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Review article

The Effects of Chromium and Tin Present in the Environment and Seawater on Biochemical Parameters in Male Rabbits

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Abstract
The biochemical effects of exposure to potassium dichromate ($K_2Cr_2O_7$) and tin chloride ($SnCl_2$) on important liver and kidney function parameters and oxidative stress markers in experimental groups compared to controls are evaluated in this study. Male animals were split into three groups: a control group, a group that received $K_2Cr_2O_7$, and a group that received $SnCl_2$. Biochemical parameters, such as total protein, albumin, bilirubin, urea, creatinine, glucose, liver enzymes (AST and ALT), and plasma TBARS, were measured. The results showed that the $K_2Cr_2O_7$ and $SnCl_2$ groups had significantly lower levels of total protein and albumin than controls, indicating impaired protein synthesis. With $SnCl_2$ having a more noticeable effect, bilirubin levels were higher in the experimental groups, indicating hepatic impairment. Urea and creatinine levels rose as a result of both toxicants, indicating renal impairment, especially in the $K_2Cr_2O_7$ group. Both therapy groups had noticeably higher glucose levels, which is an indicator of metabolic problems. In line with hepatocellular damage, the experimental groups' hepatic enzyme activity (AST and ALT) was noticeably elevated. Furthermore, lipid peroxidation marker TBARS levels were markedly increased, indicating increased oxidative stress, with $K_2Cr_2O_7$ showing a greater effect. In summary, exposure to $K_2Cr_2O_7$ and $SnCl_2$ resulted in notable biochemical changes, with $K_2Cr_2O_7$ exhibiting more marked oxidative and nephrotoxic effects and $SnCl_2$ having a greater influence on hepatic function and protein synthesis. These results highlight the necessity of more investigation into these chemicals' toxicological processes.
Keywords: Metals, Biochemical Parameters, Environment Rabbits.

Introduction

Because of their impact on both the environment and people, anthropogenic activities have emerged as a major area of study interest [1]. The atmosphere has been greatly affected by unchecked pollution brought on by human activity, which has changed biodiversity [2]. Waste that is harmful and useless is produced as a result of this phenomenon. Toxic heavy metals like chromium in the hexavalent form are produced by anthropogenic processes such electroplating, mining, wood preservation, textile, dye, and stainless-steel production, as well as leather tanning [3]. Air, water, and soil are the three main ways that toxic chromium enters the environment [4].

Because of its mutagenicity, toxicity, and carcinogenicity, chromium is one of the main causes of acute illnesses in humans [5]. The United States Environmental Protection Agency (US EPA) has listed and classified hexavalent chromium as a hazardous, high-risk chemical to humans and the environment that is difficult to degrade naturally [6]. It permeates cell membranes with the help of the membrane's sulphate anion transport system and is then reduced to other lower oxidation states, which causes accumulation in different organs and sets off a chain reaction of reactive oxygen species (ROS) and organ damage [7].

Because of their toxicity and long-term effects on living things, heavy metals like tin and chromium are ubiquitous environmental contaminants that have attracted more attention recently. Hexavalent chromium (Cr(VI)), which is the most hazardous because of its high solubility and capacity to produce free radicals that cause oxidative stress and cellular damage, is one of the different oxidation states of chromium (Cr) [8]. The main source of chromium pollution in the environment is industrial processes that discharge large amounts of Cr(VI) into water systems, such as electroplating, leather tanning, and pigment manufacture [9]. In a similar vein, tin (Sn) compounds, which are extensively utilized in industrial processes such as the manufacturing of plastics, coatings, and cans, have the potential to seep into aquatic habitats and cause creatures to bioaccumulate [10]. Particularly known for their endocrine-disrupting properties and capacity to change mammalian metabolic pathways are organ tin compounds. Immune suppression and hepatic and renal failure can result from long-term exposure to tin and its compounds [11].

Because their metabolic pathways are comparable to those of humans, rabbits are frequently used as experimental models and offer important insights into the biochemical and physiological effects of heavy metal toxicity. Research has indicated that rabbits exposed to chromium and tin exhibit significant changes in their antioxidant defenses, lipid peroxidation levels, and serum enzyme activity [12]. These biochemical alterations stress the necessity to assess preventative measures against these metals' detrimental effects in addition to highlighting their hazardous potential [13].

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The purpose of this study is to examine the biochemical effects of chromium and tin exposure in male rabbits, with an emphasis on oxidative stress indicators and liver and kidney function. To reduce the threats to the environment and human health posed by heavy metal contamination of water and the wider ecosystem, it is essential to comprehend these consequences.

Methods

Tested compound

This study examined how male rabbits' liver function and free radicals were affected by potassium dichromat (5 mg/kg; $K_2Cr_2O_7$) and stannous chloride (20 mg/kg; 400g/L; $SnCl_2$). Stannous chloride and potassium dichromat were brought from Omar Al-Mokhtar University's chemistry department in the faculty of science.

Animals and treatments

New Zealand's mature male White rabbits weighing 1.891 ± 27.6 kg at birth and 6 months of age were utilized. The animals were kept in separate cages and weighed once a week for the duration of the three-month trial. Water and food were given freely. 30% berseem (Trifolium alexandrinum) hay, 25% yellow maize, 26.2 percent wheat bran, 14% soybean meal, 3% molasses, 1% $CaCl_2$, 0.4% NaCl, 0.3% mineral and vitamin mixture, and 0.1% methionine were among the pellets that the rabbits were fed.

The following IU/gm of vitamins or minerals were present in the vitamin and mineral premix per kilogram: B12-0.004 g, B5-16.7 g, vitamin A-4000,000, vitamin D3-5000,000, vitamin E-16.7 g, K-0.67 g, vitamin B1-0.67 g, vitamin B2-2 g, vitamin B6-0.67 g, (Rabbit premix made by Holland Feed Inter. Co.) 6.67 g of pantothenic acid, 0.07 g of biotein, 1.67 g of folic acid, 400 g of choline chloride, 23.3 g of zinc, 10 g of Mn, 25 g of Fe, 1.67 g of Cu, 0.25 g of I, 0.033 g of Se, and 133.4 g of magnesium.

The pellets' chemical analysis [14] revealed that their DM base constituted 62.4% nitrogen free extract, 15.8% crude protein, 11.3% crude fiber, 3.7% ether extract, 7.2% ash, and 92.9% organic matter. Groups 2 and 3 received gavage treatment with $SnCl_2$ at a lethal dose of 20 mg/kg B.W./day (1/50 of $SnCl_2$), while the first group served as a control [15]. For 12 weeks in a row, rabbits were given $K_2Cr_2O_7$ by gavage every day at a lethal dose of 5 mg/kg B.W/day (1/50 of DM) [16].

Blood biochemical parameters and enzyme activities

The remaining portion of the separated blood samples was promptly put on ice. Samples were centrifuged at 860 xg for 20 minutes to produce plasma, which was then kept at -20°C until it was needed for analysis. Total protein (TP) was measured in stored plasma samples using the Biuret method in accordance with [17]. The method of [18] was used to quantify the concentration of albumin (A).

The methods described in references [19–21], were used to measure the concentrations of creatinine, urea, and plasma glucose, respectively. The method outlined in [22] was employed to assess the activity of plasma aspartate transaminase (AST; EC 2.6.1.1) and alanine transaminase (ALT; EC 2.6.1.2). Additionally, the thiobarbituric acid-reactive substances (TBARS) in plasma were measured following the technique described in [23].

Statistical analysis

The mean ±SEM was used to express the collected data. The Tukey test and one-way ANOVA were used to evaluate the significant differences. Less than 0.05 is regarded as significant following the identification of the data's normal distribution and the corresponding P-values.

Results

In contrast to the control group, the results show notable changes in the biochemical parameters across the experimental groups, indicating the effects of exposure to $K_2Cr_2O_7$ and $SnCl_2$ on liver and kidney function as well as oxidative stress indicators.In comparison to the control, the $K_2Cr_2O_7$ and $SnCl_2$ groups exhibited lower amounts of total protein and albumin. This decrease points to compromised protein synthesis, most likely brought on by the toxicants' hepatocellular injury. The $SnCl_2$ group's more noticeable drop in albumin levels lends credence to its perhaps more powerful hepatotoxic impact. The experimental groups' bilirubin levels were noticeably higher, with the $SnCl_2$ group showing the highest amounts. As bilirubin buildup is a sign of hepatic insufficiency, this suggests impaired liver function and potential hemolysis. Both the $K_2Cr_2O_7$ and $SnCl_2$ groups had elevated urea and creatinine levels, which indicate renal failure; the creatinine rise is most noticeable in the $K_2Cr_2O_7$ group. This implies that exposure to $K_2Cr_2O_7$ is linked to increased nephrotoxicity. The $K_2Cr_2O_7$ and $SnCl_2$ groups showed hyperglycemia, suggesting possible metabolic abnormalities.

Stress-related hormonal changes or oxidative damage-induced decreased insulin function might be the cause of the high glucose levels. Hepatocellular damage is highlighted by the significant rise in ALT and AST activity in the experimental groups. In comparison to $SnCl_2$, the $K_2Cr_2O_7$ group exhibits greater enzyme levels, which may indicate a more severe hepatotoxic effect. The $K_2Cr_2O_7$ and $SnCl_2$ groups had considerably greater amounts of TBARS, a hallmark of lipid peroxidation, with the $K_2Cr_2O_7$ group having the highest

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levels. This is consistent with the reported biochemical changes and suggests increased oxidative stress. All things considered, our results highlight the oxidative, hepatotoxic, and nephrotoxic effects of $K_2Cr_2O_7$ and $SnCl_2$.

Although both substances significantly altered biochemical measures, $SnCl_2$ had a greater influence on protein synthesis and the $K_2Cr_2O_7$ group had a more noticeable effect on indicators of oxidative stress and renal function. To clarify the underlying processes of these harmful consequences, more investigation is required.

Table 1. Plasma biochemistry of male rabbits treated with ($K_2Cr_2O_7$) and ($SnCl_2$)and their combination

Parameter	***Experimental groups***		
	Control means ± SE	**$K_2Cr_2O_7$ means ± SE**	**$SnCl_2$ means ± SE**
Total protein (g/dl)	6.8 ± 0.100^{ab}	6.4 ± 0.9^{a}	6.5 ± 0.128^{b}
Albumin (g/dl)	4.0 ± 0.060 ^{b}	3.7 ± 0.17^{c}	3.6±0.109^{c}
Bilirubin (mg/dl)	1.5 ± 0.019^{b}	1.68 ± 0.038^{c}	1.7 ± 0.030^{a}
Urea (mg/dl)	39.0 ± 0.512^{a}	46.45 ±1.70^{a}	45.0±1.816^{a}
Creatinine (g/dl)	0.77 ± 0.042^{b}	1.34 ± 0.09^{a}	1.19 ± 0.084^{a}
Glucose (mg/dl)	116.0 ± 0.503^{b}	123.0 ±1.55^{a}	119.0±0.837^{a}
AST(U/L)	41.19 ± 1.652^{a}	47.7 ±2.2^{a}	46.09 ± 3.078^{a}
ALT(U/L)	44.01 ± 1.149^{b}	59.09 ± 3.36^{a}	56.04 ± 2.844^{a}
TBARSplasma (nmol/ml)	1.106 ± 0.094^{c}	3.069 ± 0.08^{a}	2.70 ± 0.03^{b}

Means ± SE are used to express values; each treatment group has n = 15. Significant differences (p<0.05) were observed between mean values within a row that did not share a common superscript letter (a, b, c, d).

Discussion

Blood total protein and albumin levels, together with the activity of blood enzymes like ALT and AST, have been identified as important indicators of hepatic damage and function [24]. The primary cause of the rise in AST and ALT activity in the serum of rabbits given $SnCl_2$ is the enzymes' leaking into the bloodstream from the liver cytosol [25]. In these situations, the activity of AST is greatly elevated and escapes from the damaged hepatic cells into the plasma. Furthermore, because the liver contains vast amounts of this enzyme, the ALT level is useful in detecting the presence of liver disorders. When this organ experiences cellular degeneration or destruction, its level in serum rises [26]. This was supported by a histopathologic study [27] that revealed significant alterations in hepatocytes, duct epithelium proliferation, blood vessel dilatation and congestion, and mononuclear inflammatory infiltrate in rabbits treated with $SnCl_2$. The effects of $SnCl_2$ on inhibiting ALP activities are consistent with findings in rats [28].

Oxidative damage to biomolecules, including lipids, DNA, and proteins, has been linked to a number of chronic diseases, including cancer, cardiovascular disease, cataracts, aging, and other neurological conditions [29]. Important biological targets, including membranes and DNA, can be harmed by ROS, such as the hydroxyl radical (.OH) generated in cells [30]. A toxic metal that is frequently utilized in industrial settings is chromium (VI). It has harmful effects on the liver and other organs [31]. This study demonstrated that Se reduced the liver damage caused by Cr. Histopathological, morphometric, gene expression, and biochemical investigations all supported this. High blood levels of the enzymes aspartate aminotransferase (AST) and alanine aminotransferase (ALT) signify muscular injury, cardiac infraction, and liver damage, including those caused by viral hepatitis. The transformation of alanine into pyruvate and glutamate is catalyzed by serum ALT.As a result, serum ALT is a superior indicator of liver impairment since it is more specific to the liver [32]. Their rise in the current investigation raises the possibility that potassium dichromate produced reactive oxygen species, which would have caused oxidative stress and hepatotoxicity and nephrotoxicity. This might be because oxidative stress impairs their production or causes impaired liver function [33]. Similar results were observed by [34] in rats treated with potassium dichromate.

According to earlier research by [33], Cr caused a substantial and significant rise in the blood levels of the liver enzymes AST and ALT in the current investigation. They explained this by stating that the enzymes' release from the cytoplasm indicated damage to the liver tissue. Other investigations have linked this to the consumption of liver glutathione (GSH), one of the primary components of antioxidant defense, and the onset of oxidative stress [35]. The distribution of copper, iron, manganese, and zinc in organs and tissues is impacted by Cr (VI), a toxin and carcinogen [36,37].

Through the stimulation of the Akt/ERK/AMPK signaling cascade, which is mostly driven by ROS formation, Cr (VI) can cause cytotoxicity in neuronal cells [38]. According to some research, Cr (VI) damages h. epatocytes by causing oxidative damage and lipid peroxidation [39]. Our results are in line with other research that reported impaired liver function following exposure to Cr (VI). L02 hepatocyte damage caused by Cr (VI) is significantly influenced by ROS and mitochondria [40].

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Conclusion

The results of this investigation demonstrate the substantial metabolic disturbances brought on by exposure to tin chloride ($SnCl_2$) and potassium dichromate ($K_2Cr_2O_7$). Significant drops in albumin and total protein levels were brought on by both toxicants, suggesting impaired protein synthesis and possible liver damage. Liver damage was further verified by elevated bilirubin levels, with $SnCl_2$ having a somewhat stronger impact. Overall, this study shows that hepatic, renal, and oxidative stress parameters are considerably impacted by environmental exposure to chromium and tin compounds. These findings highlight the necessity of more research into the harmful processes and possible defenses against these kinds of environmental pollutants.

Conflict of interest. Nil

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المستخلص

تقيم هذه الدراسة التأثيرات الكيميائية الحيوية لتعرض ثنائي كرومات البوتاسيوم وكلوريد القصدير على معايير وظائف الكبد والكلى الرئيسية، بالإضافة إلى علامات الإجهاد التأكسدي في المجموعات التجريبية مقارنة بالضوابط. تم تقسيم الحيوانات الذكور إلى ثلاث مجموعات: مجموعة ضابطة، ومجموعة تم إعطاؤها ثنائي كرومات البوتاسيوم، ومجموعة عولجت بكلوريد القصدير. تم قياس المعايير الكيميائية الحيوية، بما في ذلك البروتين الكلي والألبومين والبيليروبين واليوريا والكرياتينين والجلوكوز وإنزيمات الكبد و المواد التفاعلية مع حمض الثيوباربيتيوريك في البلازما. كشفت النتائج عن انخفاض كبير في مستويات البروتين الكلي والألبومين في مجموعات كرومات البوتاسيوم وكلوريد القصدير مقارنة بالضوابط، مما يشير إلى ضعف تخليق البروتين. ارتفعت مستويات البيليروبين في المجموعات التجريبية، مما يشير إلى خلل في وظائف الكبد، مع ممارسة كلوريد القصدير لتأثير أكثر وضوحًا. تسبب كلا السمينين في زيادة مستويات اليوريا والكرياتينين، مما يعكس خللًا في وظائف الكلى، وخاصة في مجموعة كرومات البوتاسيوم . ارتفعت مستويات الجلوكوز بشكل ملحوظ في كلتا مجموعتي العلاج، مما يشير إلى اضطرابات التمثيل الغذائي. زادت أنشطة إنزيمات الكبد بشكل ملحوظ في المجموعات التجريبية، بما يتفق مع تلف الخلايا الكبدية. بالإضافة إلى ذلك، ارتفعت مستويات المواد التفاعلية مع حمض الثيوباربيتيوريك ، وهو مؤشر على بيروكسيد الدهون، بشكل ملحوظ، مما يسلط الضوء على الإجهاد التأكسدي المعزز، مع إظهار كرومات البوتاسيوم تأثيرًا أقوى. وفي الختام، تسبب التعرض لـ كرومات البوتاسيوم وكلوريد القصدير في حدوث تغييرات كيميائية حيوية كبيرة، حيث أظهر كرومات البوتاسيوم تأثيرات سامة للكلى وأكسدة أكثر وضوحًا، في حين كان لكلوريد القصدير تأثير أقوى على تخليق البروتين ووظيفة الكبد. وتؤكد هذه النتائج على الحاجة إلى مزيد من البحث في الآليات السمية لهذه المركبات.

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