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Research Article

Toxic Effects of Sodium Silicate: A Component of Detergents Used in Washing Vegetables

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Abstract: Detergents containing sodium silicate are widely used to wash vegetables and fruits in markets, raising public health concerns. This study investigated the toxic effects of sodium silicate accumulation in detergent-treated carrots using hematological, hepatic, antioxidant, and lipid profile parameters in Wistar rats. Eighteen male and female albino rats were randomly divided into three groups: control (Group C), and two treatment groups (CA1 and CB2) receiving detergent concentrations of 13.75 and 25.0 mg/mL, respectively, for 14 days. Results demonstrated significant dose-dependent toxic effects across all biomarkers examined, including white blood cells (WBC), red blood cells (RBC), hemoglobin (HMG), alanine transaminase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), albumin, total cholesterol, triglycerides (TAG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL) ($p < 0.05$). Additionally, sodium silicate exposure significantly increased malondialdehyde (MDA) levels and decreased superoxide dismutase (SOD) activity in liver tissue. Body weight loss was observed in both treatment groups. These findings indicate that detergent-induced sodium silicate accumulation in vegetables poses significant health risks and warrants public health intervention.

Keywords: detergent, sodium silicate, bioaccumulation, food safety, toxicity, vegetables

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1. Introduction

The use of detergents to wash fruits and vegetables has become a common commercial practice in markets across developing nations. Detergents are surface-active agents (surfactants) that contain complex mixtures of surfactants, builders, bleaching agents, and enzymes designed to remove foreign matter from contaminated surfaces (Steber et al., 2009). Sodium silicate serves as a critical builder component in detergent formulations, functioning to soften hard water and enhance detergent efficacy. However, the potential for toxic residue accumulation on food products has not been thoroughly investigated, particularly in nutrient-rich vegetables like

carrots.Carrot (*Daucus carota* L.), belonging to the family Apiaceae, is among the most widely cultivated vegetables globally, with annual production exceeding 45 million tonnes. The crop represents a major source of essential nutrients, including carotenoids, polyphenols, anthocyanins, and dietary fiber (Simon & Wolff, 1987). Carrots possess considerable antioxidant properties attributed to their flavonoid and carotenoid content, which confer protective effects against oxidative stress-related diseases (Dias, 2012a). Despite their nutritional value, carrots are frequently washed with detergent solutions by vendors to improve appearance, raising concerns about chemical residue accumulation and potential adverse health effects.Sodium silicate (Na_2SiO_3) is a colorless, water-soluble compound commonly known as water glass or liquid glass. It is utilized extensively in detergent formulations as a builder, with applications extending to pulp and paper industries, sealant manufacturing, and cement production (Lee et al., 2013). Previous toxicological studies have documented renal injury following sodium silicate exposure, with histopathological changes observed in kidney tubules of experimental animals (Basketter et al., 2012a, 2012b). However, comprehensive investigations into multiple organ system effects, particularly hepatic and hematological changes following dietary sodium silicate accumulation, remain limited.The aim of this study was to determine the toxic effects of sodium silicate accumulated in carrot samples following detergent treatment on hematological parameters, hepatic function, antioxidant and peroxidative status in an experimental animal model. Specific objectives included: (1) evaluating the effects of sodium silicate on hematological parameters; (2) assessing hepatic function markers; (3) determining changes in antioxidant enzyme activity; (4) measuring oxidative stress markers; and (5) evaluating lipid profile alterations.

2. Materials and methods

2.1 Experimental Animals and Ethical Considerations

Adult male and female albino Wistar rats weighing 70–200 g was obtained from the Animal Care House at Usmanu Danfodiyo University, Sokoto, Nigeria. Animals were housed in standard cages and provided with balanced commercial pellet diet (200–250 g per day) and unrestricted access to distilled water. Animals were acclimatized for one week prior to experimental initiation. All animal handling procedures and experimental protocols complied with the United States National Institutes of Health Guidelines for Care and Use of Laboratory Animals in Biomedical Research.

Ethical Approval: This study was approved by the Animal Ethics Committee of Usmanu Danfodiyo University, Sokoto, Nigeria (Reference: UDUS/AEC/2023/045). All procedures adhered to institutional guidelines for humane animal treatment and the principles of the 3Rs (Replace, Reduce, Refine) in animal research.

2.2 Experimental Design

Eighteen Wistar rats were randomly allocated into three treatment groups (n = 6 per group): Group C (control), Group CA_1 (13.75 mg/mL sodium silicate), and Group CB_2 (25.0 mg/mL sodium silicate). Sodium silicate was administered daily via oral gavage based on individual body weight for 14 consecutive days. This duration was

Table 1: *Experimental Design and Animal Allocation*

Parameter	**Group C (Control)**	**Group CA_1**	**Group CB_2**
Sample Size (n)	6	6	6
Sex Distribution	3M/3F	3M/3F	3M/3F
Initial Weight (g)	70–200	70–200	70–200
Treatment	Vehicle only	13.75 mg/mL	25.0 mg/mL
Administration Route	Oral gavage	Oral gavage	Oral gavage
Duration (days)	14	14	14

selected to permit assessment of subacute toxicity manifestations. The experimental design and animal allocation are summarized in Table 1.

2.3 Sample Collection and Preparation

On day 14, following a 12-hour fast, all animals were weighed and sacrificed under chloroform anesthesia. Blood samples were collected via cardiac puncture into EDTA tubes and allowed to clot for 30 minutes at room temperature. Serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at −20°C pending analysis. Liver and kidney tissues were immediately excised, weighed, and preserved in 10% neutral buffered formalin for histopathological examination. Tissue samples designated for biochemical analysis were immediately placed in ice-cold 0.1 M phosphate buffer (pH 7.4) and transported to the laboratory for processing.

2.4 Hematological Analysis

Complete blood cell counts were performed using an automated hematology analyzer (Model 6800, Shimadzu, Japan). Parameters assessed included white blood cells (WBC), red blood cells (RBC), hemoglobin (HMG), and packed cell volume (PCV). Hemoglobin was determined spectrophotometrically at 530–550 nm wavelength.

2.5 Biochemical Assays

2.5.1 Tissue Preparation

Tissue samples (1 g) were homogenized with 0.1 M phosphate buffer (pH 7.4) using a glass homogenizer. Homogenates were centrifuged at 5000 rpm for 5 minutes at 4°C. Supernatants were stored at −20°C for subsequent biochemical analyses.

2.5.2 Superoxide Dismutase (SOD) Activity

SOD activity was measured based on the inhibition of adrenaline auto-oxidation. The assay principle relies on the enzyme's capacity to catalyze dismutation of superoxide radicals into molecular oxygen and hydrogen peroxide. The reaction was monitored at 420 nm wavelength. Briefly, 200 µL tissue supernatant was mixed with 2.5 mL carbonate buffer (pH 10.2), and 300 µL adrenaline (0.13 mM) was added. Absorbance was measured at 420 nm immediately and at 1-minute intervals for 3 minutes. SOD activity was expressed as units per milligram of protein.

2.5.3 Malondialdehyde (MDA) Determination

MDA, an end product of polyunsaturated fatty acid peroxidation, was quantified as a marker of oxidative stress. Tissue samples (200 µL) were mixed with 600 µL thiobarbituric acid (TBA) solution and incubated at 95°C for 1 hour. Samples were cooled on ice for 15 minutes, and absorbance was measured at 532 nm using a spectrophotometer. MDA concentration was calculated using the extinction coefficient of $1.56 \times 10^5\ M^{-1}cm^{-1}$.

2.5.4 Hepatic Function Tests

Liver function parameters were assessed in serum using the following biomarkers:

- **Alanine Transaminase (ALT)**: Catalyzes reversible transamination of L-alanine and α-ketoglutarate. Pyruvate formed is reduced to lactic acid by lactate dehydrogenase with concurrent oxidation of NADH. Normal range: <25 IU/L in females, <33 IU/L in males.
- **Aspartate Aminotransferase (AST)**: Catalyzes reversible transamination of L-aspartate and α-ketoglutarate. Oxaloacetate is reduced to malate with NADH oxidation. Normal range: <36 U/L.
- **Alkaline Phosphatase (ALP)**: Catalyzes hydrolysis of p-nitrophenylphosphate to the yellow product p-nitrophenol at alkaline pH (10.3). Normal range: 20–140 U/L.
- **Albumin**: Combines with bromocresol purple (BCP) reagent to form a complex. Absorbance change at 600 nm is directly proportional to albumin concentration. Normal range: 35–50 g/L.

2.5.5 Lipid Profile Analysis

Serum lipids were quantified using enzymatic colorimetric methods:

- **Total Cholesterol**: Measured by the Liebermann-Burchard reaction following incubation with acetic anhydride and concentrated sulfuric acid at 570 nm.
- **Triglycerides (TAG)**: Measured using enzymatic glycerol-3-phosphate oxidase method.
- **High-Density Lipoprotein (HDL)**: Determined following selective precipitation of apolipoprotein B-containing lipoproteins.
- **Low-Density Lipoprotein (LDL)**: Calculated using the Friedewald formula: LDL = Total cholesterol − (HDL + TAG/5).

2.6 Statistical Analysis

Data are presented as mean ± standard deviation. Differences between treatment groups were analyzed using one-way analysis of variance (ANOVA). Post hoc comparisons were performed using Tukey's multiple comparison test. Statistical significance was defined as $p < 0.05$. All statistical analyses were conducted using SPSS version 20.0.

3. Results

3.1 Hematological Parameters

Sodium silicate exposure resulted in significant alterations in hematological parameters (Table 2). White blood cells (WBC) were significantly decreased in Group CB_2 ($3.32 \pm 0.33 \times 10^3$ µL) compared to control ($4.12 \pm 0.05 \times 10^3$ µL; $p < 0.01$), but showed no significant change in Group CA_1. Red blood cell (RBC) counts were significantly reduced in both treatment groups compared to control (CA_1: 0.04 ± 0.06 vs. $0.23 \pm 0.25 \times 10^6$ µL; CB_2: 0.01 ± 0.04 vs. $0.23 \pm 0.25 \times 10^6$ µL; $p < 0.001$). Hemoglobin concentrations were significantly increased in Group CA_1 (1.15 ± 1.24 g/dL) and Group CB_2 (0.20 ± 0.26 g/dL) relative to control (0.12 ± 0.20 g/dL; $p < 0.05$).

Table 2: *Hematological Parameters in Control and Sodium Silicate-Treated Rats. Values are mean ± SD; *p < 0.05, **p < 0.001 compared to control*

Parameter	Control	Group CA_1	Group CB_2	p-value
WBC ($\times 10^3$ µL)	4.12 ± 0.05	3.98 ± 0.12	3.32 ± 0.33*	<0.01
RBC ($\times 10^6$ µL)	0.23 ± 0.25	0.04 ± 0.06**	0.01 ± 0.04**	<0.001
Hemoglobin (g/dL)	0.12 ± 0.20	1.15 ± 1.24*	0.20 ± 0.26	<0.05
PCV (%)	35.2 ± 2.1	28.4 ± 3.2*	24.6 ± 2.8**	<0.01

3.2 Body Weight Changes

Body weight monitoring revealed a differential pattern between control and treatment groups (Table 3). Control animals demonstrated a progressive weight gain, with an increase of 13.8 ± 4.57 g from baseline to day 14. In contrast, both sodium silicate-treated groups exhibited significant weight loss: Group CA_1 lost 7.8 ± 4.03 g, while Group CB_2 lost 10.3 ± 0.96 g ($p < 0.001$ for both groups).

3.3 Antioxidant Enzyme Activity

Superoxide dismutase activity in kidney tissue showed no significant differences between treatment and control groups (Control: 0.585 ± 0.11; CA_1: 0.976 ± 0.05; CB_2: 1.057 ± 0.12 U/mg; $p > 0.05$; Table 4). However, hepatic

SOD activity was significantly reduced in both treatment groups compared to control (Control: 0.84 ± 0.24; CA_1: 0.638 ± 0.02; CB_2: 0.967 ± 0.10 U/mg; $p < 0.001$).

Table 3: *Body Weight Changes During the 14-Day Treatment Period. Values are mean ± SD; **p < 0.001 compared to control*

Group	Initial Weight (g)	Final Weight (g)	Weight Change (g)	p-value
Control	125.4 ± 8.2	139.2 ± 6.8	+13.8 ± 4.57	—
Group CA_1	128.6 ± 7.5	120.8 ± 5.4	−7.8 ± 4.03**	<0.001
Group CB_2	127.2 ± 8.1	116.9 ± 4.3	−10.3 ± 0.96**	<0.001

Table 4: *Superoxide Dismutase (SOD) Activity in Kidney and Liver Tissues. Values are mean ± SD; **p < 0.001 compared to control*

Enzyme/Tissue	Control	Group CA_1	Group CB_2	p-value
SOD Activity (U/mg protein)				
Kidney	0.585 ± 0.11	0.976 ± 0.05	1.057 ± 0.12	>0.05
Liver	0.84 ± 0.24	0.638 ± 0.02**	0.967 ± 0.10**	<0.001

3.4 Oxidative Stress Markers

Malondialdehyde levels, indicating lipid peroxidation, were dramatically elevated in both treatment groups (Table 5). Control animals demonstrated baseline MDA levels of 5.51 ± 0.35 µM/mL, while Group CA_1 exhibited a 337% increase (24.09 ± 0.94 µM/mL; $p < 0.001$) and Group CB_2 showed a 397% increase (27.44 ± 0.46 µM/mL; $p < 0.001$).

Table 5: *Malondialdehyde (MDA) Levels as Marker of Oxidative Stress. Values are mean ± SD; **p < 0.001 compared to control*

Biomarker	Control	Group CA_1	Group CB_2	p-value
MDA (µM/mL)	5.51 ± 0.35	24.09 ± 0.94**	27.44 ± 0.46**	<0.001
Percent Increase	—	+337%	+397%	—

3.5 Hepatic Function Parameters

All assessed hepatic function markers demonstrated significant dose-dependent elevation (Table 6). Alanine transaminase increased from 22.20 ± 0.84 U/L (control) to 48.40 ± 0.89 U/L (CA_1) and 55.00 ± 0.71 U/L (CB_2). Aspartate aminotransferase showed similar increases (control: 19.6 ± 0.55; CA_1: 23.8 ± 0.45; CB_2: 27.0 ± 0.68 U/L; $p < 0.001$). Alkaline phosphatase levels elevated markedly from 58.8 ± 0.19 U/L (control) to 168.2 ± 0.55 U/L (CA_1) and 169.8 ± 0.78 U/L (CB_2; $p < 0.001$). Serum albumin concentrations were significantly decreased in both treatment groups ($p < 0.001$).

3.6 Lipid Profile

Significant alterations in lipid metabolism were observed (Table 7). Total cholesterol increased approximately 160–177% in treated animals (CA_1: 7.92 ± 0.71; CB_2: 8.42 ± 0.71 mmol/L vs. control: 3.03 ± 0.47 mmol/L; $p <$

0.001). Triglyceride levels were reduced in Group CA_1 (8.7 ± 0.82 mmol/L) but remained elevated in Group CB_2 (14.4 ± 0.11 mmol/L) compared to control (15.2 ± 0.44 mmol/L). High-density lipoprotein cholesterol was dramatically reduced in both treatment groups (CA_1: 1.7 ± 0.33; CB_2: 1.5 ± 0.33 mmol/L vs. control: 7.4 ± 0.44 mmol/L; $p < 0.001$). Low-density lipoprotein cholesterol showed a dose-dependent increase in Group CB_2 ($p < 0.001$) but not in Group CA_1.

Table 6: *Hepatic Function Parameters in Control and Sodium Silicate-Treated Rats. Values are mean ± SD; **p < 0.001 compared to control*

Hepatic Biomarker	Control	Group CA_1	Group CB_2	p-value
ALT (U/L)	22.20 ± 0.84	48.40 ± 0.89**	55.00 ± 0.71**	<0.001
AST (U/L)	19.6 ± 0.55	23.8 ± 0.45**	27.0 ± 0.68**	<0.001
ALP (U/L)	58.8 ± 0.19	168.2 ± 0.55**	169.8 ± 0.78**	<0.001
Albumin (g/dL)	3.8 ± 0.24	2.1 ± 0.33**	1.9 ± 0.28**	<0.001

Table 7: *Serum Lipid Profile in Control and Sodium Silicate-Treated Rats. Values are mean ± SD; **p < 0.001 compared to control*

Lipid Parameter	Control	Group CA_1	Group CB_2	p-value
Total Cholesterol (mmol/L)	3.03 ± 0.47	7.92 ± 0.71**	8.42 ± 0.71**	<0.001
Triglycerides (mmol/L)	15.2 ± 0.44	8.7 ± 0.82	14.4 ± 0.11	<0.05
HDL (mmol/L)	7.4 ± 0.44	1.7 ± 0.33**	1.5 ± 0.33**	<0.001
LDL (mmol/L)	1.2 ± 0.31	1.8 ± 0.42	2.9 ± 0.58**	<0.001

4. Discussion

This investigation demonstrates that sodium silicate, a ubiquitous component of commercial detergent formulations, accumulates in vegetable tissues and exerts significant dose-dependent toxicity across multiple organ systems. The findings are particularly concerning given the common practice of using detergent solutions to wash produce in markets throughout developing regions.The observed reductions in red blood cell counts and white blood cell populations are consistent with previous reports of sodium silicate-induced hemato-toxicity. The mechanism underlying these changes likely involves direct cytotoxic effects and inflammatory responses. The significant weight loss observed in treated animals suggests systemic metabolic disturbance, potentially reflecting reduced nutritional absorption or increased metabolic demands associated with detoxification processes (Azizullah *et al.*, 2011).The dramatic elevation in malondialdehyde concentrations increases of 337% and 397% in the lower and higher dose groups, respectively indicates severe lipid peroxidation and overwhelming oxidative stress. These findings are coupled with significantly reduced hepatic superoxide dismutase activity, suggesting impaired antioxidant defense mechanisms. This combination of elevated oxidative stress markers and reduced antioxidant enzyme activity represents a critical pathological state associated with cellular injury and tissue damage (Hunyadi, 2019).The marked elevations in hepatic transaminases (ALT and AST), alkaline phosphatase, and the reduction in serum albumin collectively indicate significant hepatic dysfunction. These changes suggest sodium silicate-induced hepatocellular injury with impaired synthetic function. The mechanisms underlying hepatotoxicity may involve direct chemical injury, oxidative stress-mediated damage, or inflammatory responses triggered by foreign chemical exposure. The severity of elevation in ALP is particularly notable, suggesting possible cholestatic changes or increased hepatocellular membrane permeability (Misra & Fridovich, 1989).The lipid profile alterations demonstrate significant dysregulation of cholesterol homeostasis. The marked reduction in HDL

cholesterol coupled with elevated LDL cholesterol and total cholesterol constitutes an atherogenic lipid profile, predisposing to cardiovascular complications. These changes may reflect hepatic dysfunction affecting lipid synthesis and metabolism, or direct effects on lipid metabolism regulatory pathways.This study confirms prior observations regarding sodium silicate renal toxicity while extending understanding to include hepatic, hematological, and metabolic effects. The dose-response relationship observed across all parameters strengthens the evidence for a causal relationship between sodium silicate exposure and multisystem toxicity. The human health implications are substantial, as consumers inadvertently exposed through contaminated vegetables may accumulate sodium silicate in tissues, particularly individuals with repeated dietary exposure.

5. Conclusions

This investigation provides compelling evidence that detergent-induced sodium silicate residues accumulating on vegetables pose significant health hazards. Exposure to sodium silicate at concentrations present in detergent-treated produce induced dose-dependent toxic effects on hematological, hepatic, antioxidant, and lipid metabolism parameters in experimental animals. The constellation of toxic effects observed including hepatocellular injury, oxidative stress, dyslipoproteinemia, and hematologic abnormalities suggests potential for serious health consequences in human populations chronically exposed through contaminated food sources.Given these findings, regulatory intervention is urgently needed to restrict detergent use for washing edible vegetables. Consumer education regarding food safety and the dangers of detergent-contaminated produce is essential. Future investigations should examine human populations with varying exposure levels and explore long-term chronic exposure effects. Additionally, the identification and promotion of safer vegetable washing alternatives would constitute an important public health measure.

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