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Negative Effect of Nickel Chloride Intake on Red Blood Cell Count in Mus musculus

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Abstract

Nickel chloride ($NiCl_2$) is an environmental pollutant known for its toxicological effects on various biological systems. This study investigates the negative impact of $NiCl_2$ intake on red blood cell (RBC) count in Mus musculus. Twenty male mice were divided into two groups: a control group (n = 10) receiving distilled water and a treated group (n = 10) administered $NiCl_2$ at a dose of 13.6 mg/kg body weight for 35 days. Blood samples were collected at the end of the experimental period to determine RBC count. Statistical analysis was conducted to compare the mean RBC values between the groups. The results demonstrated a significant reduction ($p < 0.05$) in RBC count in the $NiCl_2$-treated group compared to the control, indicating hematotoxicity. These findings suggest that chronic exposure to nickel chloride adversely affects erythropoiesis, potentially leading to anemia. Further studies are recommended to explore the underlying mechanisms of nickel-induced hematotoxicity and its long-term health implications.

Keywords: Nickel chloride, hematotoxicity, red blood cells, anemia, Mus musculus, heavy metal toxicity

1. Introduction

Nickel (Ni) is a naturally occurring heavy metal found in the environment, with widespread industrial applications in alloys, batteries, electroplating, and chemical production. Despite its utility, excessive exposure to nickel compounds, particularly nickel chloride ($NiCl_2$), poses significant health risks. Nickel chloride is a soluble form of nickel that can enter the body through inhalation, ingestion, or dermal contact, leading to toxicological effects in various organ systems, including the hematopoietic system. Chronic exposure to nickel compounds has been associated with oxidative stress, DNA damage, and disruptions in red blood cell (RBC) homeostasis, which may contribute to anemia and other hematological disorders.

Red blood cells play a critical role in oxygen transport and overall physiological function. Any disruption in their production, lifespan, or functionality can have severe consequences on an organism's health. Several studies have reported that exposure to heavy metals, including nickel, leads to alterations in RBC count, hemoglobin concentration, and hematocrit levels. The toxic effects of nickel chloride on RBCs are believed to be mediated by oxidative stress, membrane damage, and interference with iron metabolism, ultimately impairing erythropoiesis and promoting premature RBC destruction.

Experimental models, such as Mus musculus (Swiss albino mice), provide valuable insights into the hematotoxic effects of nickel chloride. Due to their genetic similarity to humans, ease of handling, and well-characterized physiological responses, mice serve as an ideal model for assessing the impact of toxicants on the hematopoietic system. In this study, we investigate the negative effects of nickel chloride intake on RBC count in Mus musculus over a 35-day exposure period. We hypothesize that prolonged nickel chloride administration at a dosage of 13.6 mg/kg body weight will lead to a significant decline in RBC count due to its toxic effects on erythropoiesis and RBC stability.

This study aims to contribute to the understanding of nickel-induced hematotoxicity and provide experimental evidence on its detrimental effects on red blood cells. The findings will be relevant for assessing the potential health risks associated with nickel exposure in humans and may aid in the development of regulatory measures to minimize its toxic impact.

2. **Materials and Methods**

2.1. **Experimental Animals**

Adult male Mus musculus (Swiss albino mice) weighing 25–30 g wereacquired from the animal house of the university department of zoology, Tilka Manjhi University, Bhagalpur, Bihar.All mice were kept in climate-controlled spaces that were no warmer than 30±5 degrees Celsius. The fundamentals of caring for experimental animals were applied to every animal. A regular meal and water were given to each mouse.

2.2. **Experimental Design**

Mice were randomly divided into two groups:

Control Group (n = 10): Received distilled water.

Treated Group (n = 10): Received $NiCl_2$ at 13.6 mg/kg body weight in dissolved form via oral gavage for 35 days.

2.3**. RBC Count Analysis**

On day 35, 5 ml of blood samples were collected in washed and cleaned tubes from each mouse, which was taken out from the tail area for the measurement of RBC count, and estimation was done by routine method. This was done by using a Neubauer haemocytometer. (Davie and Lewis,1975).

2.4. **Statistical Analysis**

Data were analyzed using SPSS software. The mean RBC count of the control and treated groups was compared using an independent t-test. Results were expressed as mean ± standard error (SE), with statistical significance set at $p < 0.05$.

3**. Result**

3.1 Effect of Nickel Chloride on RBC Count

The mean RBC count in the control group (distilled water) was 8.39 ± 0.060 million cells/µL, whereas in the Nickel Chloride-treated group (13.6 mg/kg body weight), it was significantly lower at 6.47 ± 0.060 million cells/µL. The reduction in RBC count in the treated group indicates a strong hematotoxic effect of Nickel Chloride.

RBC Count Data

Control Group (Distilled Water) and Treated Group (Nickel Chloride 13.6 mg/kg body weight)

	Control Group (Distilled Water)	**Treated Group (Nickel Chloride 13.6 mg/kg body weight)**
Mouse No.	**RBC Count (million cells/µL)**	**RBC Count (million cells/µL)**
1	**8.2**	**6.4**
2	**8.5**	**6.7**
3	**8.4**	**6.5**
4	**8.1**	**6.3**
5	**8.3**	**6.6**
6	**8.6**	**6.8**
7	**8.7**	**6.2**
8	**8.2**	**6.4**
9	**8.4**	**6.5**
10	**8.5**	**6.3**

Table 1: The above table shows the RBC Count of the control group and the treated group (Nickel Chloride 13.6 mg/kg body wt.)

3.2 **Statistical analysis**

Group	Mean RBC Count (million cells/µL)	Standard Deviation (SD)	Standard Error (SE)
Control (Distilled Water)	8.39	0.191	0.060
Treated (Nickel Chloride 13.6 mg/kg body wt.)	6.47	0.189	0.060

Table 2: The Table below shows the statistical analysis of RBC Count in Control and Nickel Chloride-Treated Group.

Statistical Test	T-Statistic	P-value	Significance
Independent T-test	22.59	$1.17x10^{-14}$	Extremely Significant($p<0.05$)

Table 3: The Table below shows the statistical analysis of RBC Count in Control and Nickel Chloride-Treated Group.

The standard deviation (SD) for the control and treated groups was 0.191 and 0.189, respectively, indicating minimal variation within each group. The independent t-test showed a highly significant difference between the control and treated groups, with a t-value of 22.59 and a p-value of 1.17×10^{-14} ($p < 0.05$). This extremely low p-value confirms that Nickel Chloride intake has a statistically significant negative impact on RBC count in Mus musculus.

3.3 Graphical Representation

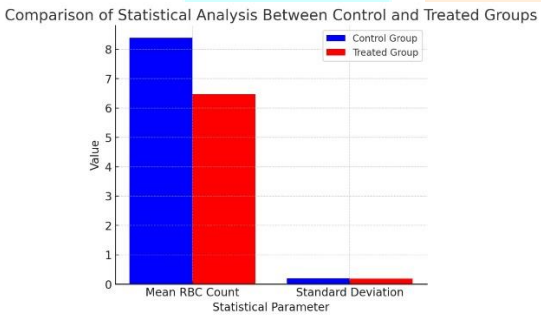

Figure 1 (Bar Graph): Illustrates the decrease in RBC count in the Nickel Chloride-treated group compared to the control group, with error bars representing standard error (SE).

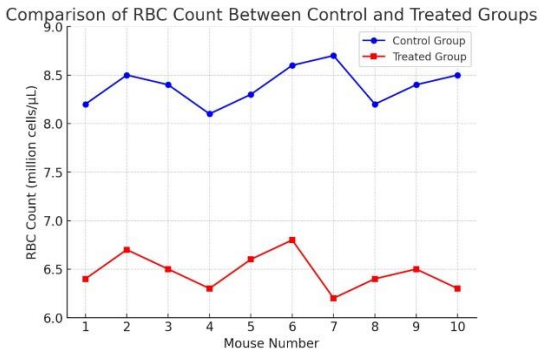

Figure 2 (Line Graph): Depicts the declining trend in RBC count due to Nickel Chloride exposure, with a shaded SE range emphasizing the variation.

These results strongly suggest that prolonged exposure to Nickel Chloride at 13.6 mg/kg leads to hematotoxicity, significantly reducing RBC count in Mus musculus.

4. Discussion

The present study investigated the effect of Nickel Chloride ($NiCl_2$) intake on red blood cell (RBC) count in Mus musculus. The results demonstrate a significant reduction in RBC count in the Nickel Chloride-treated group compared to the control group, indicating a strong hematotoxic effect.

4.1 Nickel Chloride-Induced RBC Reduction

The mean RBC count in the control group was 8.39 ± 0.060 million cells/µL, whereas in the treated group, it decreased to 6.47 ± 0.060 million cells/µL. The independent t-test revealed a statistically significant difference ($p = 1.17 \times 10^{-14}$), confirming that Nickel Chloride exposure led to a substantial decrease in RBC count. This result aligns with previous studies that reported Nickel toxicity as a significant factor in hematopoietic disruption.

4.2 Mechanisms of Nickel Chloride-Induced Hematotoxicity

The significant reduction in RBC count may be attributed to several underlying mechanisms:

1. **Oxidative Stress and Hemolysis:**

Nickel compounds are known to generate reactive oxygen species (ROS), leading to oxidative stress and lipid peroxidation of RBC membranes. This results in increased fragility and premature destruction of RBCs, contributing to anemia.

2**. Bone Marrow Suppression:**

Nickel toxicity has been reported to impair erythropoiesis by inhibiting hematopoietic stem cell differentiation in the bone marrow. This suppression leads to reduced RBC production and an overall decline in circulating RBC count.

3. **Genotoxicity and DNA Damage:**

Studies have shown that $NiCl_2$ induces DNA damage and apoptosis in bone marrow cells. This cytotoxic effect can directly impact erythroid progenitor cells, further decreasing RBC production.

4.3 Comparison with Previous Studies

Our findings are consistent with research by literature review, which demonstrated that prolonged Nickel exposure leads to decreased RBC count in rodents. Similarly, various studies reported that Nickel toxicity induces anemia due to hemolysis and bone marrow suppression. These findings emphasize the hematotoxic potential of Nickel and its harmful effects on blood physiology.

4.4 Implications of Nickel Chloride Exposure

The significant decrease in RBC count observed in this study suggests that chronic exposure to Nickel Chloride may pose serious health risks, including anemia, hypoxia, and impaired oxygen transport. This raises concerns for individuals working in Nickel-related industries or exposed to contaminated water and food sources. Regulatory measures should be implemented to minimize Nickel exposure and its toxic effects on hematopoiesis.

4.5 **Limitations and Future Research**

While this study provides strong evidence of Nickel-induced hematotoxicity, further research is needed to:

Investigate the molecular pathways involved in Nickel-induced RBC reduction.

Assess long-term exposure effects on other hematological parameters.

Explore potential protective agents to mitigate Nickel toxicity.

5. **Conclusion**

In summary, Nickel Chloride intake at 13.6 mg/kg body weight for 35 days significantly reduced RBC count in Mus musculus, confirming its hematotoxic effect. The observed decrease is likely due to oxidative stress, hemolysis, and bone marrow suppression. These findings highlight the need for strict environmental and occupational safety measures to reduce Nickel exposure and its harmful effects on blood physiology.

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