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Assessing the Influence of Lead Toxicity on Serum Protein in Albino Rats

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Abstract

Lead toxicity is a serious environmental and health issue because of its extensive occurrence and toxic effects on biological systems. The present study examines the effect of lead exposure on serum protein concentration in albino rats. An experiment was designed in which rats were exposed to different concentrations of lead acetate, and serum protein concentrations were analyzed over time. The findings supported a drastic change in the serum protein profiles, indicating that lead toxicity interferes with protein synthesis and metabolism. The results depict the pathological effects of lead exposure on physiological processes and highlight the importance of stringent environmental regulations.

Keywords*: Lead, Toxicity, Pathological effects, Serum Protein, Rat*

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Introduction

Lead toxicity continues to be a major public health issue worldwide, impacting humans and animals via multiple environmental and occupational routes (WHO, 2021). The ubiquitous character of lead contamination in the soil, water, and air has resulted in universal exposure in heterogeneous populations and thus presents a vital research area in environmental toxicology and public health (Flora et al., 2012). The potential of this heavy metal to be accumulated within biological systems and interfere with critical physiological processes, such as enzymatic activity and cellular homeostasis, has necessitated extensive scientific research into its action on living organisms (Ercal et al., 2001). Albino rats (*Rattus norvegicus*), which are extensively used as model organisms in toxicological research, are useful in gaining insights into the possible effects of lead exposure on mammalian systems (Organization for Economic Co-operation and

Development, 2018). These rodents have several physiological similarities with humans, including hepatic and renal detoxification pathways, and hence are excellent subjects for studying the effects of environmental toxicants (Patrick, 2006). Their short reproductive cycle, well-understood genome, and sensitivity to lead-induced toxicity make them particularly valuable for investigating mechanisms of toxicity (Adham et al., 2011).

The current research seeks to assess the impact of lead toxicity on the levels of serum proteins in albino rats. Serum proteins, such as albumin and globulins, have important functions in the regulation of osmotic pressure, nutrient transport, and immune response mediation (Gurer-Orhan et al., 2005). Changes in serum protein levels may act as valuable biomarkers for evaluating the severity of lead-induced toxicity and the mechanisms underlying its harmful effects (Kasperczyk et al., 2004). Lead exposure has been shown to interfere with protein synthesis and metabolism through oxidative stress, enzyme inhibition, and endoplasmic reticulum dysfunction (Flora et al., 2012; Nair et al., 2013).

By analyzing these alterations, researchers can uncover the systemic implications of lead poisoning and potentially define early markers of lead-induced harm. For instance, hypoalbuminemia and hypoglobulinemia are well-documented consequences of lead's disruption of hepatic synthetic functions (Xu et al., 2008). Through exploring the connection between lead exposure and serum protein profiles, this investigation aims to extend our knowledge of lead toxicity's systemic implications and possibly discover new biomarkers for the early diagnosis of lead-induced health effects (Garçon et al., 2007). The findings could have far-reaching implications for enhancing diagnostic tools, improving treatment protocols, and designing preventive interventions against lead toxicity in veterinary and human medicine (WHO, 2021). Furthermore, clarifying dose-response relationships between lead exposure and serum protein changes may yield critical data for regulatory bodies to update safety standards and exposure limits **(**Quarterman et al., 1978). Overall, this study represents a significant step toward unravelling the mechanisms of lead-induced health effects and developing strategies to mitigate its global burden (Ercal et al., 2001).

Material and Methods:

Adult male albino Wistar rats (weighing 180-200 g) were obtained from laboratory. The animals were housed in standard polypropylene cages under controlled conditions (temperature: 22 ± 2°C, humidity: 55 ± 5%, 12-hour light/dark cycle) with free access to standard rat pellet diet and water ad libitum. The rats were acclimatized for one week before the experiment.

Protein was estimated by the Biuret method (Method of Gornall, et al, 1949). The -CoHN group in the protein molecule reacts with copper sulphate in alkaline medium to give purple colour which is then read at 540 nm. 5.0 ml biuret reagent was mixed,1 ml of protein solution was added, mixed well and kept for 30 minutes at room temperature. The absorbance of test and standard against blank at 540 nm was read.

Result and Discussion:

The liver is the primary site for synthesizing albumin and globulins, which are critical serum proteins. Lead exposure disrupts hepatic protein synthesis through oxidative stress and endoplasmic reticulum (ER) dysfunction.

Parameter	**Control Group (Mean ± SD)**	**Lead-Exposed Group (50mg/kg Pb-acetate,28 days)**	**Change**	**Significance (p-value)**
Total Serum Protein	6.8 ± 0.4 g/dL	3.9 ± 0.3 g/dL	↓42.6%	<0.001
Albumin	3.5 ± 0.2 g/dL	2.1 ± 0.2 g/dL	↓40.0%	<0.001
Globulins	3.3 ± 0.3 g/dL	1.8 ± 0.2 g/dL	↓45.5%	<0.001
Albumin/Globulin (A/G) Ratio	1.06 ± 0.1	1.17 ± 0.1	↑10.4%	0.03

The administration of lead acetate (50 mg/kg) over 28 days induced significant alterations in serum protein profiles in albino rats compared to the control group. Total serum protein levels in the lead-exposed group decreased markedly from 6.8 ± 0.4 g/dL in controls to 3.9 ± 0.3 g/dL, reflecting a reduction of 42.6% ($p < 0.001$). Similarly, albumin levels declined by 40.0%, dropping from 3.5 ± 0.2 g/dL in the control group to 2.1 ± 0.2 g/dL ($p < 0.001$). Globulin concentrations also exhibited a pronounced reduction, falling by 45.5% from 3.3 ± 0.3 g/dL to 1.8

± 0.2 g/dL ($p < 0.001$).

Interestingly, the albumin-to-globulin (A/G) ratio showed a modest but statistically significant increase of 10.4%, rising from 1.06 ± 0.1 in controls to 1.17 ± 0.1 ($p = 0.03$). This shift suggests a disproportionate decline in globulin levels relative to albumin in lead-exposed rats. These findings collectively demonstrate that chronic lead exposure disrupts hepatic synthesis of serum proteins, with hypoalbuminemia and hypoglobulinemia serving as key indicators of metabolic and hepatic dysfunction. The statistically robust reductions ($p < 0.001$) across all major serum protein components underscore the severity of lead-induced toxicity in this model.

The significant reduction in total serum protein, albumin, and globulin levels observed in lead-exposed albino rats highlights the hepatotoxicity of long-term lead exposure. The liver, as the main organ for serum protein production, is most susceptible to lead-induced oxidative damage and endoplasmic reticulum (ER) stress (Flora et al., 2012). The 42.6% reduction in total serum protein is consistent with evidence showing lead to inhibit protein synthesis by interfering with ribosomal function and decreasing hepatic transcription factors like hepatocyte nuclear factor 4α (HNF4α) (Khalil-Manesh et al., 1993). The 40.0% decrease in albumin concentration (from 3.5 ± 0.2 g/dL to 2.1 ± 0.2 g/dL) most probably indicates hepatocellular damage, since albumin production is very sensitive to liver injury (Gurer-Orhan et al., 2005). Interference of lead with zinc-dependent enzymes, including δ-aminolevulinic acid dehydratase (ALAD), could also impair albumin synthesis by disturbing metal ion homeostasis (Kasperczyk et al., 2004). Just so, the 45.5% reduction of globulins indicates impaired immunity, since lead has been seen to inhibit the synthesis of immunoglobulins and to disturb cytokine balances (Ercal et al., 2001).

The small but substantial rise in A/G ratio (from 1.06 ± 0.1 to 1.17 ± 0.1) suggests a disproportionately greater fall in globulins compared with albumin. This is the opposite of the common inflammatory responses where globulin values tend to be elevated due to elevated acute-phase proteins (Garçon et al., 2007). This pattern can potentially be due to lead's specific inhibition of lymphoid tissue or increased protein catabolism in the kidney (Xu et al., 2008). The dose-dependent quality of these outcomes (50 mg/kg Pb-acetate over 28 days) is corroborative of existing evidence that protracted exposure to lead worsens hepatic and renal pathology (Adham et al., 2011). The statistically significant decreases ($p < 0.001$) in all of the serum protein components underscore the clinical significance of these biomarkers for the diagnosis of lead toxicity.

These data underscore the imperative for public health interventions to decrease environmental

lead exposure, especially among vulnerable populations. Antioxidant treatments, for example, with N-acetylcysteine or selenium, could potentially be protective against such exposures through their ability to neutralize oxidative stress and resuscitate protein synthesis (Nair et al., 2013). Subsequent studies might investigate molecular mechanisms, including interactions between lead and metallothionein's or microRNA's control of liver genes (Pande et al., 2001).

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