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Impact of Chronic Polyurethane Paint Aerosol Exposure on Respiratory impairment, Inflammatory makers, and Hemodynamic Parameters among Spray painters in Calabar Municipality

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Abstract

Context: Polyurethane paint is a reactive polymer made from polyol resin and isocyanates to produce a durable and resistant coating.
Objective: This study assessed the impact of chronic polyurethane paint aerosols on respiratory impairment, inflammatory markers, and hemodynamic parameters among spray painters in Calabar Municipality.
Materials and Method: A total of one hundred and forty six (n=146) eligible male participants, aged 20-40 yrs were recruited, and categorized into two groups of 73 participants per group; group I include Spray painters and group II comprised of Control subjects (n=73). Demographic data were obtained from all participants. Pulmonary function test was performed using a spirometer, lung parameters assessed include; Forced Vital Capacity [FVC], Forced Expiratory Volume in 1 second [FEV1], percentage of Forced Expiratory Volume in 1 [FEV1%], and Peak Expiratory Flow [PEF]). Hemodynamic indices was recorded using standard clinical instruments. Venous blood samples were analyzed for C-reactive protein and Interleukin-6 levels using ELISA kits.
Result: Group I showed significantly($P<0.001$) reduced lung indices (FVC, FEV1 and PEF), suggesting presence of respiratory impairment. There was significant ($P<0.001$) increase in C-rP and IL-6 levels among the spray painters compared to control participants, indicating state of chronic systemic inflammation. Also, hemodynamic indices specifically systolic and diastolic pressures were significantly ($P<0.001$) higher in spray painters, indicating elevated cardiovascular stress.
Conclusion: Chronic occupational exposure to polyurethane paint aerosols is strongly linked to heightened inflammatory responses, diminished lung function, and elevated blood pressure. These findings highlight the need for improved occupational safety standard and periodic health screening for workers in the painting industry.

Key words: Spray painters, Polyurethane aerosols, Hemodynamic parameters, Inflammatory markers, Lung parameters.

Introduction

Spray painters are frequently exposed to isocyanates and volatile organic compounds(VOCs) found in acrylic polyurethane aerosols[2]. While the acute effects like asthma and rhinitis are known, the link between chronic inhalation, progressive pulmonary decline, and systemic inflammation requires more investigation. In various industrial sectors, including automotive repair, shipbuilding, and

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construction, spray painters frequently utilize a different array of chemical agents such as solvent-based paints, epoxy resins, zinc-rich primers, and enamels[17]. While these coatings are essential for providing durability and aesthetic finishes to metallic and wooden surfaces, their application creates a high-risk environment for chemical inhalation[3].

This study investigates polyurethane paints and its associated solvents, which are predominantly utilized in automotive refinishing. Synthesized through a polyaddition reaction between diisocyanates and polyols. Polyurethane coatings have gained extensive industrial prominence due to their durability. However, a critical distinction exists between application methods. A research by Huang[7] indicates that solid flexible polyurethane foam (FPF) used in upholstery is relatively inert with minimal health risks with the aerosolized liquid form used in spray painting poses severe respiratory hazards due to the presence of reactive isocyanate monomers. While the solid form of flexible polyurethane foam (FPF) contributes primarily to environmental degradation through landfill accumulation and the subsequent contamination of soil and water[16], the liquid phase of the polymer presents a more immediate clinical risk. The liquid polyurethane is extensively used to protect metallic and wooden surfaces from oxidation[11]. Furthermore, the release of volatile organic compounds (VOCs), such as toluene and xylene, significantly compromises ambient air quality[18]. To enhance durability, manufacturers have integrated acrylic polyacrylates to develop acrylic polyurethane hybrids, a transition that increases the paint's resilience but simultaneously complicates the toxicological profile for those occupationally exposed[23]. Acrylic polyurethane coatings are highly valued in the automotive and fabrication industries due to its superior resistance to abrasion, chemical corrosion, moisture, and ultraviolet (UV) degradation[20,17]. Recent advancements like the integration of 'bulk gel' additives, have further enhanced these coatings by providing high-gloss finishes and 'self-cleaning' hydrophobic properties. This allows surfaces to remain free of contaminants with minimal maintenance, as rainwater is sufficient to remove surface debris[5]. Consequently, these aesthetic have established acrylic polyurethanes as the preferred medium for modern industrial applications[23].

The global economic footprint of acrylic polyurethane paint is expanding at a significant rate. According to Fortune Business Insights[22], the market is valued at $55.58 billion in 2024 and is projected to reach $58.25 billion by 2025, with a forecasted surge to $81.06 billion by 2032. This geometric growth is propelled by escalating demand within the automotive, aerospace, and general manufacturing sectors[22]. Such rapid industrial proliferation underscores the urgent need to address the occupational health risks associated with the increasing number of workers involved in its application.

Recent advancements in coating technology has led to the development of water-soluble acrylic bases which are associated with a reduction in cardiopulmonary complications compared to traditional solvent-based systems[2,26]. Despite these improvements, the primary routes of toxicological entry remain the respiratory tract, dermal contact, and ocular exposure. While skin contact is a documented precursor to contact dermatitis and various hypersensitivity reactions[4], acute inhalation or accidental ingestion can trigger significant respiratory distress. These clinical manifestations typically include persistent coughing, wheezing, dyspnea, and ocular inflammation[3].

Exposure to acrylic polyurethane aerosols triggers a multi-systemic toxicological response. Neurologically, acute exposure manifests as dizziness, cephalalgia (headache), and somnolence[14]. The World Health Organization[15] corroborates these findings, noting that chronic exposure causes adverse central nervous system effects as well, simultaneously escalating pulmonary inflammation, which may progress to bronchoconstriction and irreversible fibrosis. Nevertheless, the diisocyanates and volatile organic compounds (VOCs) within these paints act as potent irritants; upon reaching an effective biological dose, they induce pulmonary oxidative stress, driving the inflammatory cascade and subsequent respiratory dysfunction[12]. Beyond the lungs, VOCs have been shown to disrupt physiological indices, notably elevating heart rate, blood pressure, and total cardiac output[8].

Occupational exposure to acrylic polyurethane

aerosols presents a psychological distress and the presence of benzene and formaldehyde significantly elevates the risk of lung cancer[10]. The inhalation of atomized particles is also associated with depressive effects, manifesting as physical pain and emotional instability[3]. Addressing these risks requires rigorous objective monitoring. Consequently,spirometry is utilized as the premier tool for evaluating pulmonary health[37,36]. By analyzing the FEV1/FVCratio and other vital parameters, this will aid detect early-stage respiratory dysfunction and the prevention of chronic obstructive disorders[38].

Evidence suggests that the inorganic components in acrylic polyurethane paint aerosols act as systemic sensitizers, negatively impacting both cardiovascular stability and pulmonary efficiency[39]. These effects are characterized by fluctuations in systemic cytokine concentrations and a decline in forced (lung) volumes. Consequently, the present study investigated the respiratory and systemic health of occupationally exposed automotive spray painters. The objective is to quantify the degree of pulmonary impairment and to establish whether specific exposure patterns correlate with a heightened risk of developing occupational lung disease.

Materials and Method

Study location and Sample size Determination: This study was conducted in Calabar Municipality, Cross River State, located in Southeastern Nigeria (4.9570° N, 8.3140° E; elevation ~57m). Using a cohort design, the study identified an active population of 100 spray painters. From this group, a sample size of 73 participants was determined using the Slovin (1960) formula at a 95% confidence level.

Materials:

Instruments, Measurement of Anthropometric and Physiological Parameters

Serum levels of Interleukin-6 (IL-6) and C-reactive protein (CRP) were determined using Enzyme-Linked Immunosorbent Assay (ELISA) kits (Product IDs: EK006-MMG12 and SL0535Hu, respectively), sourced from Sunlong Biotech Co., Ltd (Zhejiang, China). Respiratory parameters were measured using a Vitalograph Alpha Touch 6000 spirometer (Vitalograph Ltd, UK). Peripheral oxygen saturation (SPO_2) was monitored via an LK89 finger pulse oximeter (China). Cardiovascular indices were assessed using a Bokang sphygmomanometer (Model, BK 1018, China) and a professional-grade stethoscope (Model, BK3008, China)[24] . Anthropometric data were collected using a calibrated weighing scale and a vertical stadiometer (long meter ruler) with structured research questionnaire[40]. Blood samples were collected using sterile syringes, needles, and plain specimen bottles, then processed via centrifugation. Procedural safety and sample integrity were maintained through the use of disposable gloves, face masks, and biohazard specimen transport bags.

Study Participants

The study comprised young adult males (N=73), aged 20–40 years, currently employed as Spray painters were consecutively recruited from different automotive workshops within the study area. Inclusion was limited to individuals with documented chronic occupational exposure to aerosolized acrylic polyurethane during routine spray-painting activities. All participants underwent a baseline medical screening to ensure they were clinically stable and free from pre-existing chronic respiratory infections at the time of enrolment.

For comparative analysis, a control group (N=73) of healthy, age-matched male participants with similar anthropometric profiles (body weight and height) was established. These control participants were recruited from the University of Calabar using simple randomized method; without prior history of occupational contact with industrial inhalants, gases, or hazardous chemical aerosols, and met the criteria for 'zero-exposure'.

Ethical approval

Ethical consent was obtained from the State Ministry of Health, Calabar. Health Research Ethics Committee (HREC) Calabar, with the registration number: CRSMOH/RP/REC/2025/584.

Study Design

A cohort study design was used to achieve the objectives of this study. A total of 146 male participants (n= 146) were recruited for this study

using structured questionnaires[25] to determine eligibility based on specific inclusion and exclusion criteria. Demographic data were rigorously screened to ensure that only eligible candidates proceeded to the clinical phase. The cohort was divided into two equal groups: Group I (Test subjects, n=73), consist of occupationally exposed spray painters, and Group II (Control participants, n=73), comprise of unexposed participants. The Physiological parameters of both groups were assessed respectively. Pulmonary Function Tests (PFT) was conducted using a spirometer (Vitalograph Alpha Touch 6000) to determine lung volumes and capacities. Concurrently, systemic inflammatory markers were quantified using ELISA kits for biochemical analysis. Hemodynamic and respiratory status were monitored using a sphygmomanometer and stethoscope for blood pressure, and a pulse oximeter was used to measure peripheral oxygen saturation (SPO2) and heart rate.

Study Procedure:

Evaluation of Pulmonary Function in Spray painters and Control participants.

A spirometer (Vitalograph Alpha touch 6000, Uk) was used to perform pulmonary function test. Each participant was given instruction and a demonstration of the breathing maneuver prior to testing. Adhering to American Thoracic Society (ATS) criteria, 2005 and updated in 2019, a minimum of three acceptable maneuvers was required for each participant. For participants who demonstrated poor technique or sub-optimal effort, up to five additional maneuvers were performed to achieve reproducibility. The three highest values from technically acceptable maneuvers were recorded and used for pulmonary evaluation. The measured parameters (spirometric); including FVC, FEV1, PEF and FEV1% were compared against the predicted reference values to distinguished the respiratory patterns of both the spray painters and control subjects.

Specimen collection/Measurement of inflammatory biomarker levels

Blood samples were obtained from both spray painters and control group in the early hours(6:30am-7am) of each day into5ml plain specimen bottle, using standard venipuncture techniques[27], and allowed to clot. The specimens were centrifuge at 1000rpm for 5mins to separate the serum from blood cells. Reagent (ELISA kits) were allowed to warm to room temperature (20-30 0c) before use. All spray painters sat on a comfortable place within their workshop prior to sample collection.

The concentration of IL-6 in the serum was determined using commercially available ELISA kits (MG12, Sunlong Biotech, China). was performed according to the procedure stated by the manufacturer and the method as described by[30].

The concentration of C- reactive protein in the serum was determined using commercially available ELISA kits (SL0535 Hu, Sunlong Biotech, China) with reference to method as described by Tillet and Francis[32] and as used by Ridker[13].

Determination of Blood Pressure, BPM and SPO_2 (hemodynamic indices) in Spray painters and Control subjects.

Systemic blood pressure was evaluated daily in the early hours (07:00 to 08:30 hours) to mitigate the influence of diurnal variations. Measurements were obtained via the auscultatory method, using a calibrated Bokang sphygmomanometer and a professional stethoscope. The clinical protocol followed a standardized diagnostic procedures and the method as used by Beevers[24], accurate recording of both systolic and diastolic pressures commenced prior daily activities.

Statistical Analysis

All experimental data obtained was expressed as mean ± standard error of mean (SEM). The data were compared using Student's T-test to determine the differences between the mean and probability level of $P< 0.05$ was considered statistically significant. Pearson correlation coefficient (r) was used to determine linear direction between spirometric indices and level of exposure.

Results

Demographic parameters of spray painters and Control subjects

In this study, the results were compared between Spray painters and Control subjects and presented as mean ± Standard error of mean (SEM). The result

of the mean values of demographic parameters between spray painters and control participants were presented in Table I below.

Table 1: Shows comparison of demographic parameters between Spray painters and Control participants

Demographic Indices	Spray painters	Control group	P-value
Age	29.41±0.60	28.52±0.59	0.103
Weight	50.62±0.42	51.63±0.38	0.420
Height	1.67±0.00	1.68±0.00	0.206
BMI	23.86±0.17	23.50±0.18	0.150

Values are expressed as mean + SEM, n=73
No significant differences between group

Hemodynamic indices of spray painters and Control participants

The result of the mean values of hemodynamic indices(Systolic-SBP, diastolic- DBP, mean arterial pressure- MAP, beat per minute- BPM, oxygen saturation-SPO_2) of spray painters and Control participants were presented in Table 2 below.

Table 2: Shows comparison of hemodynamic indices between spray painters and control participants

Blood pressure Indices	Spray painters	Control group	*P-value*
Systolic BP	128.12±0.81**	121.82±0.52	0.000
Diastolic BP	83.65±0.36 *	81.09±0.24	0.001
Mean arterial pressure	97.80±0.44**	94.82±0.28	0.000
BPM	78.76±1.00 **	71.97±0.98	0.000
SPO_2	95.47±0.32	96.82±0.26	0.118

Values are expressed as mean + SEM, n=73
**significantly different from control at P<0.05 Vs Control

Spirometric indices of spray Painters and Control participants

The result of the mean values of lung indices including; forced vital capacity (FVC), forced expiratory volume in one second (FEV1), peak expiratory flow(PEF), forced expiratory volume in one second expressed as percentage of FVC (FEV1%) in spray painters and Control participants were presented in Table 3 below.

Table 3: Shows comparison of spirometric indices between spray painters and control participants.

Spirometric indices	Spray painters	Control group	*P-value*
FVC	3.00±0.04**	3.45±0.07	0.000
FEV_1	2.12±0.06*	2.42±0.07	0.001
PEF	212.29±1.79**	239.47±4.50	0.000
FEV_1%	69.47±1.60	70.97±1.95	0.842

Values are expressed as mean + SEM, n=73
**significantly different from control at P<0.05 Vs control

Inflammatory biomarkers levels in Spray painters and Control subjects.

The result of the mean values of C-reactive peptide (C-rP) and interleukin-6 (IL-6) levels in Spray painters and Control participants were presented in Table 4 below.

Table 4: Shows comparison of inflammatory biomarkers levels between spray painters and control participants.

Inflammatory biomarkers	Spray painters	Control group	*P-value*
C-rP	1.29±0.04*	0.66±0.03	0.000
IL-6	0.57±0.02	0.50±0.02	0.021

Values are expressed as mean + SEM, n=73
**significantly different from control at P<0.05 Vs Control

Fig 1-4 Shows correlation of duration of exposure in spray painters.

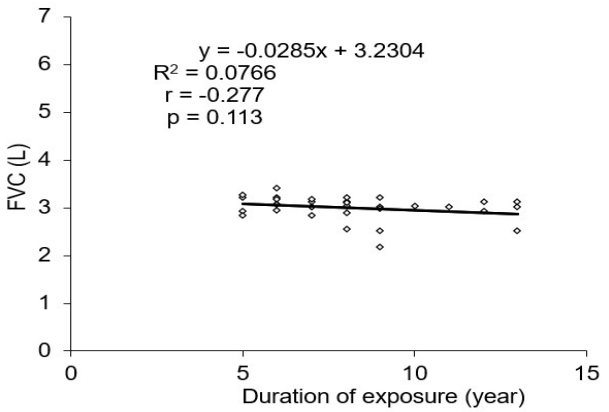

Figure 1: Correlation of forced vital capacity with duration of exposure indicates slow negative slope and represent a gradual decline in FVC among the spray painters

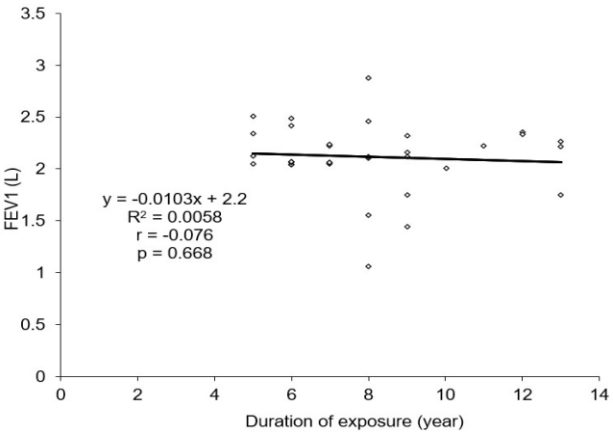

Figure 2: Correlation of forced expiratory volume in one sec. with duration of exposure indicates gradual decline in FEV1 over time.

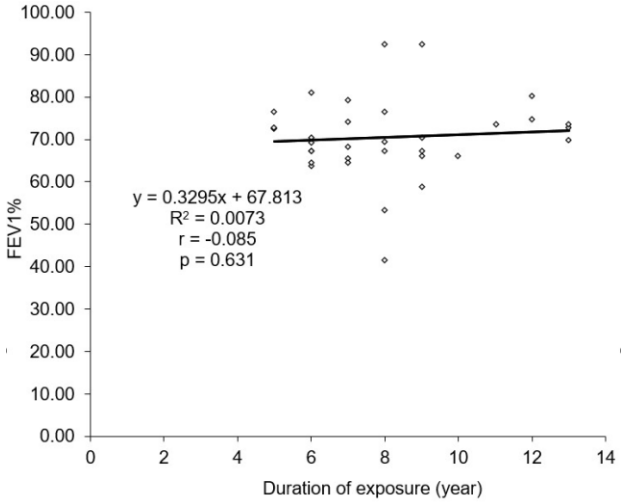

Figure 3: Correlation of FEV1% with exposure to paint aerosols showed slow downward slope (negative) and indicates gradual decline in FEV1% over time.

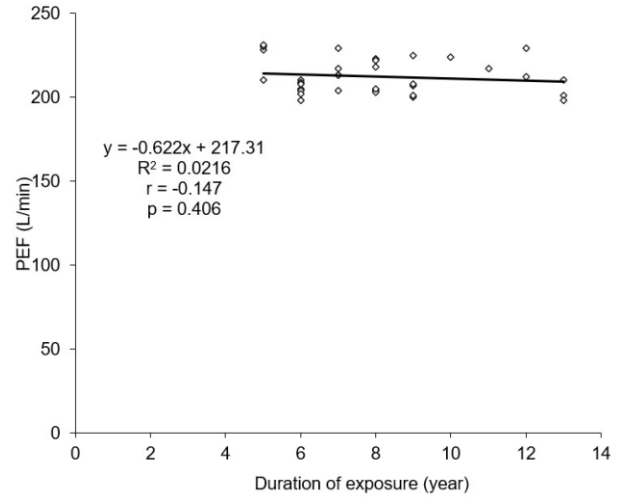

Figure 4: Correlation of peak expiratory flow (PEF) with exposure showed a slow negative slope and indicates reduction in PEFas exposure time increases over time in the spray painters

Discussion

Occupational spray painting creates a hazardous environment characterized by dense concentrations of aerosolized particulates and isocyanates[7]. These substances act as primary respiratory sensitizers, inducing localized allergic reactions and mucosal irritation[1,6]. Study indicated that prolonged inhalation of these inorganic solvent-based aerosols results in significant clinical morbidity; specifically, the constant irritation of the bronchial lining is a known precursor to the development of chronic bronchitis and other obstructive airway disorders[8]. According to epidemiological data, VOCs are directly implicated in the pathogenesis of occupational bronchitis. Research suggests that the inhalation of these solvents leads to persistent airway inflammation, coupled with their broader toxicological profile, significantly compromises the general health outcomes of workers in the painting industry 9. From the result above, demographic parameters showed no significant difference ($P<0.05$) between the spray painters and control participants. The result also showed no significant reduction in the pulmonary function indices of the control subjects, confirming a stable respiratory baseline in the absence of occupational aerosol exposure. Spray painters showed significant decrease ($P<0.05$) in spirometric values indicating airway resistance and compromised lung function. These findings aligned with the study by Utionkpan et al[31], which concluded that pulmonary toxicity manifests as localized inflammation within the respiratory tract. Such inflammatory responses are precursors to significant reductions in spirometric volumes, ultimately compromising overall pulmonary efficiency. A study by Utionkpan et al[31], opined that inflammatory cascade triggers the release of histamine, a potent chemical mediator that induces tension within the airway smooth muscles. This sustained contraction results in significant bronchoconstriction, which is clinically reflected as an abnormal decline in main spirometric parameters, specifically FEV1, FVC, PEFR, and the FEV1%. The observed reductions in FVC, FEV1 and PEF, within the spray painter indicate a profound physiological compromise resulting from chronic inhalation of acrylic polyurethane mists. These findings suggest that persistent occupational exposure serves as a primary driver for respiratory impairment, significantly increasing the subjects' susceptibility to obstructive pulmonary disorders, such as occupational asthma and chronic bronchitis. While the FEV1% ratio (FEV1/FVC ratio) of the spray painters exhibited only a marginal decrease, relative to the control group; correlation analysis revealed a significant trend. Specifically, the duration of occupational exposure to acrylic polyurethane aerosols demonstrated a strong

negative correlation with all measured spirometric indices. This suggests that as the timeframe of chronic inhalation increases, there is a steady and significant decline in lung volumes (FVC, FEV1, PEF).

This confirmed that consistent breathing of air containing polyurethane mists gradually decline pulmonary function. Evidence found in health literatures implicated particulate matters and volatile organic aerosols to cause damage to the walls of the respiratory tree which makes the walls inflamed and potentiate histamine secretion[35].

The significant ($P<0.05$) reductions in FEV1 and FVC as observed in the experimental group indicate a decrease in bronchial diameter and respiratory muscle strain, likely exacerbated by the physical demands of daily occupational tasks. The decline correlates with the demographic information obtained, which revealed that spray painters had been active in the profession for over ten years. The findings points to the cumulative impact of long-term exposure to polyurethane aerosols.

This analysis revealed a significant elevation ($P<0.05$) in serum c-reactive protein (C-rP) levels among the spray painter compared to control participants, indicating a state of chronic systemic inflammation. Interleukin-6 (IL-6) levels was non-significant ($P<0.05$) among the spray painters. As a multifaceted cytokine, IL-6 plays a dual role in immune regulation; during the acute inflammatory phase it stimulates hepatic synthesis of acute-phase reactants, such as C-rP[33]. While IL-6 is involved in the induction of M2-like macrophages to facilitate inflammatory resolution; the statistically insignificant levels observed in this study suggest that aerosol exposure caused a non-sensitized immune provocation. The C-reactive protein (C-rP) functions as a primary acute-phase reactant, characterized by a rapid elevation in concentration following systemic inflammation, infection, or localized tissue injury. As a critical component of the innate immune system, C-rP facilitates the clearance of cellular debris by binding to phosphocholine and subsequently activating the classical complement pathway[34]. In the present study, the significantly elevated serum concentrations of both IL-6 and CRP in spray painters as compared to the control group, provide biochemical evidence of a sustained systemic inflammatory response triggered by chronic aerosol exposure.

All measured hemodynamic parameters in this study revealed significant increase ($P<0.05$) among the spray painters compared to control participants. This includes marked increase in systolic (SBP), diastolic (DBP), mean arterial pressure (MAP), and resting pulse rate(BPM), while oxygen saturation was statistically non-significant ($P<0.05$). These findings corroborate the research by Hwang[19], which suggests that individuals chronically exposed to volatile organic compounds (VOCs) exhibit elevated blood pressure with significantly higher risk of developing clinical hypertension.

Inhalation of chemical aerosols causes significant autonomic dysregulation, disrupting the neural control mechanisms that govern cardiac function. Research by Damiati et al[29] opined that such exposure leads to a marked reduction in heart rate variability (HRV), a critical independent risk factor for cardiovascular mortality. This autonomic interference is further manifested by compensatory increase in both resting heart rate and systemic blood pressure, contributing to long-term hemodynamic instability in exposed participants.

Also, ultrafine particulates are inherent in atomized polyurethane and implicated in direct cardiotoxicity. These agents undergo systemic translocation from the pulmonary alveolar into the bloodstream, where they directly interact with myocardial tissue, this interaction results in cellular injury and impaired myocardial contractility, thereby cause elevations in systemic blood pressure[28].

In conclusion, this study demonstrated that chronic occupational exposure to acrylic polyurethane aerosols and its constituent particulates exerts a significant detrimental impact on both cardiovascular and pulmonary systems. It is evident from this study that elevated resting heart rates is also implicated in the disruption of cardiovascular function, characterized by systemic hypertension. Simultaneously, pulmonary health is compromised as evidenced by a marked reduction in spirometric volumes and increase systemic inflammation among spray painters. This finding revealed a high risk for the development of obstructive pulmonary diseases and idiopathic hypertension. To mitigate

these risks It is essential to use protective gear like nose masks, and work in well-ventilated chambers to minimize inhalation to safeguard respiratory health.

Conflict of interest
The authors declared no conflicts of interest.

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Authors' Contributions
Dr. Utionkpan, L.P -Designed the concept of the study and Manuscript preparation.
Ushuple C. L - Statistical analysis and Manuscript editing.
Usang S. E - Data acquisition.

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