Knowledgeable Research *ISSN 2583-6633* Vol.02, No.03, October, 2023
An International Peer-Reviewed Multidisciplinary Journal

Knowledgeable Research
ISSN 2583-6633
Vol.02, No.03, October,2023
http://knowledgeableresearch.com/

Serum Cholesterol Alterations in Albino Rats Induced by Lead Toxicity

Neetu Sharma
Department of Zoology
Maharaja Agrasen Mahavidyalaya, Bareilly

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Abstract:

Lead toxicity is a serious global health issue, with the potential to affect multiple physiological processes, including lipid metabolism. This research examined the dose-dependent effects of lead acetate exposure on serum cholesterol in albino rats over 28 days. Rats were allocated to control and three treatment groups receiving 10, 30, or 50 mg/kg/day of lead acetate. Serum total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglyceride concentrations were assessed. Lead exposure resulted in significant dose-dependent increases in total cholesterol, LDL cholesterol, and triglycerides, with a reduction in HDL cholesterol. The LDL/HDL ratio, an indicator of cardiovascular risk, rose exponentially in the high-dose group. These results indicate that lead toxicity disrupts lipid metabolism, inducing dyslipidemia with increased atherogenic lipids and decreased HDL. The research points to the intricate interaction between environmental toxins and metabolic pathways, underscoring the importance of effective measures to reduce lead exposure and its related health risks. Additional research is needed to clarify the mechanisms of lead-induced lipid dysregulation and its possible implications for human health.

Keywords: *Lead toxicity, Lipid metabolism, Cholesterol levels, Albino rats, Dose-dependent*

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Introduction

Lead is a ubiquitous environmental pollutant with well-documented toxic effects on various organ systems. Despite global efforts to reduce lead exposure, it remains a significant public health concern, particularly in developing countries. The pervasive nature of lead contamination stems from its historical use in various industrial applications, including gasoline additives, paints, and plumbing materials. Although many developed nations have implemented stringent regulations to limit lead use, its persistence in the environment and continued use in some industries contribute to ongoing exposure risk. The impact of lead toxicity on lipid metabolism, specifically cholesterol levels, has garnered considerable attention in recent years. Cholesterol, a crucial component of cell membranes and a precursor of various hormones, plays a vital role in maintaining

Dr.Neetu Sharma
Received Date: 12.10.2023
Publication Date: 30.10.2023

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cellular homeostasis. This sterol molecule is essential for numerous physiological processes, including cell signaling, vitamin D synthesis and bile acid production. Alterations in cholesterol levels can have far-reaching consequences on overall health and physiological functions, potentially affecting cardiovascular health, endocrine function and neurological processes.

Previous studies have suggested that lead exposure may disrupt lipid metabolism, potentially leading to changes in serum cholesterol levels. The mechanisms underlying this disruption are complex and not fully understood but may involve alterations in cholesterol biosynthesis, transport, and catabolism. Lead-induced oxidative stress, a well-established consequence of lead toxicity, may also contribute to lipid peroxidation and subsequent changes in the cholesterol homeostasis. Furthermore, Pb's ability to interfere with calcium-dependent processes and enzyme functions may indirectly affect lipid metabolism pathways. Albino rats have long been used as model organisms in toxicological studies because of their physiological similarities to humans and well-characterized responses to various toxicants. These animals provide a valuable platform for investigating the effects of lead exposure on cholesterol metabolism in controlled laboratory conditions. The use of albino rats allows for precise control of exposure levels, duration, and environmental factors, enabling researchers to isolate the specific effects of lead on the cholesterol metabolism. Additionally, the relatively short lifespan and rapid reproductive cycle of rats facilitate the study of both acute and chronic exposure scenarios and potential transgenerational effects.

This study aimed to elucidate the relationship between lead toxicity and serum cholesterol levels in albino rats. By examining the effects of different lead concentrations and exposure durations on cholesterol levels, we aimed to provide insights into the mechanisms underlying lead-induced lipid dysregulation. This study will employ a multifaceted approach, combining biochemical analyses, histopathological examinations, and molecular techniques to comprehensively assess the impact of lead on cholesterol metabolism. This integrated methodology will allow for a more nuanced understanding of the dose- and time-dependent effects of lead exposure on lipid profiles. Understanding these processes may contribute to the development of more effective strategies for mitigating the health impacts of lead exposure in both animal and human populations. The findings of this study could have significant implications for public health policies, environmental regulations, and the clinical management of lead-exposed individuals. Moreover, elucidating the mechanisms underlying lead-induced cholesterol alterations may provide valuable insights into the broader field of environmental toxicology and the complex interactions between environmental pollutants and metabolic processes. In conclusion, this study aimed to bridge the gap in our understanding of lead toxicity and its effects on cholesterol metabolism, utilizing the albino rat model to generate translatable insights for human health. By unraveling the intricate relationship between lead exposure and serum cholesterol alterations, this study aims

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to contribute to the broader goal of mitigating the global health burden associated with environmental lead contamination.

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.

Cholesterol, in presence of acetic acid and sulphuric acid reacts with FeCl3 and produces a red coloured complex. 1 ml of serum was taken in a clean test-tube followed by addition of 5 ml FeCl3 reagent. The mixture was boiled in a boiling water- bath for 5- 35 minutes, cooled at room temperature and 3 ml conc. H2SO4 was added to it and kept at room temperature for 30 minutes. OD.0. at 555 rpm was read (Chiaman and Henry, 1959).

Results and Discussion:

The administration of lead acetate over 28 days resulted in significant dose-dependent alterations in serum cholesterol levels in albino rats. The following numeric data summarize the findings:

Table 1. Total Serum Cholesterol

Group	**Dose (mg/kg/day)**	**Total Cholesterol (mg/dL)**	**Change vs. Control**	**p-value**
Control	0	68.5 ± 3.2	-	-
Low-dose Lead	10	82.3 ± 4.1	↑20.1%	<0.05
Medium-dose Lead	30	97.6 ± 5.3	↑42.5%	<0.001
High-dose Lead	50	110.4 ± 6.7	↑61.2%	<0.001

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Table 2. Low-Density Lipoprotein (LDL) Cholesterol

Group	LDL Cholesterol (mg/dL)	Change vs. Control	p-value
Control	22.1 ± 1.8	-	-
Low-dose Lead	34.5 ± 2.3	↑56.1%	<0.01
Medium-dose Lead	48.7 ± 3.1	↑120.4%	<0.001
High-dose Lead	62.9 ± 4.5	↑184.6%	<0.001

Table 3. High-Density Lipoprotein (HDL) Cholesterol

Group	HDL Cholesterol (mg/dL)	Change vs. Control	p-value
Control	35.4 ± 2.1	-	-
Low-dose Lead	28.7 ± 1.9	↓18.9%	<0.05
Medium-dose Lead	23.2 ± 1.5	↓34.5%	<0.001
High-dose Lead	17.8 ± 1.2	↓49.7%	<0.001

Table 4. Triglycerides

Group	Triglycerides (mg/dL)	Change vs. Control	p-value
Control	85.3 ± 4.5	-	-
Low-dose Lead	102.6 ± 5.1	↑20.3%	<0.05
Medium-dose Lead	128.7 ± 6.8	↑50.9%	<0.001
High-dose Lead	154.2 ± 7.9	↑80.8%	<0.001

Exposure to lead acetate over a 28-day period caused significant dose-dependent changes in serum cholesterol levels in albino rats. Rats treated with 10 mg/kg/day of lead acetate exhibited a 20.1% increase in

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total serum cholesterol compared to the control group (82.3 ± 4.1 mg/dL vs. 68.5 ± 3.2 mg/dL), with a statistically significant difference ($p < 0.05$). Higher doses exacerbated this effect: the medium-dose group (30 mg/kg/day) showed a 42.5% rise (97.6 ± 5.3 mg/dL, $p < 0.001$), while the high-dose group (50 mg/kg/day) had a 61.2% increase (110.4 ± 6.7 mg/dL, $p < 0.001$).

Low-density lipoprotein (LDL) cholesterol levels followed a similar trend, escalating by 56.1% (34.5 ± 2.3 mg/dL) in the low-dose group, 120.4% (48.7 ± 3.1 mg/dL) in the medium-dose group, and 184.6% (62.9 ± 4.5 mg/dL) in the high-dose group compared to controls (22.1 ± 1.8 mg/dL). In contrast, high-density lipoprotein (HDL) cholesterol decreased progressively, dropping by 18.9% (28.7 ± 1.9 mg/dL), 34.5% (23.2 ± 1.5 mg/dL), and 49.7% (17.8 ± 1.2 mg/dL) in low-, medium-, and high-dose groups, respectively, relative to the control group (35.4 ± 2.1 mg/dL).

Triglyceride levels also rose markedly, with increases of 20.3% (102.6 ± 5.1 mg/dL), 50.9% (128.7 ± 6.8 mg/dL), and 80.8% (154.2 ± 7.9 mg/dL) in the low-, medium-, and high-dose groups compared to controls (85.3 ± 4.5 mg/dL). The LDL/HDL ratio, an indicator of cardiovascular risk, surged from 0.62 in controls to 3.53 in the high-dose group, reflecting a shift toward atherogenic lipid profiles.

Statistical analysis (one-way ANOVA with Tukey's post-hoc test) confirmed the significance of these changes ($p < 0.05$ for low-dose effects; $p < 0.001$ for medium- and high-dose effects). These results demonstrate that lead toxicity disrupts lipid metabolism, promoting dyslipidemia characterized by elevated LDL, total cholesterol, and triglycerides, alongside reduced HDL.

The observed dose-dependent increase in total cholesterol, LDL, and triglycerides, coupled with a decline in HDL levels in lead-exposed albino rats, underscores lead's disruptive impact on lipid metabolism. These findings align with studies demonstrating that lead exposure induces oxidative stress and hepatic dysfunction, critical drivers of dyslipidemia (Jomova and Valko, 2011). The liver, the primary site for cholesterol synthesis, is vulnerable to lead's inhibition of hepatic enzymes such as HMG-CoA reductase, a regulator of cholesterol biosynthesis (Ahamed and Siddiqui, 2007).

The reduction in HDL cholesterol (↓49.7% in high-dose groups) may reflect lead's suppression of lecithin-cholesterol acyltransferase (LCAT), an enzyme essential for HDL maturation (Garçon et al., 2006). Concurrently, elevated triglycerides (↑80.8%) likely stem from lead-induced lipolysis inhibition and enhanced fatty acid mobilization, as shown in rodent models (Vaziri and Khan, 2007). The atherogenic LDL/HDL ratio (0.62 vs. 3.53) mirrors patterns in humans with chronic lead exposure, suggesting conserved dysregulation mechanisms (Navas-Acien et al., 2007).

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Histopathological findings, such as hepatic steatosis in lead-treated rats, further implicate multi-organ dysfunction in lipid imbalance (Flora et al., 2012). The dose-response relationship highlights lead's cumulative toxicity, with even low doses (10 mg/kg) causing hypercholesterolemia (↑20.1%), reinforcing the absence of a safe exposure threshold (CDC, 2020). Antioxidant depletion (e.g., reduced glutathione) exacerbates lipid peroxidation, altering lipoprotein stability (Pande et al., 2012).

These findings emphasize stricter lead regulation and lipid monitoring in high-risk populations. Antioxidants like N-acetylcysteine show preclinical efficacy in mitigating dyslipidemia (Flora and Pachauri, 2010) and warrant clinical trials.

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