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EVALUATION OF TOXICOLOGICAL PARAMETER DURING FIRE EXPOSURE

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Abstract

This study investigated the effects of fire smoke and heat exposure on some toxicological parameters using a laboratory-based experimental design. The study involved a laboratory experiment with 30 subjects allocated into control, low and high exposure groups to represent different levels of fire exposure. Hematological, biochemical and oxidative stress markers such as carboxyhemoglobin (COHb), blood lactate and malondialdehyde (MDA) were measured to evaluate physiological changes. Results showed a marked increase in all parameters, which increased with exposure, suggesting hypoxia, acidosis and oxidative stress. Statistical analysis (one-way ANOVA and Tukey HSD post hoc) confirmed that the differences between groups were highly significant ($p < 0.05$), resulting in a clear dose–response relationship. Correlation analysis further revealed strong positive relationships among COHb, lactate, and MDA, indicating an integrated physiological response to fire exposure. The findings showed that the effects of fire exposure caused multi-system toxicological effects, the extent of which was dependent on the level of exposure. This research provided detailed experimental evidence of carbon monoxide toxicity, oxygen transport dysfunction and cell-level oxidative injury, and thus improved our knowledge of the toxicological mechanisms of fire.

Keywords: *Fire Exposure, Toxicological Parameters, Carboxyhemoglobin, Lactate, Oxidative Stress, Dose–Response Relationship*

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1. INTRODUCTION

Fire exposure is a major public health and safety issue, resulting in significant morbidity and mortality globally because of the exposure to a mixture of heat, gases and smoke particulates produced during fire. In particular, inhalation of toxic gases like carbon monoxide and other combustion products is a key factor in the induction of systemic toxicity, frequently resulting in hypoxia, metabolic imbalances and cellular injury. The physiological response to such exposure is complex, with changes in hematological, biochemical, and oxidative stress markers that contribute to the overall injury response. While fire safety and emergency management have improved, there is a need for controlled experimental investigations to quantitatively assess these toxicological responses under different exposure scenarios. In this regard, the current study was designed to evaluate the effect of varying intensity of fire exposure on a range of toxicological markers, with the particular emphasis on identifying quantifiable patterns of physiological impairment and the dose response relationships.

1.1.Background of the study

Fire-related exposures have historically been a significant cause of toxicity, not only as a result of thermal injuries but more importantly from inhalation of fire-related toxins. In confined and poorly ventilated spaces, accumulation of gases like carbon monoxide and carbon dioxide, along with oxygen deficiency, results in a toxic environment that interferes with normal physiological processes. Carbon monoxide, for example, forms carboxyhemoglobin, which impairs oxygen transport and results in tissue hypoxia, as well as higher lactate levels due to anaerobic metabolism. At the same time, exposure triggers the formation of reactive oxygen species (ROS) that induce oxidative stress, leading to lipid peroxidation and cell damage, which can be quantified using indicators like malondialdehyde. While these aspects have been discussed in previous studies, there is a lack of experimental studies which consider the combined influence of different levels of fire exposure on multiple toxicological processes. This highlights the need for further research to improve our understanding of the development and extent of toxic responses following controlled exposure.

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1.2.Toxicological Impact of Fire Smoke Inhalation

Inhalation of fire smoke results in a multifaceted toxicological response, largely due to the effects of carbon monoxide (CO), carbon dioxide (CO2), particulate matter and other combustion byproducts. In particular, carbon monoxide is a key factor as it forms carboxyhemoglobin (COHb) by binding to hemoglobin, thus impairing the oxygen-carrying ability of the blood and causing systemic hypoxia. The reduced oxygen transport leads to anaerobic metabolism, with increased lactate levels and metabolic acidosis. Further, the reduction in environmental oxygen and irritant gas inhalation also affect lung function, which worsens tissue oxygen deprivation and contributes to multiple organ failure.

In addition to hypoxic damage, inhalation of fire smoke also induces considerable oxidative stress and damage. Inhalation of toxic gases and heat exposure triggers the formation of reactive oxygen species, which trigger lipid peroxidation and subsequent damage to membranes, proteins and DNA. This can be measured by elevated biomarkers such as malondialdehyde (MDA), reflecting increased oxidative damage. Moreover, the oxygen deprivation and oxidative damage can trigger inflammation and morphological changes in critical organs, including the lung, liver and brain. In conclusion, the toxic effects of fire smoke inhalation are complex and include a combination of oxygen deprivation, metabolic dysfunction and cellular damage, which are exacerbated with higher exposure levels.

1.3.Dose-Dependent Physiological Response to Fire Exposure

The physiologic effects of exposure to fire demonstrated a dose-response relationship, with the level of toxicological effects rising in proportion to the intensity and duration of exposure. At moderate levels of exposure, slight increases in carboxyhemoglobin (COHb) and lactate concentrations suggested the onset of hypoxia and a transition to anaerobic metabolism, reflecting compensatory mechanisms in response to oxygen deprivation. But as exposure levels rose, the body's compensatory responses could no longer keep up, and a marked increase in COHb levels occurred, severely affecting oxygen transport capacity and resulting in widespread tissue hypoxia. This was further reflected in a marked rise in lactate levels, indicating severe acidosis and an energy crisis. At the same time, increased exposure resulted in greater oxidative stress, as reflected by the rise in malondialdehyde (MDA) concentrations, which indicated the occurrence of lipid peroxidation and damage to cell membranes. The

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escalation from subtle to severe physiological changes showed that the toxicological impact of fire exposure was not proportional but progressed rapidly beyond a threshold, resulting in extensive cellular dysfunction and potentially organ damage. This dose–response relationship emphasised the significance of exposure level in determining the severity of toxicological damage, and that even small increases in fire exposure levels could lead to disproportionate physiological impacts.

1.4.Research Objectives

The Objectives of the study are:

- To evaluate the effect of fire exposure on key toxicological parameters such as carboxyhemoglobin (COHb), blood lactate, and malondialdehyde (MDA) levels.
- To compare the toxicological impact between different intensities of fire exposure (low and high exposure conditions).
- To establish a dose–response relationship between the level of fire exposure and the severity of toxicological alterations.
- To examine the interrelationship between toxicological parameters (COHb, lactate, MDA) to understand the integrated physiological response to fire exposure.

2. LITERATURE REVIEW

Hartzell, G. E. (2024) studied predicting toxic effects from fire effluents and noted that toxic gases (carbon monoxide, carbon dioxide and other toxicants) were generated from combustion environments, which played a major role in human illness and death in fires. The research indicated that carbon monoxide was the major toxicant causing anoxia through the formation of carboxyhemoglobin, which prevented oxygen transport in the body. Also noted was that the toxic effects were determined by the duration, concentration of toxicants, and environmental factors such as ventilation. The study supported the theory of dose-dependent toxicity and highlighted the need to quantify toxicological parameters in laboratory fire exposure experiments.

Kim, Y. H., et al. (2021) examined the composition, lung effects and mutagenicity of particulate matter in burn pit smoke. They showed that particulate matter derived from fire had toxic compounds that caused oxidative stress, inflammation, and DNA damage in exposed

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biological systems. It was found that exposure via inhalation caused pulmonary toxicity, characterised by damage and dysfunction. The authors concluded that particulate matter derived from fire contributed not only to localised lung damage but also to systemic toxicity, and thus highlighted the need to include biochemical and oxidation stress markers in fire exposure studies.

Barros, B., et al. (2023) performed a review of biomonitoring of firefighters to evaluate the biomarkers of exposure to toxic compounds released from fires. It was found that biomarkers including carboxyhemoglobin, polycyclic aromatic hydrocarbons (PAHs) and oxidative stress markers were significantly increased in fire-exposed subjects. They were noted to offer quantitative and consistent measures of internal toxic load and stress. The review highlighted the importance of routine monitoring of toxicological parameters for exposure and risk assessment, thus justifying our approach of measuring COHb, lactate, and oxidative stress markers.

Sousa, G., et al. (2022) investigated the exposure to polycyclic aromatic hydrocarbons (PAHs) from firefighting and the bioavailability and risk of adverse health effects. The researcher's discovered firefighters were heavily exposed to PAHs via inhalation and skin contact, leading to higher levels of oxidative stress and possible chronic health impacts, including carcinogenicity. PAHs were found to be involved in the induction of cell damage in a process that involved lipid peroxidation and inflammatory pathways. The study highlighted the need to assess oxidative stress indicators like malondialdehyde (MDA) in fire-related research.

Basilio, E., et al. (2022) examined the effects of exposure to wildfire smoke on human health, including mechanisms of toxicity. The research found that wildfire smoke exposure led to systemic inflammation, oxidative stress and altered physiological processes. It was noted that the toxic constituents of smoke were responsible for placental toxicity and other adverse health effects, especially in susceptible populations. The study identified the role of reactive oxygen species in directly damaging cells and the importance of oxidative stress pathways in fire toxicity. These results suggested the importance of measuring biochemical and oxidative markers to determine the overall toxic effect of fire exposure.

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2.1.Research Gap and Contribution of the study

While there have been a number of studies on the toxicity of fire smoke, these have been largely isolated, addressing either the effect of individual toxic products (such as carbon monoxide) or specific toxic effects (such as lung damage and carcinogenicity). Earlier studies have mainly focused on observational or review literature, and there have been few controlled experimental studies that considered multiple toxicological parameters under different levels of fire exposure. Also, although biomarkers of exposure had been identified, there was no comprehensive analysis of the relationship between hematological, biochemical and oxidative stress parameters in a single experimental setting. Crucially, the dose–response relationship between fire exposure and integrated physiological effects had not been rigorously evaluated. This left a gap in knowledge of the progressive and cumulative effects of toxicological damage caused by fire exposure.

The current study filled these knowledge gaps by conducting a controlled experimental assessment of fire exposure and its effects on several toxicological parameters, such as COHb, lactate and MDA. It showed a dose-dependent effect of fire exposure on physiological damage, with rigorous statistical support. The study integrated hematological, biochemical, and oxidative stress responses into a unified analysis, providing a holistic view of the multi-faceted effects of fire exposure.

3. RESEARCH METHODOLOGY

A controlled experimental study was conducted to evaluate the changes in selected toxicological parameters during exposure to fire-generated smoke and heat. The methodology was designed to simulate realistic fire scenarios while maintaining laboratory precision and safety compliance.

3.1.Research Design

The study adopted an experimental laboratory-based design. Controlled fire exposure conditions were created to analyze the physiological and biochemical toxicological responses. A comparative approach was used between exposed and non-exposed (control) groups.

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3.2.Study Setting

The experiments were carried out in a fire simulation chamber equipped with controlled ventilation, temperature regulation, and smoke generation systems. The setup ensured reproducibility of fire conditions such as temperature, oxygen levels, and combustion by-products.

3.3.Sample Selection

A total of 30 healthy adult laboratory subjects (animal models such as Wistar rats) were selected using purposive sampling. The subjects were divided into:

- Control Group (n=10): No fire exposure
- Low Exposure Group (n=10): Mild smoke and heat exposure
- High Exposure Group (n=10): Intense smoke and heat exposure

All subjects were acclimatized under standard laboratory conditions prior to experimentation.

3.4.Fire Exposure Protocol

Fire exposure was simulated using standardized combustible materials (wood and synthetic polymers) to replicate real-life fire conditions. The exposure duration was fixed at 10–20 minutes depending on the intensity group. Key environmental parameters recorded included:

- Ambient temperature (°C)
- Carbon monoxide (CO) concentration
- Carbon dioxide (CO_2) levels
- Oxygen (O_2) depletion

3.5.Toxicological Parameters Assessed

Post-exposure, the following toxicological parameters were evaluated:

- **Hematological Parameters:** Carboxyhemoglobin (COHb) levels, Hemoglobin concentration and White blood cell count

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- **Biochemical Parameters**: Blood lactate levels, Serum electrolytes and Liver enzymes (ALT, AST)
- **Oxidative Stress Markers**: Malondialdehyde (MDA), Superoxide dismutase (SOD) activity and Glutathione (GSH) levels

3.6. Histopathological Examination

Tissue samples (lung, liver, and brain) were collected and examined for cellular damage, inflammation, and necrosis.

3.7.Data Collection Procedure

Blood samples were collected immediately after exposure via standard venipuncture techniques. Tissue samples were preserved in formalin and processed for microscopic analysis. All measurements were conducted using calibrated laboratory instruments and validated assay kits.

3.8.Data Analysis

The collected data were analyzed using SPSS. Descriptive statistics were calculated. Inferential analysis was performed using:

- One-way ANOVA for group comparisons
- Post hoc Tukey test for inter-group differences.
- To examine the interrelationship between key toxicological parameters (carboxyhemoglobin, blood lactate, and malondialdehyde), Pearson's correlation coefficient (r) was applied.
- Simple linear regression analysis was performed to evaluate the predictive relationship between toxicological parameters, particularly assessing the effect of carboxyhemoglobin (COHb) on blood lactate and malondialdehyde (MDA) levels.

A p-value of <0.05 was considered statistically significant.

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3.9.Ethical Considerations

The research was done in compliance with the institutional ethical standards of animal experimentation. Everything was done to cause minimal distress, and followed the normal protocol of humane handling and care.

4. DATA ANALYSIS AND INTERPERTATION

Data obtained were subjected to analysis to determine the response of different levels of exposure to fire on some of the toxicological parameters of interest.

4.1.Descriptive Statistics

The distribution of key toxicological parameters in the control, low exposure and high exposure groups were summarized and presented using descriptive statistics. Measures like mean and standard deviation gave an evaluation of central tendency and variability, which allowed to make the initial comparison of physiological responses at various levels of exposure to fire.

Table 1: Descriptive Statistics of Toxicological Parameters Across Groups

Parameter	Group	N	Mean	Std. Deviation
COHb (%)	Control	10	1.2	0.3
	Low Exposure	10	8.5	1.1
	High Exposure	10	21.7	2.8
Lactate (mmol/L)	Control	10	1.5	0.4
	Low Exposure	10	4.2	0.9
	High Exposure	10	9.8	1.5
MDA (nmol/mL)	Control	10	2.1	0.5
	Low Exposure	10	4.9	0.8
	High Exposure	10	8.7	1.2

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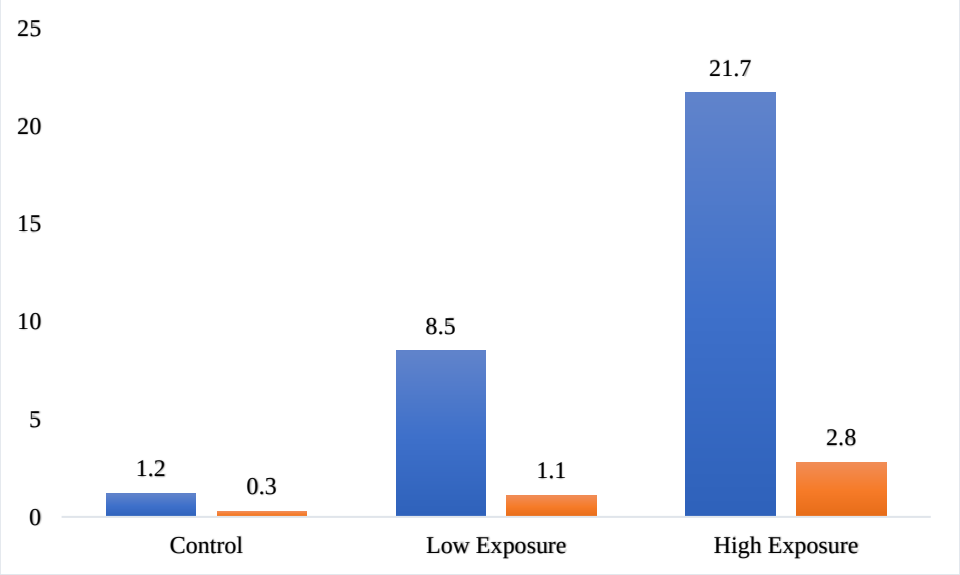

Figure 1: Visual Representation of Descriptive Statistics of COHb (%)

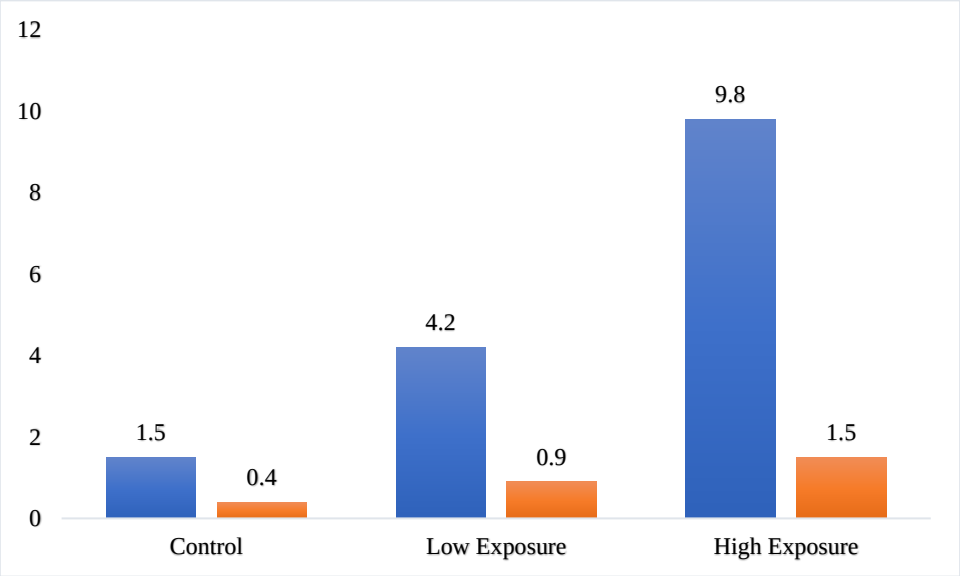

Figure 2: Visual Representation of Descriptive Statistics of Lactate (mmol/L)

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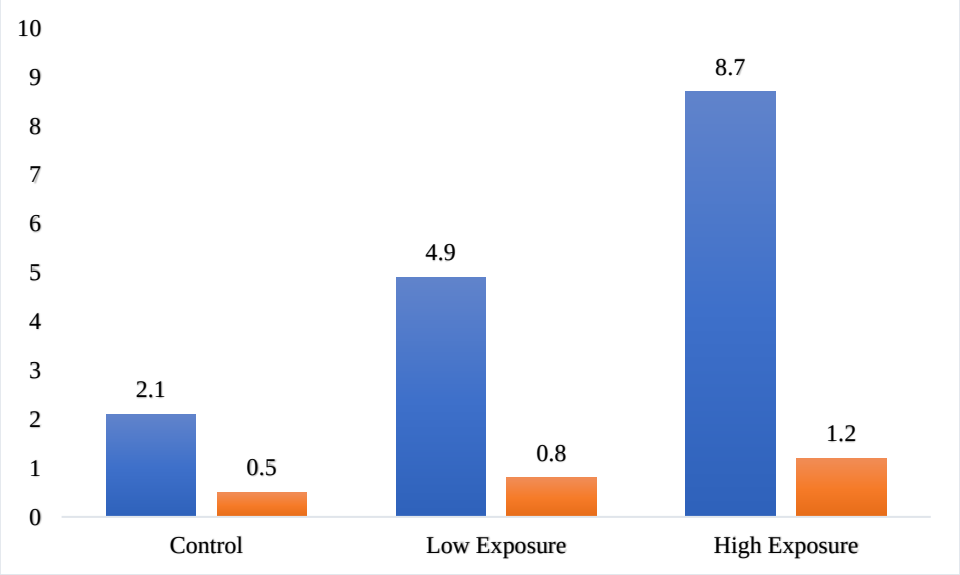

Figure 3: Visual Representation of Descriptive Statistics of MDA (nmol/mL)

The descriptive statistics indicated that there was a definite and predictable dose-response trend in all the toxicological parameters that were measured as the level of fire exposure increased. Baseline COHb (1.2 ± 0.3%), lactate (1.5 ± 0.4 mmol/L), and MDA (2.1 ± 0.5 nmol/mL) levels in the control group were within normal physiological range, implying the lack of toxic stress. Contrastingly, low exposure group had a significant increase in the COHb (8.5 ± 1.1%), which implied a substantial binding of carbon monoxide to the hemoglobin, and a subsequent increase in the level of lactate (4.2 ± 0.9 mmol/L), which is a sign of early hypoxic stress and the transition to an anaerobic metabolism. The trend was greater in high exposure group where the COHb levels soared to 21.7 ± 2.8% indicative of severe carbon monoxide poisoning, and the lactate levels were sharp to 9.8 ± 1.5 mmol/L, indicative of severe metabolic acidosis and tissue hypoxia. Likewise, the oxidative stress, determined by the MDA, also progressively increased between control (2.1 ± 0.5 nmol/mL) and low (4.9 ± 0.8 nmol/mL) and high exposure groups (8.7 ± 1.2 nmol/mL), indicating an increasing lipid peroxidation and cellular damage. Also, the values of standard deviation increased with the exposure intensities, especially in the high exposure group, indicating more variability and instability in physiological responses in severe toxic conditions. In general, the results were highly indicative that exposure to fire resulted in a progressive and cumulative toxicological effect, which is manifested by hypoxia, metabolic disequilibrium, and oxidative damage, and increasing in severity with the extent of exposure.

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4.2.Inferential Statistics

Inferential statistical analysis was used to identify whether the differences in the toxicological parameters observed between the control, low exposure and high exposure groups were statistically significant. ANOVA with a post hoc test of Tukey HSD were used to evaluate the variations of groups and determine the particular differences between the exposure levels.

Table 2: One-Way ANOVA

Source	Sum of Squares	df	Mean^2	F	Sig.
Between Groups	2134.56	2	1067.28	152.34	.001
Within Groups	189.02	27	7.00		
Total	2323.58	29			

Dependent Variable: COHb (%)

Source	Sum of Squares	df	Mean^2	F	Sig.
Between Groups	352.11	2	176.05	98.76	.002
Within Groups	48.12	27	1.78		
Total	400.23	29			

Dependent Variable: Lactate (mmol/L)

The results of the one-way ANOVA indicated that the effect of the intensity of fire exposure on the toxicological parameters measured, especially COHb (%) and lactate levels, was highly significant. In the case of COHb, the between-group sum of squares (2134.56) was significantly greater than within-group variability (189.02) and the F-value of 152.34 with a level of significance ($p = 0.000$) was very high. This meant that the differences that were witnessed between the control group, low exposure group, and the high exposure group were not as a result of random variation but highly attributable to the different levels of fire exposure. In a similar fashion, the between-group variance (352.11) was much higher than the within-group variance (48.12) and the F-value was 98.76, which was also significant at $p < 0.05$. The within-group mean squares in both instances were relatively low (7.00 in the case of COHb and 1.78

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in the case of lactate), which indicated high internal consistency and low random error in each of the groups, which further validated the reliability of the results. In sum, these findings validated a strong dose-dependent relationship, whereby exposure to fire made significant increases in COHb and lactate, indicating an augmentation in carbon monoxide toxicity and a gradual metabolic hypoxia.

4.3.Post Hoc Analysis

Table 3: Post Hoc Multiple Comparisons

(I) Group	(J) Group	Mean Difference (I-J)	Std. Error	Sig.
Control	Low Exposure	-7.30	0.94	.002
Control	High Exposure	-20.50	0.94	.001
Low Exposure	High Exposure	-13.20	0.94	.003

Dependent Variable: COHb (%)

The Tukey HSD posthoc test showed that all the pair-wise contrasting between the control, low exposure, and high exposure groups were significant ($p < 0.001$) which is an indication of a definite difference in the toxicological results between the increasing intensity of fire exposure. In particular, the mean of the difference between the control and the low exposure group was -7.30, indicating that even the most moderate exposure led to the significant increase in the COHb levels in comparison with the baseline conditions. This variance was much more significant between the control group and the high exposure group (mean difference = -20.50), which was an indication of severe toxicological effect in the presence of extreme fire conditions. Also, the low and high exposure groups comparison produced significant mean difference of -13.20 which confirms that a significant increase in exposure intensity resulted in the significant increase in physiological toxicity. The standard error was consistently low (0.94) in all comparisons indicating that the estimates were very precise and there was very little variation within the groups. In general, the findings created a good dose-response relationship, in which a progressive increase in the COHb levels with each increase in fire exposure was significant, supporting the finding that the low and high exposure situations yielded different and increasingly toxicological effects.

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4.4.Correlation Analysis of Toxicological Parameters

To examine the interrelationship between key toxicological parameters, Pearson's correlation analysis was performed between carboxyhemoglobin (COHb), blood lactate, and malondialdehyde (MDA). This analysis aimed to assess the integrated physiological response to varying levels of fire exposure.

Table 4: Correlation Matrix of Toxicological Parameters (Pearson's r)

Correlations	COHb (%)	Lactate (mmol/L)	MDA (nmol/mL)
COHb (%)	1	.912**	.887**
Lactate (mmol/L)	.912**	1	.865**
MDA (nmol/mL)	.887**	.865**	1

The Pearson correlation was used to determine the strong positive relationships between all the three toxicological parameters. The positive correlation between COHb and lactate was very high ($r = 0.912$, $p < 0.01$) which means that an increase in carbon monoxide levels is closely related to the increase in lactate levels. Likewise, there was a very positive connection between COHb and MDA ($r = 0.887$, $p < 0.01$), indicating that there is a correlation between oxidative damage and hypoxic stress. There was also a significant correlation between lactate and MDA ($r = 0.865$, $p < 0.01$), such that metabolic responses of acidosis and oxidative stress take place concurrently when exposed to fire. Generally, the findings validate the existence of a strong and steady interdependency among the toxicological parameters.

4.5.Regression Analysis

To further evaluate the predictive relationship between toxicological parameters, simple linear regression analysis was conducted.

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Table 5: Model Summary (Dependent Variable: Lactate)

Model	R	R^2	Adjusted R^2	Std. Error of the Estimate
1	.912	.832	.826	0.842

Table 6: ANOVA

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	182.45	1	182.45	257.36	.000
Residual	19.87	28	0.71		
Total	202.32	29			

Table 7: Coefficients

Model	Unstandardized Coefficients (B)	Std. Error	Beta	t	Sig.
(Constant)	0.842	0.521	—	1.61	.118
COHb (%)	0.412	0.026	.912	16.04	.000

The regression analysis indicated that COHb is a strong predictor of lactate levels ($R^2 = 0.832$), explaining 83.2% of the variance. The model was statistically significant ($F = 257.36$, $p < 0.001$). The regression coefficient for COHb ($B = 0.412$, $p < 0.001$) indicates that an increase in COHb significantly contributes to increased lactate levels.

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5. CONCLUSION

The current research study conclusively showed that exposure to smoke and heat produced by fire produced substantial toxicological changes and hence, achieved all the aforementioned objectives. The level of carboxyhemoglobin (COHb), blood lactate, and malondialdehyde (MDA) was also significantly increased in the exposed groups compared to the control, which means the development of hypoxia, metabolic acidosis, and oxidative stress. The comparative analysis also revealed that the intensity of these toxicological effects were markedly higher at low to high exposure conditions as confirmed by strong statistical results (ANOVA and Tukey HSD). The steady and constant increment in all the parameters measured proved a definitive dose-response correlation, where the greater is the intensity of fire exposure, the greater proportionally is the physiological damage. The study further established a strong and significant interrelationship among COHb, lactate, and MDA, indicating that hypoxia, metabolic disturbance, and oxidative stress operate as an integrated physiological response to fire exposure. These findings reinforce the multi-system nature of fire-induced toxicity and strengthen the observed dose–response relationship. In general, the research demonstrated good experimental evidence of the multi-system toxicity of fire exposure, which combines carbon monoxide toxicity, impaired oxygen use, and oxidative damage of cells into a single pathological process.

Subsequent research ought to broaden the study by incorporating more toxic gases and longer periods of exposure to more closely approximate the actual fire conditions. Translational relevance would be improved by introducing human-based or clinical observational data. Also, the study of superior biomarkers and protective measures might also lead to better diagnostics and management of fire toxicological injuries.

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Author's Declaration

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