

Impact of Long-Term Quarry Dust Inhalation on Antioxidant Defense Systems in an Experimental Animal Model

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Abstract

Quarrying is essential for supplying construction materials for modern society, but has been associated with several chronic health conditions. Inhaled quarry dust can enter the bloodstream and act as a source of toxicants capable of inducing cellular injury in vital organs. This study evaluated oxidative damage in the liver, heart, and kidneys through the assessment of oxidative stress indices following chronic exposure to quarry dust. An animal-based randomized controlled experimental design was employed, involving seventy-two (72) guinea pigs divided into three groups: Umuoghara (umr), Amofia-Ngbo (amn), and a control group (ctr), each comprising 24 animals. Groups were further subdivided based on duration of exposure (three, six, nine, and twelve months), with six guinea pigs per subgroup. Oxidative stress was evaluated by estimating antioxidative parameters comprising superoxide dismutase (SOD), glutathione reductase (GR), catalase (CAT) activities, and malondialdehyde (MDA) in organ homogenates. Data analysis was done using IBM SPSS version 23.0 and data presented as mean $\pm$ standard deviation. Results indicated that activities of SOD, GR, and CAT were significantly ($p < 0.05$) lower in guinea pigs subjected to quarry dust in relation to the control groups and consistent in all exposure periods. Conversely, MDA levels were significantly elevated and increased with exposure duration. These results indicate that chronic exposure to quarry dust can lead to marked oxidative stress and severe organ damage. It provides basic knowledge which can be utilized in development of therapeutic interventions, regulatory framework and environmental monitoring systems, thus integrating experimental toxicology and mechanistic biology with translational applications.

Keywords: Quarry dust, Umuoghara and Amoffia, organ homogenates, duration of exposure, oxidative stress indices

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Introduction

Quarrying of stones started in Abakaliki in 1902 during British colonial administration for the production of construction materials for public infrastructure. In the early 1950s, sedimentary rock was discovered in commercial quantities in Abakaliki. The Nigerian Mining Corporation (NMC) granted mining leases to Trade Afrik in 1956, which subsequently issued blasting licenses to several private companies in the area (Othuke, 2021). Stone-crushing industries within Abakaliki metropolis were relocated to Umuoghara in Ezza-North Local Government Area in 2007 to improve operational efficiency and easy environmental control and monitoring. However, quarry activities are still widely distributed across other parts of Ebonyi State, often in close proximity to residential communities.

Quarrying in Ebonyi State ranges from manual operations to large-scale industrial activities with heavy machinery (Okafor et al., 2023). Although industrialization has reduced manual labor, it has significantly increased dust emissions, exposing workers and nearby residents to airborne particulates through occupational and passive inhalation (Rajanayagam et al., 2023). Globally, rock quarrying has constituted a public health threat, especially in developing countries with very poor environmental regulation mechanism (Tsoloto, 2024).

The quarry workforce in Ebonyi State largely comprises untrained and unskilled individuals, with women constituting a substantial proportion of workers. Widespread poverty has driven vulnerable populations, including pregnant women, into quarry work, often without adequate personal protective equipment (NEITI, 2020). Poor compliance with dust emission standards, limited awareness of potential hazards, and suboptimal nutritional status of rural residents further aggravate the health risks associated with quarrying in this region. While short-term exposure to quarry or cement dust may pose minimal health risks, reports have associated prolonged exposure to multisystem health effects, depending on duration of exposure, dust concentration, and individual susceptibility (Kalu, 2018; Orinya et al., 2024). Dust particulates, especially PM₁₀ and PM_{2.5}, can remain airborne over long distances, penetrate deep into the respiratory tract, and induce chronic health challenges in man and animals. Beyond respiratory issues, researchers have recorded

increased risks of cardiovascular, hepatic, renal, neurological, and hematological disorders due to prolonged exposure to quarry dust (Bama *et al.*, 2023c; Orinya et al., 2024). Animal studies have demonstrated biochemical and physiological changes due to heavy metal exposure leading to disruption of the antioxidant defense mechanism and multiorgan dysfunction (Ungureanu and Mustatea, 2022). The effects of dust exposure on the respiratory system are well documented (Rajanayagam et al., 2023; Mwangi et al., 2023; Ike-Samuel et al., 2025). However, empirical data on the extent to which long-term quarry dust exposure damage antioxidant defense systems across vital organs are limited. Furthermore, ethical, logistic, and methodological concerns have limited the extent of investigations using human populations, hence the use of controlled experimental animal models. This study aimed to evaluate the antioxidant biomarkers and oxidative stress indices in selected organs as indicators of oxidative damage due to chronic exposure to quarry dust.

Materials and Methods

Ethical Approval

Ethical approval was obtained from the Research and Ethics Committee of the Faculty of Basic Medical Sciences of Ebonyi State University, with the number EBSU/EC/2410/01/003.

Experimental design

An animal based randomised controlled experimental research design was employed in the study to evaluate the antioxidant indices of the organ homogenates of the heart, kidneys and liver tissues of Guinea pigs exposed to quarry dusts in two quarry mines, Umuoghara and Amofia-Ngbo communities in Ebonyi state.

Animal selection and Experimental animal grouping.

Seventy-two (72) male guinea pigs (800–1200 g) were divided into three groups of 24 animals each and labeled Umuoghara (umr), Amoffia Ngbo (amn), and control (ctr). Four subgroups (n = 6) were created from each group. The umr and amn subgroups were exposed at the Umuoghara and Amoffia Ngbo quarry sites in a wooden cage at a distance of 3 meters from the crusher, respectively, while controls were kept at the

College of Agriculture (CAS), Ebonyi State University. The exposed animals were brought out in the morning (6 am) and taken into a nearby house daily (6 pm) after the closure of work. All animals were allowed free access to feed and water. Subgroups 1–4 were exposed for 3, 6, 9, and 12 months respectively and were evaluated accordingly.

Sample Collection

The ethical care protocols adopted in handling of the animals is according to the regulations of the ethical committee of Ebonyi State University, Abakaliki Nigeria. The guinea pigs were anesthetized using diethyl ether until loss of the righting reflexes and deep anaesthesia were obtained. The animals were sacrificed and tissue samples collected immediately for antioxidant analysis.

Determination of Oxidative Stress Indices

0.5 g of tissues from each organ was homogenized in 2.0 mL Phosphate buffer and KCl, and was subjected to centrifugation for 10 min at 2000 g. The supernatant was collected for analysis.

Measurement of SOD Activity

The activity of SOD was determined with a spectrophotometer following the nitroblue tetrazolium reduction method (Weydert and Cullen, 2010). The samples are mixed with phosphate buffer, methionine, nitroblue tetrazolium (NBT), ethylenediaminetetraacetic acid (EDTA), and riboflavin and allowed for 10–15 minutes. Superoxide radicals generated in the mixture reduced NBT to a blue-colored formazan and absorption measured at 560nm against the reagent blank. The SOD activity was recorded as Unit/mg-protein. The quantity of the enzyme that would inhibit the reduction of NBT by 50% is taken as one unit of the enzyme.

Determination of GR activity

The organ homogenates were spectrophotometrically assayed for GR activity at 340 nm by monitoring NADPH oxidation (Mannervik, 2001). The reagents consisted of phosphate buffer (pH 7.4), EDTA (1 mM), NADPH, and GSSG (1 mM). After addition of the sample the mixture, the absorbance was read at

340nm for 3 minutes (at 30 seconds interval). The GR activity was calculated from the rate of oxidation of NADPH to NADP⁺ using an extinction coefficient of $6.22 \times 10^3 \text{ M}^{-1} \text{ cm}^{-1}$ and expressed as units per milligram protein.

Determination CAT activity

This was determined with a spectrophotometer at 620 nm using 5% potassium dichromate and glacial acetic acid (1:3). Sample were incubated with a known concentration of H₂O₂ and the reaction terminated using potassium dichromate/acetic acid reagent. The unused H₂O₂ converts acidified dichromate to green colored chromic acetate complex and the absorbance measured spectrophotometrically. The number of moles of hydrogen peroxide converted was calculated as the enzyme activity (Weydert and Cullen, 2010).

Malondialdehyde (MDA) estimation

The formation of malondialdehyde (MDA) was assayed using the principle that Thiobarbituric acid (TBA) can react with MDA when formed to give an adduct which can be measured spectrophotometrically at 535 nm according to Nielsen et al., (1997) as reported by Rizzo, (2024). Samples were incubated for 30 min at 37 °C with 1.0 ml of 10% trichloroacetic acid (TCA), followed by centrifugation for 15 min at 2,000 g. 1 ml of supernatant was mixed with 1 ml of thiobarbituric acid in a separate tube. The tubes were allowed in warm water bath for 10 min, then cooled and optical density read at 535 nm.

Data analysis

Data was analyzed statistically using IBM SPSS version 23.0. Values are presented as mean $\pm$ standard deviations. Comparison of groups were done with one-way analysis of variance (ANOVA) followed by Tukey's post-hoc test for multiple comparison. Means with $P < 0.05$ were considered to be statistically significant.

Results

The Oxidative Stress Indices of the Heart, Kidney and Liver Tissue Homogenates

Table 1 shows that the SOD maintained significantly higher activities in the exposed groups than the controls at all exposure periods, as shown by different lowercase superscripts.

The SOD activities in amn and umr decreased numerically with length of exposure although not

statistically significant, while that of the control increased.

Table 1: Activity of Superoxide dismutase (SOD) in heart homogenates (U/mg)

Groups	3 months	6 months	9 months	12 months
Umr	11.30 ± 0.56 ^{aA}	9.90 ± 0.26 ^{aA}	9.26 ± 0.12 ^{aA}	8.00 ± 0.82 ^{aA}
Amn	9.08 ± 1.82 ^{aA}	4.74 ± 0.49 ^{aA}	4.20 ± 0.48 ^{aB}	2.60 ± 0.28 ^{bA}
Ctr	17.34 ± 1.10 ^{bA}	20.15 ± 0.24 ^{bA}	21.86 ± 0.69 ^{bA}	21.00 ± 0.54 ^{cA}

Values are presented as mean ± SD (n=6) and means with p<0.05 are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

In Table 2, GR activity was significantly lower in umr and amn groups than in the controls throughout the study. It also decreased significantly (p<0.05) between 3 months exposure and 12 months exposure for both

quarry sites, while that of the control increased across the exposure periods. The reduction in the enzyme activities was more pronounced in site amn than in umr.

Table 2: Activity of Glutathione reductase (GR) in heart homogenates (U/mg)

Groups	3 months	6 months	9 months	12 months
Umr	10.91 ± 0.18 ^{aB}	7.70 ± 1.04 ^{aA}	6.72 ± 0.50 ^{aA}	6.10 ± 0.49 ^{aA}
Amn	7.70 ± 0.17 ^{bB}	6.40 ± 0.81 ^{aB}	6.30 ± 0.06 ^{aB}	5.40 ± 0.80 ^{aB}
Ctr	19.08 ± 1.02 ^{cA}	20.73 ± 0.51 ^{bA}	22.70 ± 1.71 ^{bA}	24.60 ± 1.90 ^{bB}

Values are presented as mean ± SD (n=6) and means with p<0.05 are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

In Table 3, the heart homogenates recorded a significant decrease in the activities of CAT across the exposed groups when compared to the controls which were the reverse. The activities decreased across the exposure periods

with some fluctuations. The activities at 12 months group showed a significant (p<0.05) decline in the enzyme activities compared to the 3 months exposure.

Table 3: Activity of Catalase (CAT) in heart homogenates of guinea pigs (U/mg)

Groups	3 months	6 months	9 months	12 months
Umr	152.14 ± 1.93 ^{aA}	126.56 ± 0.20 ^{aC}	158.20 ± 0.14 ^{aA}	110.80 ± 1.26 ^{aB}
Amn	141.98 ± 0.30 ^{bA}	94.80 ± 0.87 ^{bD}	149.30 ± 2.19 ^{bC}	102.30 ± 0.15 ^{bB}
Ctr	188.13 ± 1.20 ^{cA}	196.70 ± 2.20 ^{cA}	202.46 ± 0.40 ^{cB}	184.00 ± 1.60 ^{cA}

Values are presented as mean $\pm$ SD (n=6) and means with $p < 0.05$ are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

Table 4 shows the level of MDA in the heart homogenates of exposed guinea pigs. The exposed groups recorded significantly elevated levels of MDA than the control groups. The MDA

increased consistently in amn and umr up till 9 months exposure. The impact was more significant in Amn compared to Umr

Table 4: Level of MDA in heart homogenates (nmol/g)

Groups	3 months	6 months	9 months	12 months
Umr	13.85 $\pm$ 0.12 ^{aA}	36.19 $\pm$ 0.81 ^{aC}	42.30 $\pm$ 1.63 ^{aB}	41.50 $\pm$ 1.32 ^{aB}
Amn	26.88 $\pm$ 0.64 ^{bA}	51.60 $\pm$ 0.31 ^{bB}	52.10 $\pm$ 1.29 ^{bB}	54.60 $\pm$ 1.27 ^{bB}
Ctr	15.01 $\pm$ 1.81 ^{aA}	13.13 $\pm$ 1.37 ^{cA}	16.70 $\pm$ 1.02 ^{cA}	22.80 $\pm$ 2.00 ^{cB}

Values are presented as mean $\pm$ SD (n=6) and means with $p < 0.05$ are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

Table 5 showed that the SOD activities in kidney homogenates of the exposed guinea pigs were all significantly ($p < 0.05$) lower relative to the control. The decrease across the exposure

periods was not significant except at Amn where the 12 months exposure was significantly lower than the 3 months exposure.

Table 5: Activity of superoxide dismutase (SOD) in kidney homogenates (U/mg)

Groups	3 months	6 months	9 months	12 months
Umr	10.25 $\pm$ 0.88 ^{aA}	10.03 $\pm$ 0.75 ^{aA}	11.00 $\pm$ 1.24 ^{aA}	8.20 $\pm$ 0.16 ^{aA}
Amn	10.01 $\pm$ 0.08 ^{aA}	8.40 $\pm$ 0.19 ^{aA}	9.60 $\pm$ 0.50 ^{aA}	5.40 $\pm$ 1.20 ^{aB}
Ctr	15.87 $\pm$ 1.01 ^{bA}	17.60 $\pm$ 0.28 ^{bA}	17.80 $\pm$ 1.09 ^{bA}	18.20 $\pm$ 1.11 ^{bA}

Values are presented as mean $\pm$ SD (n=6) and means with $p < 0.05$ are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

Table 6 showed that the GR activities in kidney homogenates were all significantly ($p < 0.05$) lower than those of the control. The enzyme

activities at 3 months exposure were significantly different from other exposure periods with some fluctuations.

Table 6: Activity of Glutathione Reductase (GR) in kidney homogenates (U/mg)

Groups	3 months	6 months	9 months	12 months
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Umr	12.60 ± 1.50 ^{aA}	7.32 ± 0.24 ^{aB}	10.60 ± 0.80 ^B	8.40 ± 0.34 ^{aB}
Amn	10.22 ± 0.35 ^{aA}	4.87 ± 0.16 ^{aB}	6.30 ± 1.39 ^{bB}	6.80 ± 1.20 ^{bB}
Ctr	20.11 ± 0.08 ^{bA}	26.53 ± 1.31 ^{cB}	22.70 ± 1.71 ^{cB}	22.80 ± 1.44 ^{cB}

Values are presented as mean ± SD (n=6) and means with p<0.05 are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

Table 7 showed significantly lower activities of CAT in the exposed groups relative to the controls. The enzyme activities decreased significantly (p<0.05) across the exposure

periods in Umr. At Amn, there was a significant decrease between 3 months exposure and other exposure periods which only showed numerical reductions across.

Table 7: Activity of Catalase (CAT) in kidney homogenates (U/mg)

Groups	3 months	6 months	9 months	12 months
Umr	140.00 ± 1.00 ^{aA}	112.00 ± 1.00 ^{aB}	105.00 ± 0.00 ^{aC}	102.00 ± 1.00 ^{aC}
Amn	135.00 ± 1.00 ^{aA}	105.00 ± 1.00 ^{bB}	102.00 ± 0.00 ^{aB}	100.00 ± 0.00 ^{aC}
Ctr	176.00 ± 1.00 ^{bA}	183.00 ± 0.00 ^{cB}	202.00 ± 0.00 ^{bC}	190.00 ± 2.00 ^{Bd}

Values are presented as mean ± SD (n=6) and means with p<0.05 are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

In Table 8, the concentration of MDA was significantly lower in the control compared to both exposed groups. The level of MDA increased consistently in amn and umr in the first three

exposure periods but dropped at the 12 months exposure. The impact was more significant in Amn compared to Umr.

Table 8: Malondealdehyde (MDA) level in kidney homogenates (nmol/g)

Groups	3 months	6 months	9 months	12 months
Umr	18.10 ± 0.22 ^{aA}	30.09 ± 0.20 ^{aB}	42.30 ± 1.00 ^{aC}	24.82 ± 0.18 ^{aD}
Amn	27.12 ± 0.34 ^{bA}	47.10 ± 1.00 ^{bB}	52.10 ± 1.00 ^{bC}	30.60 ± 1.00 ^{bA}
Ctr	10.33 ± 1.00 ^{cA}	10.81 ± 0.00 ^{cA}	16.70 ± 1.00 ^{cB}	18.60 ± 1.00 ^{cB}

Values are presented as mean ± SD (n=6) and means with p<0.05 are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

The SOD activities in the liver homogenates (Table 9), showed a significant (p<0.05) compared to the controls and decreased numerically across the exposure periods with no

statistical difference. The enzyme activities recorded at Amn also indicated no significant (p<0.05) difference compared to their counterparts at Umr.

Table 9: Activity of Superoxide Dismutase (SOD) in liver homogenates (U/mg)

Groups	3 months	6 months	9 months	12 months
Umr	13.40 ± 0.29 ^{aA}	11.24 ± 0.45 ^{aA}	10.60 ± 0.44 ^{aA}	10.00 ± 0.90 ^{aA}
Amn	12.20 ± 0.34 ^{aA}	11.00 ± 0.39 ^{aA}	9.80 ± 0.14 ^{aA}	8.70 ± 0.42 ^{Aa}
Ctr	22.22 ± 0.87 ^{bA}	18.90 ± 1.50 ^{bA}	22.10 ± 1.14 ^{bA}	26.10 ± 0.27 ^{aA}

Values are presented as mean ± SD (n=6) and means with p<0.05 are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

The activity of the enzyme GR in liver homogenates (Table 10), were all significantly (p<0.05) lower than those of the control and decreased with length of exposure. The decrease was not significant between 3 months and 9

months exposure periods but at 9 and 12 months. The enzyme activities are generally higher in the control groups throughout the period of exposure.

Table 10: Activity of Glutathione Reductase (GR) in liver homogenates (U/mg)

Groups	3 months	6 months	9 months	12 months
Umr	19.00 ± 1.00 ^{aA}	18.00 ± 2.00 ^{aA}	14.00 ± 2.00 ^{aA}	8.00 ± 0.00 ^{aC}
Amn	16.00 ± 2.00 ^{aA}	14.00 ± 2.00 ^{aA}	10.00 ± 0.00 ^{aB}	6.00 ± 0.00 ^{aC}
Ctr	28.00 ± 0.00 ^{bA}	20.00 ± 0.00 ^{bB}	28.00 ± 0.00 ^{bA}	26.00 ± 0.00 ^{bA}

Values are presented as mean ± SD (n=6) and means with p<0.05 are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

In Table 11, the CAT activities were significantly lower in exposed groups than the control. It decreased significantly with duration of exposure in Umr exposed guinea pigs. Amn recorded

significant decrease only at 12 months exposure. In contrast, control animals maintained relatively stable catalase activity throughout the study period.

Table 11: Activity of Catalase (CAT) in kidney homogenates (U/mg)

Groups	3 months	6 months	9 months	12 months
Umr	187.30 ± 1.00 ^{aA}	180.20 ± 1.00 ^{aB}	165.92 ± 1.20 ^{aC}	101.40 ± 1.00 ^{aD}
Amn	160.70 ± 90 ^{bA}	160.22 ± 0.52 ^{bA}	158.30 ± 1.00 ^{bA}	118.36 ± 0.86 ^{bB}
Ctr	198.54 ± 0.45 ^{cA}	188.60 ± 0.38 ^{cB}	196.60 ± 1.00 ^{cA}	204.00 ± 1.40 ^{cC}

Values are presented as mean ± SD (n=6) and means with p<0.05 are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

Table 12 showed that MDA concentrations were significantly higher in exposed animals compared to controls. The Amn group expressed the highest values, particularly at 12 months. Across the period of exposure, there was a

significant increase between 3 months and 12 months periods. The control animals maintained relatively stable levels of MDA throughout the study period.

Table 12: Activity of Malondealdehyde (MDA) in liver homogenates (nmol/g)

Groups	3 months	6 months	9 months	12 months
Umr	30.18 ± 0.32 ^{aA}	34.30 ± 1.63 ^{aB}	32.42 ± 1.21 ^{aB}	34.18 ± 1.33 ^{aB}
Amn	32.14 ± 0.90 ^{aA}	38.75 ± 1.57 ^{aB}	36.31 ± 0.88 ^{bB}	40.80 ± 1.50 ^{bC}
Ctr	28.32 ± 0.42 ^{aA}	30.32 ± 0.15 ^{bA}	30.85 ± 2.05 ^{aA}	30.00 ± 0.11 ^{aA}

Values are presented as mean ± SD (n=6) and means with $p < 0.05$ are significantly different. Different lowercase superscripts in the same column, and uppercase superscripts in the same row differ significantly.

Discussions

In this study, the trend in the oxidative index of the prolonged exposure to quarry dust at Umuoghara (umr) and Amoffia Ngbo (amn) quarry sites pointed to a duration-dependent oxidative injury to the vital organs (heart, kidney, and liver) of exposed guinea pigs, as shown by significant ($p < 0.05$) reductions in the activities of key antioxidative enzymes relative to the controls. These trends were progressive with increase in duration of exposure with some fluctuations, indicating that quarry dust can substantially cause a disruption of the antioxidative system. This was consistent with the reports of Al-Otaibi et al., (2018) who recorded significant elevations in the concentrations of Cadmium, Lead, Nickel, and Vanadium in liver, kidney, lung, and furs of rodents. The heavy metals or toxic concentration of safe metals have long been established to induce oxidative damage. Similarly, other reports have shown increased oxidative damage following chronic exposure to stone dust, evidenced by elevated ROS concentrations (Ophir et al., 2025). Orinya et al., (2025), has previously reported time-dependent increase in the concentration of heavy metals in whole blood of exposed guinea pigs at Umuoghara (umr) and Amoffia Ngbo (amn) quarry sites. Metals in quarry dust may bind to cellular proteins,

displacing essential metal cofactors leading to enzyme malfunction. Since several antioxidant enzymes including SOD and GR are metalloenzymes, inhalation of quarry dust would likely have contributed to the observed activity loss (Jomova et al., 2023). A corresponding and significant ($p < 0.05$) elevation of MDA, a sensitive marker of oxidative stress particularly in the heart, was observed in the exposed animals consistently with the duration of exposure. This is in tandem with the reports that heavy metals promote oxidative stress and increase MDA concentrations (Al-Otaibi et al., 2018). Both ROS released by toxicants, and inflammatory response due to sensitivity to dust can cause oxidative damage to cellular macromolecules (Muscolo et al., 2024). The higher oxidative burden observed in animals exposed to site 'amn' compared to umr in most cases may not be unconnected to the higher emissions of dust with higher concentrations of heavy metals and silica as recorded in this result. Similar relationships between metal-rich particulates and the antioxidative markers were reported by Widiastuti et al. (2019). Basically, SOD converts superoxide radicals into H_2O_2 and O_2 , while catalase reduces H_2O_2 to water and oxygen, preventing accumulation of hydroxyl-radicals (Zheng et al., 2023). The body requires GR to covert oxidized glutathione (GSSG) to the reduced form, thus maintaining adequate pool of

reduced glutathione (GSH) from GSSG, which is key to all antioxidative processes. Reduced GR activities recorded in this research may have resulted in reduced GSH/GSSG ratio and increased susceptibility to oxidative damage (Georgiou-Siafis et al., 2023). Thus, combined depletion of SOD, CAT, and GR activities recorded may have resulted in the concomitant elevation of MDA seen in this study.

Decreased antioxidant enzyme activities recorded with concomitant increase in MDA is an already established pattern of particulate-induced oxidative stress consistent with reports that inhaled particles directly or indirectly produce ROS that can overwhelm endogenous antioxidant defenses (Lim & Kim, 2024). Generation of ROS by particulate matters could be directly from redox-active metals through Fenton reaction, or indirectly from systemic oxidative stress due to inflammation and cytokines release in the lungs. Finer dust particulate matter containing redox-active metals (Fe, Cu, Mn, Cr), have been reported to exhibit higher oxidative potentials due to increased surface area (Laniyan et al., 2024). The more severe biochemical effects of quarry dust on the oxidative indices observed in Amoffia Ngbo quarry site compared to Umuoghara quarry site may be attributed to the higher emissions of dust containing higher concentrations of heavy metals and silica or smaller particle sizes as reported in previous work (Orinya et al., 2025). The few fluctuations in the activities of the enzymes observed across the exposure periods in some cases as indicated by the uppercase superscripts may be as a result of reduced activities in the quarry sites which sometimes occurs or the ability of some organs to regenerate.

The results showed clear organ-specific changes. The consistently higher SOD activity of the exposed groups compared to the controls as indicated by different lowercase superscripts demonstrated that quarry dust exposure significantly suppressed cardiac SOD activity. Within the Umr group, the identical uppercase superscript across all exposure periods indicates that although SOD values declined numerically from 11.30 to 8.00 (U/mg), the decrease was not statistically significant. Conversely, the significant decrease exhibited by Amn group at 12 months (B versus A), is indicative of progressive decline of antioxidant defenses with prolonged exposure.

The significant reductions in most antioxidative enzyme activities with highest elevation of MDA in the heart, could be attributed to high mitochondrial density with limited capacity to regenerate, making it very susceptible to oxidative damages. These results are concordant with findings from other particulate-exposure models which linked PM-induced oxidative stress with cardiac dysfunction and markers of myocardial damage. The significantly higher changes seen in site 'amn' is suggestive of either a higher redox-active constituent (e.g., transition metals) or finer PM fractions that more readily permeates the systemic circulation (Vilas-Boaz et al., 2024).

The kidney exhibited substantial oxidative disruptions. It has the ability to concentrate xenobiotics and exhibit redox cycling, but with reduced expression of antioxidant system, making it susceptible to metal-induced oxidative stress. Bama (2023), has reported similar mechanisms linked to occupational nephrotoxicity in workers exposed to quarry dust. The kidney's consistent and marked decreases in SOD, GR and CAT activities, with elevation of MDA across the exposure periods compared to the controls, is suggestive of the accumulation of the toxicants in the kidney, or damage from circulating inflammatory mediators due to dust exposure. Kidneys concentrate xenobiotics and are susceptible to redox imbalance; heavy metals and other particulate-borne redox active contaminants (Bama, 2023). The observed significant reduction in the enzyme activities at other exposure periods compared to 3 months exposure is suggestive of impairment of renal oxidative defense, although the decrease in SOD activities at site Umr was not significant across all exposure periods as indicated by same uppercase superscript. The consistent elevation in MDA concentrations up to 9 months exposure period suggests a cumulative oxidative disruption of renal cell integrity. Although the concentration of MDA declined at 12 months exposure period, they are still substantially higher than control values, indicative of continuous oxidative injury. Such fluctuations may be due to adaptation by the renal tissues, or alterations in metabolic processes of membrane lipids as a result of long-term exposure. Several studies have reported elevated renal MDA concentrations and environmental pollutant-induced nephrotoxicity

as indicators of oxidative kidney damage (Halliwell and Gutteridge, 2015).

Although the liver initially recorded a lower MDA response, the progressive decline in the activities of liver antioxidant enzymes in this work is suggestive of overwhelmed compensatory detoxification mechanism leading to hepatic dysfunction (Lim and Kim, 2024). The significant ($P<0.05$) decrease in hepatic SOD activity recorded in exposed groups indicates impaired removal of superoxide radicals. The decline in the activities across exposure periods were not statistically significant, but the progressive decrease relative to controls indicates prolonged oxidative challenge. Reduction in GR activity further indicates disruption in the ability of the liver to regenerate reduced glutathione (GSH) critical in maintenance redox homeostasis. The significant decline recorded at 12 months relative for other exposure periods as indicated by the uppercase superscripts suggests progressive depletion of the antioxidant reserves. Similar findings have been reported in animals exposed to silica dust, diesel exhaust particles etc (Xiao et al., 2024). The significant reduction in CAT activity further supports the presence of chronic oxidative damage.

While the liver showed significant reductions in antioxidative enzyme activities, increase in MDA levels were moderate compared to heart and kidney. The liver's extensive detoxification apparatus and higher antioxidant pools can temporarily mitigate oxidative challenge, which might explain the modest MDA trajectory on initial exposure. There was persistent increase in MDA level in all exposure periods relative to the control as indicated by the lowercase superscripts suggesting continuous oxidative damage to membrane lipids. Increased hepatic lipid peroxidation is a well-documented consequence of exposure to particulate pollutants and industrial dusts (Xiao et al., 2024). Nevertheless, progressive decline in the activities of liver antioxidant enzymes in this report is suggestive of overwhelmed antioxidant capacity as a result of prolonged exposure.

Generally, this study has indicated that long-term exposure to quarry dust produce systemic oxidative stress and organ damage which significantly increases with duration of exposure. These records are consistent with epidemiologic

data of cardiovascular, renal, and hepatic dysfunction among workers exposed to stone dust and highlight significant public health issues surrounding quarry sites and populations within the vicinity.

Although, this study employed animal models, the biochemical changes reported are relevant to human occupational and environmental health studies. Long-term exposure to redox active dust can lead to multi-organ dysfunction, fibrosis, and chronic diseases such as CVD, CKD and necrosis over time. Epidemiologic and occupational studies have reported kidney problems and systemic health effects among stone quarry workers and nearby communities similar to our findings (Bama, 2023).

Conclusion

Long-term exposure to quarry dust from Umuoghara and Amoffia Ngbo quarry sites caused progressive reduction in SOD, CAT, and GR activities with concomitant elevation in MDA levels in vital organs confirming systemic oxidative stress. These findings underscore the urgent need for a wholistic approach involving innovative biotechnological, genetic and epigenetic strategies to mitigate particulate matter (PM) induced adverse health. Recombinant and bioengineered antioxidant enzymes with enhanced antioxidative capacity, and advanced delivery systems such as PEGylation and nanoparticle encapsulation that improve their stability, bioavailability, and tissue targeting. Also, biotechnological innovations can provide biomarker-driven diagnostic platforms for early detection and environmental biotechnology for pollution control. Collectively, these will provide promising avenues for preventing PM-related diseases, facilitating early diagnosis, attenuating inflammatory and oxidative damage, and promoting restoration of damaged tissues.

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Declaration of conflict of interest

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