EFFECT OF STRONGYLOIDIASIS (*STRONGYLOIDES PAPILLOSUS*) ON BLOOD ENZYME ACTIVITY IN RABBITS

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Parasitic infections continue to represent a significant constraint in rabbit production systems worldwide [1]. Among them, strongyloidiasis caused by *Strongyloides papillosus* is widely distributed and particularly relevant in intensive farming conditions [2, 3]. The parasite affects the intestinal mucosa, leading to secondary metabolic disturbances [4].

Recent studies emphasize that helminth infections are not limited to local intestinal damage but can induce systemic effects, including oxidative stress, immune dysregulation, and hepatic dysfunction [1, 5, 6]. Enzymatic blood markers such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and cholinesterase are considered sensitive indicators of liver integrity and metabolic status [7, 8].

Despite growing interest, data on biochemical alterations in rabbits with naturally acquired strongyloidiasis remain limited. Therefore, the present study aims to expand knowledge on pathophysiological mechanisms underlying enzyme activity changes in relation to infection intensity.

The study was conducted during 2015–2022. The experimental part was carried out at rabbit farms with a cage housing system. The level of infection of rabbits with *Strongyloides papillosus* was determined using coprological examination by the McMaster egg-counting technique (McMaster slide method). Animals were naturally infected with Strongyloides papillosus and allocated into two experimental groups based on fecal egg counts (EPG) [9]. Group I showed moderate infection (459.26±46.91 EPG), while group II exhibited high infection intensity (2370.37±311.45 EPG). A control group consisted of non-infected animals. Biochemical analysis of blood serum was performed using commercial reagent kits produced by LLC RPC “Filisit-Diagnostics” (Dnipro, Ukraine). Enzyme activity was determined spectrophotometrically: alanine aminotransferase (ALT) and aspartate aminotransferase (AST) by the Reitman–Frankel method, α-amylase by the Karaway method, cholinesterase using acetylcholine chloride as a substrate, and gamma-glutamyl transferase (GGT) using γ-L-(+)-glutamyl-4-nitroanilide as a substrate.

Significant alterations in enzyme activity were observed in infected animals, with a clear dependence on invasion intensity. In rabbits of group II, alanine aminotransferase (ALT) activity increased by 36.37%, aspartate aminotransferase (AST) by 67.78%, and gamma-glutamyl transferase (GGT) by 41.48% ($p<0.001$) compared to control animals. In group I, similar but less pronounced changes were detected. No significant changes were found in α-amylase activity.

Cholinesterase activity decreased significantly in infected rabbits, by 1.32 times in group I ($p<0.01$) and 1.64 times in group II ($p<0.001$), indicating impaired liver synthetic function.

The observed elevation of ALT and AST reflects hepatocellular damage and increased membrane permeability. These enzymes are released into the bloodstream following structural and functional disruption of hepatocytes [4, 6]. The stronger increase in AST compared to ALT may indicate broader tissue involvement, including muscular and systemic effects [7].

The significant rise in GGT activity suggests involvement of the hepatobiliary system and potential cholestatic processes. Helminth infections are known to induce inflammatory responses in hepatic tissue, leading to bile flow disturbances and enzyme induction [5, 10].

A key finding is the reduction in cholinesterase activity, which serves as a sensitive marker of hepatic synthetic capacity. Decreased cholinesterase reflects impaired protein synthesis and is commonly associated with chronic intoxication and liver dysfunction [4, 7].

From a pathophysiological perspective, *Strongyloides papillosus* contributes to systemic metabolic imbalance through multiple mechanisms: nutrient malabsorption, inflammatory mediator release, oxidative stress induction, and direct toxic effects of parasite metabolites [3, 8]. Recent literature supports the concept that helminth infections trigger host oxidative stress pathways and disrupt mitochondrial function in hepatocytes [5, 10].

These findings are consistent with modern parasitology research emphasizing the systemic nature of helminth infections and their impact beyond the gastrointestinal tract. Moreover, the data support the use of biochemical markers as reliable indicators for disease monitoring and severity assessment in veterinary practice [6, 7].

Strongyloidiasis caused by *Strongyloides papillosus* induces significant alterations in blood enzyme activity in rabbits. The severity of biochemical disturbances correlates with infection intensity. ALT, AST, GGT, and cholinesterase may serve as informative biomarkers for assessing liver damage and systemic intoxication.

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BOVINE ADENOMYOSIS AS A ONE HEALTH MODEL: NEW HISTOPATHOLOGICAL CLASSIFICATION AND STAGE-DEPENDENT MOLECULAR ALTERATIONS

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Introduction and Aim: Adenomyosis is a uterine disorder characterized by the presence of endometrial glands and stromal cells within the uterine musculature (myometrium), which may contribute to impaired reproductive performance in cattle. Reduced fertility represents a major challenge in dairy production systems, with direct implications for animal health, economic sustainability, and resource efficiency. Importantly, adenomyosis shares key pathophysiological