

Evaluation of Serum Uric Acid, Calcium, and Albumin in Medical Students Exposed to Short-Term Formalin in Okofia, Nnewi, Nigeria

Augustine Chinedu Ihim¹, Sunday Joy Ebubechi¹, Judith Ndukwu¹, Tochukwu Anthony Ikwelle¹, Patrick Chinedu Obi², Ifeoma Joy Onuora³, and Romanus Ogai Ogalagu⁴

¹Department of Medical Laboratory Science, Nnamdi Azikiwe University, Nnewi Campus, Awka, Nigeria. ²³Department of Internal Medicine, Federal University Teaching Hospital, Owerri, Nigeria. ³Department of Medical Laboratory Science, Faculty of Health Sciences and Technology, Chukwuemeka Odumegwu Ojukwu University, Igbariam, Nigeria ⁴Department of Biochemistry, Tansian University, Umunya, Nigeria

ABSTRACT

Background: Formaldehyde, a widely used preservative in cadaveric dissections, exposes medical students to potential health risks during their anatomical education. **Objectives:** To evaluate the levels of uric acid, calcium, and albumin in medical students exposed to short-term (3-hour) formalin in Nnamdi Azikiwe University, Nnewi, Nigeria. **Materials and Methods:** Forty-five participants within the age range of 18 – 30 years were recruited for this research. Their baseline samples (before 3-hour exposure) were considered as control (pre-exposure) while their samples collected after 3 hours of exposure were considered as post-exposure samples (test). The levels of uric acid, calcium, and albumin were determined using a spectrophotometric technique. The results were subjected to statistical analysis using a paired t-test and Pearson correlation coefficient. **Results:** A significant decrease in the mean values of uric acid (0.19 ± 0.09) (mmol/l), and calcium (1.87 ± 0.23) (mmol/l), compared to the baseline ($p < 0.05$) was observed. The mean value of the diastolic blood pressure (mmHg) was significantly increased post-exposure to formalin compared with the baseline ($p < 0.05$). There was a weak positive correlation between post-formalin uric acid and body mass index (BMI) of the participants ($r = 0.466$, $p = 0.001$). **Conclusion:** The findings suggest that a three-hour formalin exposure caused a significant drop in uric acid and calcium serum levels, suggesting early signs of kidney stress and disrupted calcium balance. These effects, if repeated, could raise long-term health risks like renal damage and bone loss. The study calls for improved safety practices and ongoing health checks for those regularly exposed.

Keywords: Albumin, Calcium, Formaldehyde, Diastolic, Systolic, Uric Acid.

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*Correspondence

Augustine Chinedu Ihim.

Department of Medical Laboratory Science, Faculty of Health Sciences and Technology, Nnamdi Azikiwe University, Awka, Nigeria.

Email: ac.ihim@unizik.edu.ng

Tel: +2348035985883

Article Metrics

Submitted: 16 April 2025

Accepted: 21 May 2025

Published: Jan-June 2026

Citation:

AC Ihim, SJ Ebubechi, J Ndukwu, TA Ikwelle, PC Obi, IJ Onuora, et al., Evaluation of serum uric acid, calcium, and albumin in medical students exposed to short-term formalin in Okofia, Nnewi, Nigeria

Trop J Med Res. 2025;25(1):53-62.

DOI: 10.5281/zenodo.21461885.

Journal Metrics

ISSN: p1119-0388, e2505-0338

Website: www.tjmr.org.ng

E-mail: info.tjmr@gmail.com

Publisher: cPrint, Nig. Ltd

Email: cprintpublisher@gmail.com

Access Code



www.tjmr.org.ng

INTRODUCTION

Formaldehyde is a chemical compound naturally occurring in the atmosphere, in some foods, and in the organisms of mammals as a product of oxidative metabolism and, thus, is considered a ubiquitous pollutant [1] and in its pure form, has a characteristic pungent odour and is an irritant to the respiratory tract [2]. Formaldehyde is an organic compound with the formula, CH_2O and structure $\text{H}-\text{CHO}$. The compound is a colourless gas that is manufactured by passing a mixture of methanol vapour and oxygen over a silver catalyst heated to a temperature of 635°C .

Formaldehyde was first reported in 1859 by the Russian chemist Aleksandr Butlerov [3]. It was conclusively identified by August Wilhelm von Hofmann (1867), who first announced the production of formaldehyde by passing methanol vapour in air over a hot platinum wire [4]. Commercially, formaldehyde gas is dissolved in water (a maximum of 40 parts of gas to 100 ml of water), and an aqueous solution, which is 40% by volume (or 37% by mass) of the gas, is formed and is referred to as formalin (100% formalin). A small amount of stabilizer, such as methanol, is usually added to suppress oxidation and polymerization [5]. Formalin is commonly used as a tissue preservative or bactericide in embalming fluids and medical laboratories. Nurses, medical technicians, laboratory scientists, and even medical students are exposed to formaldehyde in their everyday work [4]. The primary route of exposure to formaldehyde is through inhalation, from where it can be absorbed into the pulmonary system. The other routes of exposure are through the skin and gastrointestinal tract, but these are to a very lesser extent [6].

According to the National Cancer Institute, when formaldehyde is present in the air at levels exceeding 0.1 ppm, some individuals may experience adverse effects such as watery eyes, burning sensations in the eyes, nose, and throat, coughing, wheezing, nausea, and skin irritation. Some people are very sensitive to formaldehyde, whereas others do not react to the same level of exposure [1]. Inflammations of the skin, eyes, and lungs may result from brief exposure

to formalin at concentrations as low as 0.3 ppm of air. Around 0.05 ppm is the lowest concentration at which many people can detect the smell of formaldehyde, and multiple exposures (between 5 and 30 ppm) might start or worsen asthma symptoms. Its gas or vapour can irritate the eyes, nose, and respiratory tract, causing watery eyes, sneezing, headaches, burning sensations in the throat, cough, and respiratory difficulties. In addition to cancer, breathing in formaldehyde for an extended amount of time can cause digestive, cardiac, nephrological, pulmonary, cardiac, and neurological issues [7].

As with other toxicants, the precise mechanism of action of formalin depends on the cell type, dose, and duration of exposure [8]. Short-term formaldehyde exposure causes renal dysfunction, inflammation, and oxidative damage, leading to changes in kidney function [8, 9, 10]. Uric acid is a by-product of purine metabolism and is involved in the antioxidant defence system. Research has shown that uric acid, as a biomarker to assess renal function, has been implicated in the short-term effects of formalin exposure [2]. When the kidney function is impaired, excretion of uric acid would be affected, resulting in elevated levels of uric acid (hyperuricemia) [11]. Calcium is a vital electrolyte involved in bone health, muscle contraction, nerve signalling, and plays a regulatory role in kidney function through its influence on renal calcium handling and vitamin D metabolism [12]. The primary source of calcium is diet. It is essential in bone mineralization, molecular contraction, nerve impulse transmission, blood clotting, and hormone secretion. Alterations in calcium levels can impair physiological processes that depend on calcium, including muscle contraction, nerve transmission, blood clotting, and bone metabolism [12]. When there is impairment of the kidney, calcium activity would be affected resulting in either hypocalcaemia ($<8.8\text{mg/dl}$) or hypercalcaemia ($>10.7\text{mg/dl}$) as the case may be [13, 14].

Formaldehyde is mostly processed by the liver after exposure, regardless of the mode of exposure. But this metabolism hurts the liver and, at certain

quantities, can cause hepatotoxicity, or damage to the liver [15]. This is because exposure to formaldehyde, which is a toxic gas, increases intracellular reactive oxygen species (ROS) more than is physiologically allowed. This leads to an imbalance between oxidant molecules and the body's antioxidant defences. An alteration to the protective mechanisms of the body's antioxidants results in oxidative damage to biomolecules, including proteins (through denaturation), lipids (through peroxidation), and nucleic acids (through change in DNA structure) [8, 16, 17]. Serum albumin is essential for antioxidant defence in both oxidative stress and normal circumstances [18]. The structure of the protein has a direct bearing on its particular antioxidant properties. Its antioxidant properties result from its numerous ligand-binding abilities as well as its ability to trap free radicals [19]. For the sake of medical students' health, it is crucial to identify alterations in these biomarkers as a result of formaldehyde exposure, which could lead to improved safety procedures in anatomy laboratories being improved. The results of the study can also make a substantial contribution to occupational health standards that will protect healthcare workers' health and safety against formaldehyde exposure in a range of work environments. This study is consistent with wider worries about workplace health, environmental exposure, and the general health of medical professionals.

Therefore, the problem to be addressed in this study is to investigate and determine the effects of short-term exposure to formalin on uric acid, calcium, and albumin levels and thereby, assess the potential health risks associated with this exposure on medical students in the College of Health Sciences and Technology, Nnewi Campus, Okofia, Anambra State, Nigeria. This study aims to evaluate the biochemical alterations in serum uric acid, calcium, and albumin levels resulting from short-term formalin exposure among medical students, to better understand the potential renal and hepatic implications and inform necessary preventive health strategies in educational laboratory settings.

MATERIALS AND METHODS

Study site

The study was conducted at the Anatomy laboratory of the Department of Anatomy, Faculty of Basic Medical Sciences, College of Health Science, Nnamdi Azikiwe University, Nnewi Campus, Anambra state.

Study design

This study is a longitudinal study designed to assess the effects of short-term exposure (three hours) to formalin on the uric acid, calcium and albumin levels of medical students who attend dissection classes and have already been exposed in the Anatomy Laboratory in College of Health Science, Nnewi Campus, Anambra State. The dissection class was slated to last for 3 hours. A total of 45 participants within the age bracket of 18 – 30 years were recruited. Their baseline samples (before 3 hours of exposure) were considered as control (pre-exposure) while their samples collected after 3 hours of exposure were considered post-exposure samples (test). A structured data collection proforma was used to obtain objective biochemical data and relevant participant information, including demographic details such as age, sex, duration and frequency of formalin exposure (hours per week), environmental/exposure variables such as time since last exposure, location and ventilation status of the dissection room, self-reported symptoms (e.g., eye irritation, headache), as well as medical history.

Sampling procedure

Venous blood samples (5 mL each) were collected from participants using standard venepuncture techniques and dispensed into plain vacutainer tubes. The samples were allowed to clot at room temperature and then centrifuged at 3,000 rpm for 10 minutes using a six-bucket centrifuge model China to separate the serum. The resulting serum was transferred into sterile cryovials and stored at -20°C until analysis. All samples were transported and handled under cold chain conditions to maintain analyte integrity. Serum uric acid concentration was determined using the Uricase enzymatic colorimetric method, as described [20]. This method involves the

enzymatic oxidation of uric acid to allantoin, carbon dioxide, and hydrogen peroxide, with the latter reacting with a chromogen in the presence of peroxidase to produce a red quinonimine complex. The absorbance of this complex, measured at 505 nm, is directly proportional to the uric acid concentration. Serum calcium levels were quantified using the Arsenazo III colorimetric method, according to [21]. In this assay, calcium forms a purple-colored complex with Arsenazo III in a slightly alkaline medium. The intensity of the color, measured at 570 nm, corresponds to the calcium concentration in the sample. Serum albumin concentration was assessed using the Bromocresol Green (BCG) dye-binding method, based on the protocol described by [22]. In this technique, albumin binds specifically to the BCG reagent under acidic conditions to form a green-colored complex. The absorbance of the resulting solution was measured at 630 nm, and the color intensity was directly proportional to the albumin concentration. All analyses were performed using an automated spectrophotometer, and appropriate internal quality control procedures were followed throughout the analytical process to ensure precision and reliability of results. Blood pressure was measured using a manual sphygmomanometer and stethoscope with the participant in a seated position after a five-minute rest. Measurements were taken twice at one-minute intervals on the left arm positioned at heart level, and the average of the two readings was used for analysis following the international standard [23].

Eligibility criteria

Inclusion criteria: Participants from the Faculty of Basic Medical Sciences exposed to formalin on the course of their study within the age range of 18 – 30 years were recruited for the research. Their baseline samples were considered as control (pre-exposure) while their samples collected after 3 hours of exposure were considered as post-exposure samples (test).

Exclusion criteria: Participants who are not within the age range of 18 – 30 years, participants who are sick or non-students of the Faculty of Basic Medical Sciences, and unwilling participants were excluded from the research.

Statistical analysis:

Data analysis was calculated using Pearson's correlation coefficient test and paired Student's t-test. Statistical Package for Social Sciences (SPSS) version 23.0 was used for the study of the results. Data was presented as mean $\pm$ standard deviation. The level of significance was set at $p < 0.05$. Values were deemed significant when $p < 0.05$.

RESULTS

Comparison of Serum Uric Acid, Calcium, and Albumin Levels in Participants Before and After Short-Term Formalin Exposure

A short-term (3-hour) exposure to formalin resulted in a statistically significant reduction in mean serum uric acid levels from 0.31 ± 0.19 mmol/l to 0.19 ± 0.09 mmol/l ($p < 0.05$), and serum calcium levels from 2.20 ± 0.23 mmol/l to 1.87 ± 0.23 mmol/l ($p < 0.05$). In contrast, no significant change was observed in mean serum albumin levels, which slightly increased from 39.47 ± 6.95 g/l to 40.72 ± 6.26 g/l ($p > 0.05$). (Table 1)

Comparison of Systolic and Diastolic Blood Pressure in Participants Before and After Short-Term Formalin Exposure

Short-term formalin exposure did not produce a statistically significant change in mean systolic blood pressure, which decreased slightly from 126.49 ± 12.25 mmHg to 122.87 ± 14.75 mmHg ($p > 0.05$). However, a significant increase was observed in mean diastolic blood pressure, rising from 73.84 ± 9.32 mmHg to 76.87 ± 9.55 mmHg ($p < 0.05$). (Table 2).

Variables	Pre-formalin Exposed (n=45)	Post-formalin Exposed (n=45)	t-value	p-value
Uric Acid (mmol/l)	0.31 ± 0.19	0.19 ± 0.09	3.722	0.001
Calcium (mmol/l)	2.20 ± 0.23	1.87 ± 0.23	6.384	< 0.001
Albumin (g/l)	39.47 ± 6.95	40.72 ± 6.26	-0.954	0.345

Correlation Between Post-Formalin Biochemical Parameters and Body Mass Index (BMI), Systolic Blood Pressure (SBP), and Diastolic Blood Pressure (DBP)

A moderate positive correlation was observed between post-formalin serum uric acid and body mass index

(BMI) ($r = 0.466$, $p = 0.001$), suggesting that higher BMI may influence uric acid levels following exposure. However, no significant correlations were found between post-formalin uric acid and either systolic ($r = -0.113$, $p = 0.461$) or diastolic blood pressure ($r = -0.057$, $p = 0.709$). Additionally, post-formalin calcium levels showed no significant associations with BMI ($r = 0.116$, $p = 0.447$), systolic BP ($r = 0.004$, $p = 0.981$), or diastolic.

BP ($r = 0.079$, $p = 0.604$). Similarly, no significant correlations were observed between post-formalin albumin levels and BMI ($r = 0.033$, $p = 0.830$), systolic BP ($r = 0.097$, $p = 0.528$), or diastolic BP ($r = 0.168$, $p = 0.270$). (Table 3).

Table 1. The values of serum Uric Acid (mmol/l), Calcium (mmol/l), and Albumin (g/l) of the participants before formalin exposure and after formalin exposure (Mean ± SD)

*Statistically significant at ($p < 0.05$).

Table 2. The values of the systolic blood pressure (mmHg) and diastolic blood pressure (mmHg) of the participants before and after formalin exposure (Mean ± SD).

Variables	Pre-formalin Exposed (n=45)	Post-formalin Exposed (n=45)	t-value	p-value
SBP (mmHg)	126.49 ± 12.25	122.87 ± 14.75	1.408	0.166
DBP (mmHg)	73.84 ± 9.32	76.87 ± 9.55	-2.221	0.032

*Statistically significant at ($p < 0.05$).

SBP = Systolic Blood Pressure

DBP = Diastolic Blood Pressure

Table 3. Association of the serum levels of Uric Acid (mmol/l), Calcium (mmol/l), and Albumin (g/l) with the Body Mass Index (kg/m^2), systolic blood pressure (mmHg), and diastolic blood pressure (mmHg) of the participants exposed to short-term formalin.

Parameters		BMI (kg/m ²)	SBP (mmHg)	DBP (mmHg)
Uric Acid (mmol/l)	R	0.466	-0.113	-0.057
	P	0.001	0.461	0.709
Calcium (mmol/l)	R	0.116	0.004	0.079
	P	0.447	0.981	0.604
Albumin (g/l)	R	0.033	0.097	0.168
	P	0.830	0.528	0.270

*Statistically significant at ($p < 0.05$).

SBP = Systolic Blood Pressure

DBP = Diastolic Blood Pressure

BMI = Body Mass Index

DISCUSSION

The main findings of this study indicate that short-term exposure to formalin significantly decreased serum uric acid and calcium levels among medical students, while serum albumin levels remained statistically unchanged. A significant increase was observed in diastolic blood pressure following exposure, with no significant effect on systolic pressure. A moderate positive correlation was also identified between post-exposure uric acid levels and BMI. These results collectively suggest that even brief formalin exposure can induce measurable physiological alterations, particularly affecting renal and vascular functions.

Formaldehyde's long-term stability and non-degradability make it a popular choice for cadaver preservation in medical schools [24]. As a result, there is a greater possibility that medical instructors and students will breathe in formaldehyde from cadavers during the dissection process [25]. This study assessed the serum levels of uric acid, calcium, and albumin in medical students with prior formalin exposure, following an additional controlled exposure lasting three hours at the Nnewi Campus, to determine the biochemical impact of short-term formalin exposure on these parameters. A significant decrease in the mean value of the serum uric acid of the participants post-exposure to short-term formalin compared with the baseline was observed ($p < 0.05$). This finding aligns with the results of a previous animal study involving twenty-eight male Fischer rats [26], which demonstrated a significant reduction in serum uric acid levels following high-

concentration formaldehyde exposure. The observed decrease suggests that uric acid may function as a free radical scavenger within renal tissue, counteracting oxidative damage induced by formaldehyde and potentially contributing to the pathogenesis of renal injury. Formaldehyde exposure has been shown to provoke renal dysfunction, inflammation, and oxidative stress, all of which may either suppress uric acid synthesis or enhance its renal clearance, ultimately resulting in hypouricemia [27]. Conversely, a study conducted on fifty medical students exposed to formalin reported findings that contradict the previously noted and the present study's reduction in uric acid levels [28]. In that study, the mean serum uric acid concentration was significantly elevated following formalin exposure. These opposing outcomes may be attributed to several factors, including the complexity of metabolic responses to formaldehyde, variations in study design, differences in exposure dose and duration, as well as individual physiological variability among participants.

Similarly, a significant decrease was observed in the mean serum calcium levels of participants following short-term formalin exposure, compared to baseline values. According to [29], alterations in intracellular calcium levels, whether elevated or reduced, are influenced by the site of exposure, the intensity of exposure, and its duration, highlighting the critical role of location, magnitude, and exposure time in determining physiological outcomes. The observed reduction in calcium concentration may be due to formalin's interference with calcium metabolism

and absorption, potentially through disruption of calcium-binding proteins or calcium channels [30]. The kidneys are central to calcium homeostasis by regulating calcium reabsorption and excretion; thus, formalin-induced renal tubular damage or compromised kidney function could impair this regulatory mechanism, leading to decreased serum calcium levels [31].

However, no statistically significant difference was observed in the mean serum albumin concentration of participants following short-term (3-hour) formalin exposure compared to baseline values. This outcome may be attributed to several factors, including the possibility that the concentration of formalin was insufficient to elicit measurable changes in serum albumin levels or that formaldehyde-albumin interactions were minimal. The body's defence mechanisms may have facilitated the immediate clearance of formaldehyde or triggered compensatory responses aimed at maintaining albumin homeostasis within the normal physiological range. These factors collectively could explain the stability in serum albumin levels observed post-exposure. This finding is consistent with the study [32], which also reported no significant changes in mean serum albumin levels following low-concentration formaldehyde exposure. Conversely, it contradicts [28], who reported a significant increase in serum albumin levels post-formaldehyde exposure, suggesting that variations in exposure conditions and biological response may account for the discrepancy.

No significant difference was observed in the mean systolic blood pressure (SBP) of participants following a 3-hour formalin exposure. A significant increase was noted in the diastolic blood pressure (DBP) post-exposure. This finding contrasts with the study [33], which reported no significant changes in either systolic or diastolic blood pressure following a 90-minute formaldehyde exposure. [34] Documented a significant elevation in systolic blood pressure in individuals exposed to formalin for six hours per week over six months. The disparity among these findings may be attributed to variations in the duration and frequency of formalin exposure, as well as differences in sample sizes used across

studies. The lack of significant change in systolic blood pressure and the significant increase in diastolic pressure following short-term exposure may suggest a selective vascular response to formaldehyde [35]. Formalin is known to irritate mucosal surfaces and may trigger sympathetic nervous system activation through stress-induced mechanisms. This sympathetic stimulation could result in peripheral vasoconstriction, thereby increasing diastolic pressure [36]. Formaldehyde exposure has also been implicated in oxidative stress and endothelial dysfunction, which can impair vasodilation and contribute to elevated vascular resistance [35], further explaining the rise in diastolic blood pressure. The absence of a corresponding increase in systolic pressure may be due to the brief exposure duration or effective cardiovascular compensatory mechanisms that modulate cardiac output. These findings warrant further exploration, particularly in controlled studies designed to assess cardiovascular outcomes following chemical exposure.

A moderate positive correlation was observed between post-formalin exposure serum uric acid levels and the body mass index (BMI) of participants. This finding aligns with the study [37], which also reported a significant positive association between uric acid concentration and BMI. Elevated BMI, particularly in overweight and obese individuals, has been linked to increased uric acid production and reduced renal excretion, thereby contributing to hyperuricemia [38]. This suggests that BMI may act as a contributing factor in uric acid metabolism, particularly following chemical exposures such as formalin.

A key strength of this study lies in its structured design involving pre- and post-exposure biochemical assessments, which enabled the evaluation of immediate physiological responses to formalin exposure in a real-world academic setting. The use of objective biochemical markers, standardized laboratory methods, and a controlled exposure window enhances the internal validity of the findings. However, the study is not without limitations. The relatively small sample size and the short-term (3-hour) exposure duration limit the

generalizability and the ability to draw definitive conclusions regarding long-term effects. The potential confounders, such as diet, hydration status, and pre-existing health conditions, were not extensively controlled for. Therefore, while the study provides preliminary insights into acute biochemical responses to formalin exposure, larger-scale, longitudinal research is recommended to fully understand its chronic health implications.

CONCLUSION

The findings of this study indicate that a three-hour exposure to formalin led to a significant reduction in serum uric acid and calcium levels, hypouricemia and hypocalcaemia, respectively, among the exposed participants. These biochemical alterations suggest a potential physiological response to short-term formaldehyde exposure, possibly reflecting early renal stress and disruption in calcium metabolism. If sustained or repeated over time, such changes may predispose individuals to renal impairment and increased risk of osteoporosis. These findings underscore the need for stricter safety measures and regular monitoring of individuals routinely exposed to formalin, particularly in educational or laboratory settings. Further longitudinal studies with larger sample sizes are recommended to explore the long-term health consequences of repeated formalin exposure. It is essential to implement adequate safety protocols in educational environments to reduce formalin exposure. Regular monitoring of uric acid and calcium levels in students exposed to formalin should be considered as part of a proactive approach to detect and address any potential adverse health effects on time.

Acknowledgments

We sincerely appreciate the contributions of the individuals who participated in this study. Their time, effort, and invaluable insights have been essential to the success of this research.

Authors Contribution

IAC conceptualized and designed the study. IAC, SJE, and NDJ contributed to the implementation of the project and revision of the manuscript. IAC, ITA, PCO, OIJ, and ROO were involved in data analysis, manuscript preparation, and final revisions. All authors participated in the writing and revision of the manuscript. The authors read, approved the final manuscript, and agree to be accountable for all aspects of the work.

Data Availability: The data used to support the findings of this study are available from the corresponding author upon reasonable request.

Funding: No funding sources

Conflict of Interest: None declared

Ethical Approval: The ethical approval for this research was obtained from the Research and Ethics Committee of the Faculty of Health Sciences and Technology, College of Health Sciences, Nnewi Campus, with protocol number: FHST/REC/023/567.

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