71%, respectively (Figures 3 and 4). The activity of IF manifested itself in an increase already in the initial period of the study: 18.98% on days 7, 14, 21, and 28; 13.02%; 31.9% and 47.71% (3;

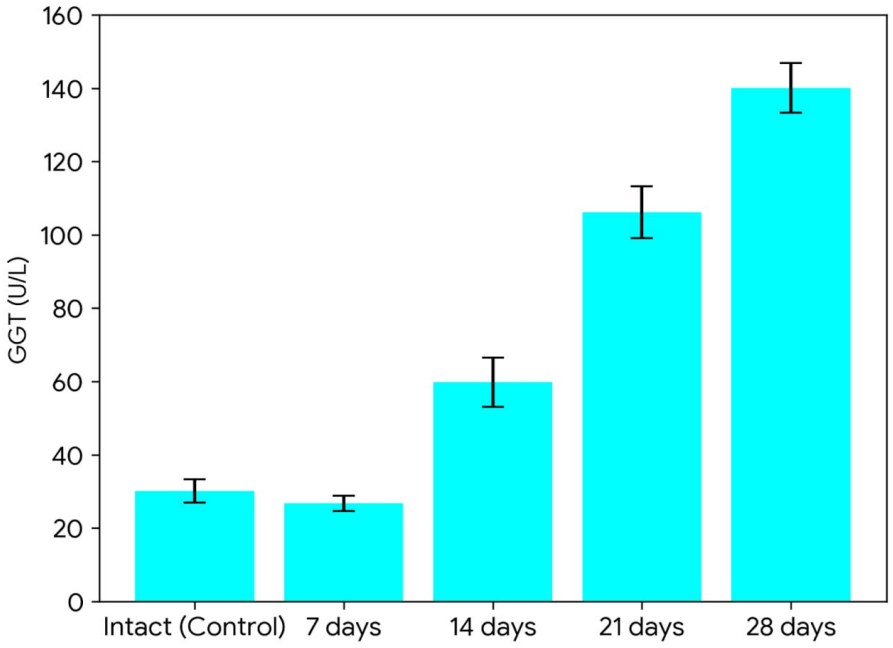

Figure 3. GGT activity in blood serum over time under conditions of alcohol intoxication.

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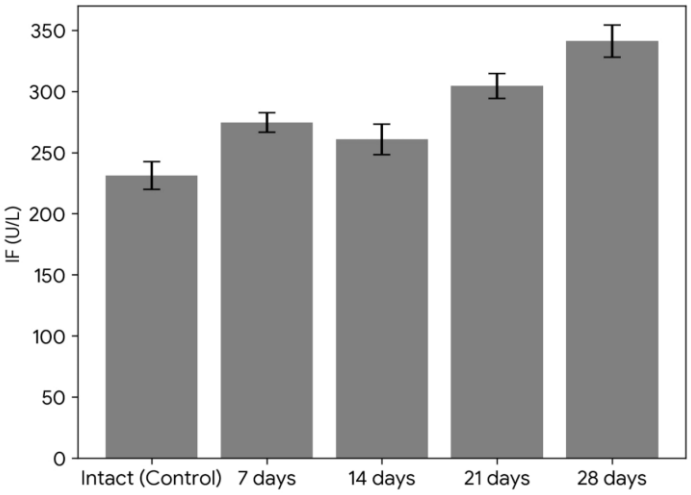

Figure 4. AP activity in blood serum over time under conditions of alcohol intoxication.

The albumin level consistently decreased throughout all experimental periods. Specifically, while the albumin level decreased by 6.8% in the 7-day group, a 10.8% decrease was observed in the 14-day period. At the 21-day mark, this decrease became more pronounced, reaching 24.1%, and in the 28-day period, the albumin level was found to have decreased by 30.4%, indicating a significant impairment of the liver's synthetic function (Figure 5).

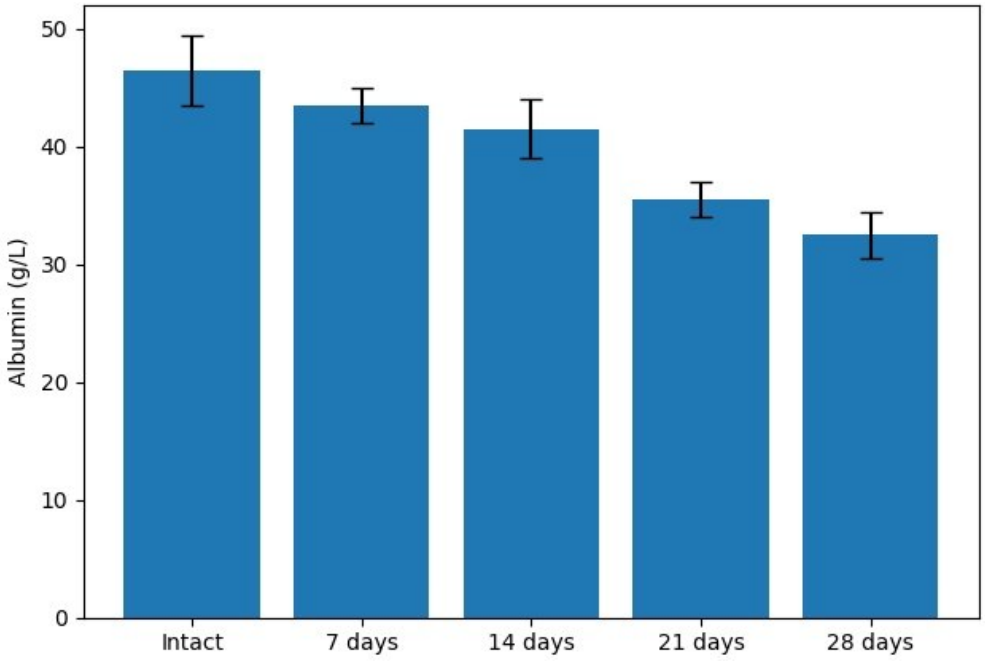

Figure 5. Albumin level in blood serum over time under conditions of alcohol intoxication.

According to the study results, the triglyceride (TAG) level in the experimental groups consistently increased compared to the intact group. Specifically, the TAG level increased by 19.4% in the 7-day observation period and reached 52.8% in the 14-day period. In the subsequent stages, a more pronounced increase was observed, with a rise of 80.6% at 21 days and 102.8% at the 28-day mark, indicating the progressive nature of the lipid metabolism disorder (Figure 6).

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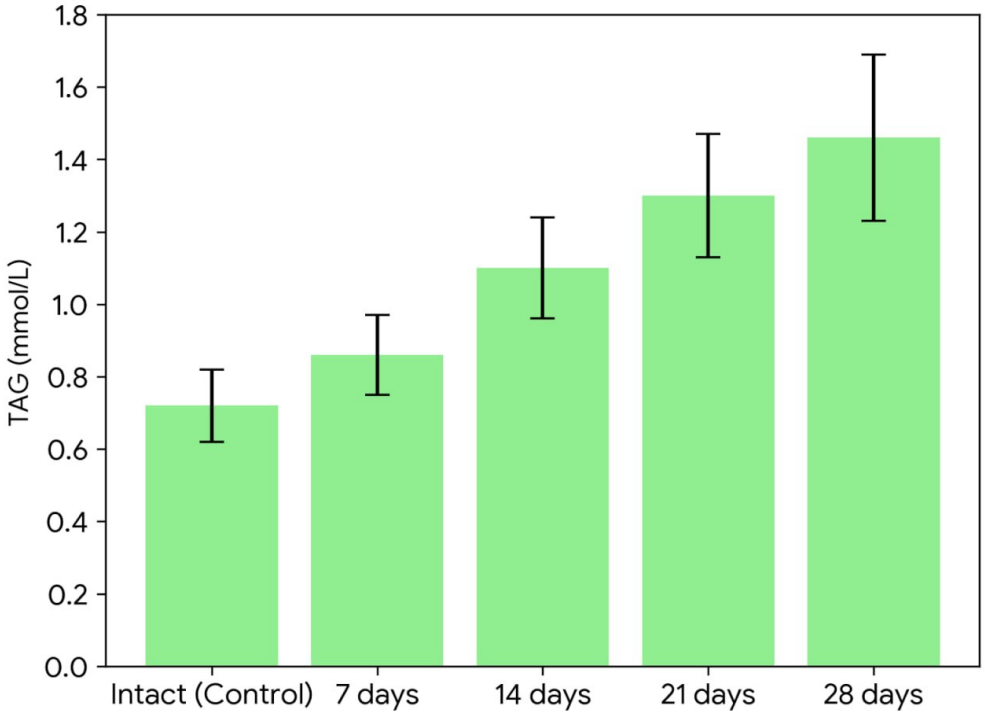

Figure 6. Triglyceride levels in blood serum over time under conditions of alcohol intoxication.

Cholesterol levels also demonstrated a similar trend. Specifically, its level increased by 34.0% in the 7-day period, and an increase of 41.3% was recorded at 14 days. By the 21-day mark, the cholesterol level peaked, showing a 68.1% increase. Although a slight decrease was observed in the 28-day period, it remained 65.1% higher compared to the intact group. These changes indicate a stable and deepening disruption of lipid metabolism (Figure 7).

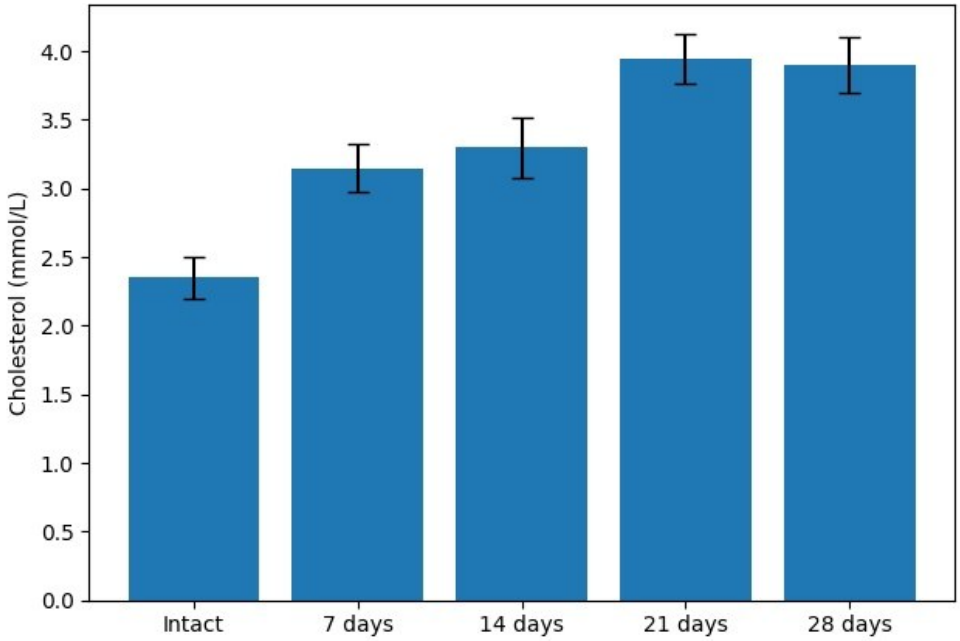

Figure 7. Cholesterol levels in blood serum over time under conditions of alcohol intoxication.

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Lipid metabolism indicators were also characterized by a consistent increase: TAG increased by 19.44%, 52.78%, 80.56%, and 102.78% on days 7, 14, 21, and 28, respectively, compared to the intact group, while cholesterol increased by 34.04%, 41.28%, 68.09%, and 65.11%. A downward trend was observed in indicators reflecting protein synthesis and overall trophic status: total protein decreased by 2.47%, 6.44%, 11.34%, and 15.49% on days 7, 14, 21, and 28, while albumin decreased by 6.84%, 10.76%, 24.05%, and 30.44%, respectively. Regarding energy metabolism, glucose levels decreased by 11.98%, 18.15%, 30.13%, and 33.76% on days 7, 14, 21, and 28 compared to the intact group. Meanwhile, urea, an indicator of nitrogen metabolism, decreased by 6.23%, 11.21%, 14.33%, and 17.29% during the same periods. 68.09% and 65.11% respectively. A tendency towards a decrease in indicators reflecting protein synthesis and the overall trophic state was observed: total protein on days 7, 14, 21, and 28 - 2.47%; 6.44%; 11.34% and 15.49%, albumin 6.84%; 10.76%; 24.05% and 30.44% respectively. From the energy metabolism side, the glucose content was 11.98% on days 7, 14, 21, and 28 compared to the intact group; 18.15%; 30.13% and 33.76%, and urea, which is an indicator of nitrogen metabolism, at the same time decreased by 6.23%; 11.21%;

Numerous studies have established that chronic ethanol exposure increases the activity of enzymes in the blood. Ethanol metabolism results in the formation of reactive oxygen species, which damage mitochondria and cell membranes. This leads to the release of transaminases into the bloodstream and an increase in their activity. A change in AST is a clear sign of severe liver damage, reflecting mitochondrial injury. An increase in ALT is more reliable, as its half-life is longer than that of AST.

The enlargement of hepatocytes leads to the obstruction of bile ducts and the development of intracellular cholestasis syndrome. This condition is accompanied by an increase in the level of excretory enzymes in the blood. The enzyme alkaline phosphatase is found in several tissues, and an increase in its blood level indicates impaired excretory function in the liver. The obtained results are consistent with literature data, which indicate a significant increase in the GGT enzyme and a relative increase in AP in cases of toxic liver damage.

Thus, in cases of ethanol poisoning, the activity levels of the liver's excretory and indicator enzymes indicate damage to both the outer membrane of hepatocytes and to mitochondrial and cytoplasmic components. Furthermore, the transient necrosis of parts of liver cells explains the significant increase in GGT enzyme activity. The dynamics of the changes in the activity of the enzymes studied in our research indirectly indicate a disruption in the structural integrity of the hepatocyte membrane.

Chronic ethanol poisoning leads to changes in protein, carbohydrate, and fat metabolism. In our studies, this was manifested by the development of hypoglycemia resulting from the inhibition of gluconeogenesis, hypercholesterolemia resulting from the inhibition of transport proteins in the liver, and hypoalbuminemia resulting from the impairment of the liver's synthetic function.

One of the consequences of ethanol-induced liver damage is the syndrome of synthetic process deficiency in the liver, which manifests as a decrease in proteins synthesized by hepatocytes. A classic indicator of this syndrome is a reduction in blood albumin levels. The results from studying protein metabolism indicators show a significant disruption of protein metabolism in animals administered ethanol. In animals with chronic ethanol intoxication, the amount of albumin decreased. However, the decrease in total protein was not as pronounced, due to an increase in blood globulins. In our opinion, these results indicate an impairment of the liver's synthetic function, resulting from damage by free radicals.

To draw conclusions about pathological changes in fat metabolism during ethanol intoxication, it is important to study the changes in serum cholesterol levels. In the untreated group of rats, an increase in cholesterol levels was detected compared to healthy rats. The reason for such changes in lipid metabolism is the accumulation of lipid peroxidation products

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in lipoprotein units, a disruption of the cholesterol esterification process, and consequently, an impairment in both the distribution of total cholesterol within lipoprotein units and the excretion of cholesterol from the body.

The liver is the organ that carries out cholesterol synthesis and lipoprotein metabolism. Therefore, alcohol consumption is one of the main factors that causes dyslipidemia. A significant increase in total blood cholesterol levels due to alcohol consumption has been identified. In this process, the activation of the HMG-CoA reductase enzyme leads to hypercholesterolemia. As is known, cholesterol is the main component of the biological membrane of all cells. Changes in cholesterol levels in chronic alcoholism can cause chronic liver diseases. The reliability of the results obtained in our research is supported by the above-mentioned findings.

Thus, in chronic alcohol intoxication, impairments were observed in the processes of cytolysis (increased transaminase activity, impaired pigment metabolism), cholestasis (increased activity of alkaline phosphatase and GGT, increased cholesterol levels), and the synthetic function of the liver (decreased blood glucose and albumin levels). The results obtained indicate a disruption of the structural and functional capacities of the liver under the influence of ethanol.

Table 1

The effect of herbal preparations on changes in body weight and liver index in a model of chronic ethanol poisoning

Groups	Initial weight	Final weight	Difference in weight	Liver weight	Liver index (%)
Intact	178.67±7.26	217.33±7.59	38.67±0.861	5.895±0.33	2.70±0.07
7 days	227.50±11.76	203.50±8.95	-24±3.21	6.13±0.18	3.03±0.12
14 days	202.17±10.66	180.17±8.22	-23.3±8.68	5.688±0.15	3.17±0.09
21 days	235.33±10.15	194.50±14.42	-40.8±5.80	6.49±0.32	3.41±0.25
28 days	225.33±9.5	189.3±12.7	-55.03±3.2	8.5±0.42	4.41±0.25

In conditions of chronic ethanol intoxication, a dynamic decrease in the body weight of animals is observed, along with a concurrent increase in the liver index (Table 1). Specifically, the intact group exhibited positive dynamics in body weight during the experiment, increasing from an initial 178.67±7.26 g to a final 217.33±7.59 g, with a total weight gain of 38.67±0.86 g. Conversely, a decrease in body weight was recorded in the groups receiving ethanol: on day 7, it fell from an initial 227.50±11.76 g to a final 203.50±8.95 g, a decrease of 24.0±3.21 g; on day 14, it decreased from 202.17±10.66 g to 180.17±8.22 g, for a loss of 23.3±8.68 g; and on day 21, it dropped from 235.33±10.15 g to 194.50±14.42 g, amounting to a loss of 40.8±5.80 g. The most significant weight loss was observed on day 28, with a decrease from 225.33±9.5 g to 189.3±12.7 g, resulting in a difference of 55.03±3.2 g. This suggests that long-term ethanol consumption may be associated with decreased appetite, intensified catabolic processes, and a general metabolic imbalance. Simultaneously, the increase in liver mass and liver index confirms the hepatotoxic effect of ethanol. While the liver weight in the intact group was 5.895±0.33 g and the liver index was 2.70±0.07%, in the ethanol-treated

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groups, the liver weight and index were found to increase as follows: to 6.13±0.18 g and 3.03±0.12% by day 7; to 5.688±0.15 g and 3.17±0.09% by day 14; to 6.49±0.32 g and 3.41±0.25% by day 21; and to 8.5±0.42 g and 4.41±0.25% by day 28, respectively. Therefore, chronic ethanol intoxication is accompanied by liver enlargement (hepatomegaly) and an elevated "liver index," which may be intrinsically linked to pathological changes in the liver, such as edema, inflammatory processes, and fatty infiltration/steatosis.

In rats with chronic alcohol intoxication, a syndrome of toxic damage to hepatocytes is observed, resulting from impaired mitochondrial function. Ethanol and its metabolites, particularly acetaldehyde, which is formed directly in the mitochondria, exert a toxic effect. Consequently, ATP synthesis is disrupted, which pathologically affects the function of endobiotic detoxification. Along with the neutralization of ammonia produced in the body and the inactivation of hormones, the processes of xenobiotic hydroxylation and detoxification in the microsomal apparatus of hepatocytes are inhibited and disrupted [11].

The results of this study confirmed the development of structural and functional liver impairments in rats under chronic ethanol intoxication, and that these processes are accompanied by consistent changes in protein, carbohydrate, and lipid metabolism indicators. During dynamic observation, cytolysis syndrome developed against the background of damage to liver cell membranes and the mitochondrial apparatus, leading to increased transaminase activity. By day 28, a significant rise in AlAT and AsAT levels indicated toxic damage to hepatocytes and the development of hyperfermentemia. An increase in the activity of GGT and alkaline phosphatase, reflecting signs of cholestasis, as well as a progressive increase in bilirubin levels, pointed to impaired excretory function and intensified intracellular cholestasis in the liver. Analysis of metabolic parameters confirmed that ethanol intoxication disrupts carbohydrate metabolism, resulting in hypoglycemia, while also causing hypertriglyceridemia and hypercholesterolemia in lipid metabolism; this is attributed to the mechanisms of hepatic steatosis and lipotoxicity. Impairment of the liver's synthetic function was manifested by a decrease in total protein and especially albumin levels, indicating reduced protein synthesis in hepatocytes and limited detoxification capabilities. Overall, the findings demonstrated that chronic ethanol intoxication exacerbates cytolysis and cholestasis in the liver, causes metabolic dysfunction and synthetic insufficiency, and that these changes progressively worsen throughout the course of the experiment.

CONCLUSION

In conclusion, chronic ethanol poisoning in rats leads to profound structural and functional liver damage. This condition manifested as increased activity of transaminases, GGT, and alkaline phosphatase; decreased levels of albumin and total protein; a reduction in glucose; and elevated levels of triglycerides and cholesterol. These changes indicate a progressive intensification of cytolysis, cholestasis, synthetic insufficiency, and metabolic imbalance against the backdrop of ethanol intoxication. Thus, the obtained results confirm that damage to hepatocyte membranes and mitochondria, disorders of lipid metabolism, and a decline in detoxification capabilities play a leading role in the pathogenesis of liver injury in chronic alcohol intoxication.

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