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Effect of Air Pollution on Human Health of Workers in A Factory

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Abstract

Indoor air pollution (IAP) threatens human health, causing millions of deaths each year. The aim of this work is to determine the levels of pollutant gas NO_2 and particulates ($PM_{2.5}$, PM_{10}) in indoor air in investigated site (factory) and how it affects the health of workers. Pollutant gas NO_2 and particulates($PM_{2.5}$,PM_{10})were measured and blood samples were taken from twenty workers in the same investigated site working in manufacture of casing and tubing and tested for creatine kinase test, catalase test and alpha tumor necrosis test .Three samples of NO_2 gas , Three samples of $PM_{2.5}$,Three samples of PM_{10}were taken in three months each in winter and also in spring .The levels of Selected pollutant gas NO_2 and particulates($PM_{2.5}$,PM_{10}) mean and St. Deviation are 4.42 ±0.01 µg /m3, 1.59± 0.51 µg /m3, 2.38±0.01 µg /m3 respectively in winter and in Spring are 4.43 ±0.02 µg /m3, 1.86± 0.02 µg /m3, 1.22±0.02 µg /m3. Results of blood samples of workers were creatine kinase test, catalase test, Alpha tumor necrosis test were 39.35±11.43ng/ml, 9.30±4.18ng/ml, 16.15±3.34pg/ml respectively which showed that it is highly significant for causing stroke and cardio vascular diseases .This means that exposure to indoor air pollutants may be cause stroke and cardiovascular diseases.

Keywords: NO_2 air pollutant gas- particulates- blood samples-- Alpha Tumor necrosis test-Creatine kinase test-catalase test

1. Introduction

The consequences of air pollution on health are diverse. Even on days with less air pollution, sensitive and vulnerable people's health may be affected. The following conditions are strongly associated with short-term exposure to air pollution: asthma, respiratory diseases, wheezing, cough, shortness of breath, and high rates of hospitalization (a measure of morbidity). Chronic asthma, pulmonary insufficiency, cardiovascular illnesses, and cardiovascular mortality are the long-term impacts linked to air pollution [1]. Many types of power plants, factories, automobiles, and other forms of transportation are known to be examples of anthropogenic sources. The main source of anthropogenic NO_X emissions is fuel-burning machinery. Annually, into the atmosphere burning fossil fuels releases more than 23 million tons of NO_X. Moreover, combustion sources that generate electricity for contractual sale to a grid or power pool are known as electric generating units (EGUs). EGUs are classified as steam and non-steam generating boilers and are all point sources. Boilers that don't produce steam include internal combustion engines, gas turbines, and Large sources that fall into the categories of industrial processes or non-EGU fuel

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combustion sources are classified as non-EGU point sources. Several small-to-moderate-sized boilers are employed as industrial fuel combustion sources used in manufacture of casing and tubing to produce steam and/or power for on-site consumption at the facilities. Cement and lime kilns, industrial furnaces, and other industrial processes that use burners to provide process heat are also included in this category. Coal, oil, gas, coke, and wood are the main fuels used in industrial furnaces and boilers. industrial procedures such as:

- Manufacturing of chemicals and related products;
- Processing of metals;
- Use of solvents;
- Petroleum refining and associated sectors;
- Disposal of waste;
- Storage and transportation;

Smaller sources that do not exceed the size criterion for reporting emissions individually are usually categorized as stationary area sources. Commercial, institutional, residential, and other fuel combustion sources are all considered "other fuel combustion sources." Some examples of non-road mobile sources are as follows:

- Aircraft,
- Commercial and recreational marine boats,
- Railroads,
- Lawn and garden equipment,
- Logging and construction equipment,
- And industrial engines

Highway vehicles, which are certified for usage on highways, comprise diesel vehicles, pickup trucks and vans, motorbikes, sport utility vehicles, and passenger cars and trucks.

Natural resources such as Emissions from naturally occurring biological activity are referred to as biogenic sources (i.e., living organisms). Forests, crops, and soils can all produce biogenic NOX emissions. Land use and climatology—or temperature—are two major variables that affect the amount of biogenic emissions that are produced. NOX emissions from geogenic sources include:

- Stratospheric intrusion (NO_X)
- Marine ecosystems (NO_X, N_2O)
- Material burning (NO_X, NH_3)
- Lightning (NO_X, N_2O)
- Geysers and volcanoes (NO_x , N_2O ,NH_3)

The photolysis of nitrates in saltwater near the ocean's surface releases NOX emissions into the atmosphere. The atmospheric reactions over landmasses are not notable affected by the modest amount of NOX that marine ecosystems contribute. A sequence of lightning strikes makes up a lightning flash. Fires such as Wildfires, prescribed burning, coal refuse fires, burning solid waste, burning agricultural drums, and structural fires are examples of burning sources. These sources release very variable and challenging to measure emissions. The incursion of stratosphere produces very little NOX. Sunlight causes nitrous oxide in the stratosphere to oxidize and dissociate. The troposphere is where the freshly created NO_X compounds descend. Geysers and volcanoes are other sources [2].

. Nitrogen oxides (NO x), the sum of nitric oxide (NO) and nitrogen dioxide (NO_2), play a critical role in regulating ozone, aerosol, and hydroxyl radical levels in the troposphere [3]

Humans are generally exposed to various air pollutants through inhalation and ingestion, with skin contact being a less common mode of exposure. Food and water contamination is largely caused by air pollution with NOx, making ingestion the main method of pollutant intake in many situations. Pollutants may be absorbed by gastrointestinal tracts and the respiratory tract, and many of the harmful chemicals are detected in the general circulation and accumulate in various tissues. Excretion contributes to elimination to some extent. [4].

NO_X family (for NADPH Oxidase) The transmembrane proteins known as NADPH oxidases are responsible for the single electron that is transferred from NADPH to molecular oxygen, resulting in the creation of superoxide. The oxidative burst, or NO_X-mediated release of ROS, is a process that helps in the destruction of invasive microorganisms in macrophages and neutrophils. Unlike other enzymes, which produce ROS as a byproduct of their primary biological function, NO_X enzymes do not produce ROS as a physiological byproduct. The fact that individuals with a hereditary NO_{X2} impairment suffer chronic granulomatous disease (CGD) and are unable to fight off common infections highlights the significance of ROS in the host immune response. Phagocytes were the source of NOX2, the first NADPH oxidase which was followed by the identification of other NO_X family NADPH oxidases, which are not just present in phagocytes but are present in almost all tissues . Evidence suggests that NO_X enzymes are critical to the pathogenesis of a number of CVDs [5].

The term "particulate matter" (PM) refers to a class of air pollutants that are created by a wide range of human and natural activities. These particles come in many sizes and shapes and can be made up of hundreds of different chemicals. Some are emitted directly from a source, such as construction sites, unpaved roads, fields, smokestacks or fires.

Most particles form in the atmosphere as a result of complex reactions of chemicals such as sulfur dioxide and nitrogen oxides [6], Parts of Particulates can enter the human body through four different routes, as with any chemical contaminant: ingestion, skin absorption, and inhalation [7].These pollutants are composed of complex and variable combinations of particles suspended in the breathing air, varied in composition and size. Manufacturing facilities, power plants, waste incinerators, automobiles, building sites, wildfires, and naturally occurring wind-blown dust are the main causes of particle pollution. Particle sizes vary; $PM_{2.5}$ and PM_{10} correspond to aerodynamic diameters of less than 2.5 mm and 10 mm, respectively [4].

The majority of inhaled particles with an aerodynamic diameter of 10 μm or more have an effect on the nasopharyngeal membranes. Particles that are 5–10 μm in size when inhaled often settle in the airways and are cleared by lung lymphatics and alveolar macrophages. When a patient with centrilobular emphysema, particles in the 1–2.5 μm range typically go to the terminal bronchiole, which is the region of maximum accumulation and tissue degradation.

Numerous studies have demonstrated that PM, as indicated by higher C-reactive protein (CRP), circulating polymorph nuclear leukocytes, platelets, fibrinogen, plasma viscosity, and other indicators, induces systemic inflammation and coagulation alterations that predispose to ischemic cardiovascular disease. Particulate increases vascular inflammation, atherosclerosis, and endothelial dysfunction [8].

According to recent research, particle-phase material derived from secondary-organic aerosol (SOA; simulated atmospheric processing of aerosol precursors) may also have oxidative potential [9]. According to Shen and Anastasio, real PM from the San Joaquin Valley can also produce OH and H_2O_2 in lung fluid used as a surrogate [10,11]. Redox-active materials can sustain, catalyze, or induce reactions within the epithelial lining fluid that produce reactive oxygen species (ROS), including hydroxyl radical (OH), superoxide (O_2), hydroperoxy radical (HO_2), singlet oxygen, alkoxy radicals (RO), , ozone (O_3), and hydrogen peroxide (H_2O_2), alkylperoxy (ROO). This is after the materials are inhaled and deposited onto the epithelium of the airway. Although the reactivity of these compounds differs, ROS frequently show reduction potentials of E0 >+0.9V, indicating that they are highly reactive and essentially unaffected by the type of reaction partner [12]. ROS frequently combine with oxygen, transition metals, or other materials to create catalytic cycles that raise oxidative stress and renew reaction mediators. These chemical cycles, which are thought to occur in the fluid surrounding the epithelium, have recently been compiled by [13] work The transfer of electrons to transition metals or quinones is the first stage of a redox cycle.

Statistical methods used

2. Materials and Methods

2.1. Monitoring of NO_2 gas

Gases is measured by vRAE from indoor air of investigated site working on the manufacture of casing and tubing.

The VRAE is a multigas monitor that may be programmed to offer workers in hazardous settings with continuous exposure monitoring of oxygen, flammable gases, and poisonous gases. VRAE are accessible: PGM-7840 and ASTM D6216-03 measurement methodology [14].

2.2. Monitoring of particulates ($PM_{2.5}$, PM10)

Particulates ($PM_{2.5}$, PM_{10}) concentrations are measured by pDR-1500 from indoor air of investigated site working on the manufacture of casing and tubing.

The legacy light scattering sensor configuration of the pDR-1500, a very sensitive nephelometric (also known as photometric) monitor, has been enhanced for the detection of the respirable proportion of airborne dust, smoke, fumes, and mists in industrial and other indoor and outdoor environments[15].

2.3.1. Determination of blood parameters for workers

2.3.1. (A) Creatine Kinase –mb (Ck-mb) Test (Beacon)

40Blood samples, 20 samples from male workers (exposed to $N0_2$,$PM_{2.5}$,PM_{10} pollution) from investigated site as cases and20 males (not exposed) as control outside the investigated site are made by Ck-mb (mono) IFCC method [16] by using bio analyzer

2.3.1. (B) Catalase kit test: Cayman chemical

40 Blood samples, 20 samples from male workers (exposed to $N0_2$,$PM_{2.5}$,PM_{10} pollution) from investigated site as cases and 20 males (not exposed) as control outside the investigated site are made by Catalase test by a method described by [17].

2.3.1. (C) Alpha tumor necrosis factor kit test

40 Blood samples, 20 samples from male workers (exposed to $N0_2$,$PM_{2.5}$,PM_{10} pollution) from investigated site as cases and20 males (not exposed) as control outside the investigated site are made by Alpha tumor necrosis test was carried out by a method described by ELIZA Technique [18].

Data were unloaded through a statistical package for Social Sciences Statistical Program IBM SPSS V. 25 Statistical Package and through it was using the following test:
Mean and standard deviation. Mann-Whitney Test to study difference between samples

3. Results and discussion

3.1. By analyzing the collected air samples, it was found that:

It seemed that there are higher levels of NO_2, $PM_{2.5}$ and PM_{10} in indoor air in winter and spring in investigated site (the factory) in the year 2022 but not exceeding the concentration of those pollutants in the executive rules of the Egyptian Environment Law (4) for the year 1994.
This agreed with Vardoulakis et al., [19] in higher levels of NO_2, $PM_{2.5}$ and PM_{10} in 141 studies from 29 countries. Also, agreed with Alberts [20] in higher level of NO_2 in Florida.

Table 1: showed Selected pollutant gas NO_2 and particulates ($PM_{2.5}$, PM_{10}) concentration (µg /m3) in the collected samples in the selected industrial site during the year 2022 of the study in winter in the following

	NUMBER	NO_2	$PM_{2.5}$	PM_{10}
JANUARY	1	4.41	1.89	2.37
	2	4.42	1.88	2.38
	3	4.43	1	2.39
	Mean	4.42	1.59	2.38
	SD	0.01	0.51	0.01

P- Value: significant at level less than (0.01)

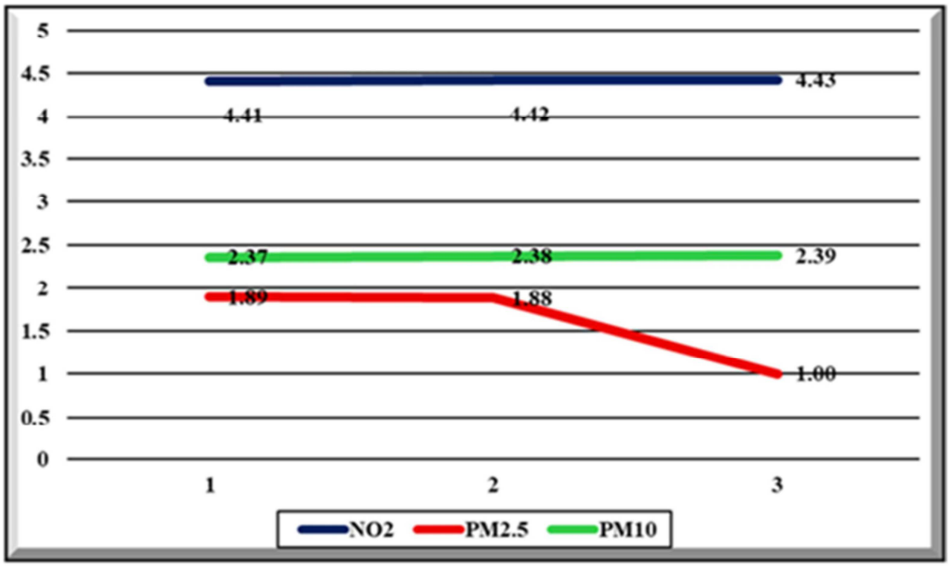

Figure 1: Shows (winter) the average of PM2.5, PM10 and NO_2

Table (2): showed Selected pollutant gas NO_2 and particulates ($PM_{2.5}$, PM_{10}) concentration (µg /m3) in the collected samples in the selected industrial site during the year 2022 of the study in spring in the following

SEASON	NUMBER	NO_2	$PM_{2.5}$	PM_{10}
March	1	4.44	1.88	1.24
	2	4.41	1.86	1.21
	3	4.45	1.85	1.22
	Mean	4.43	1.86	1.22
	SD	0.02	0.02	0.02

P- Value: significant at level less than (0.01)

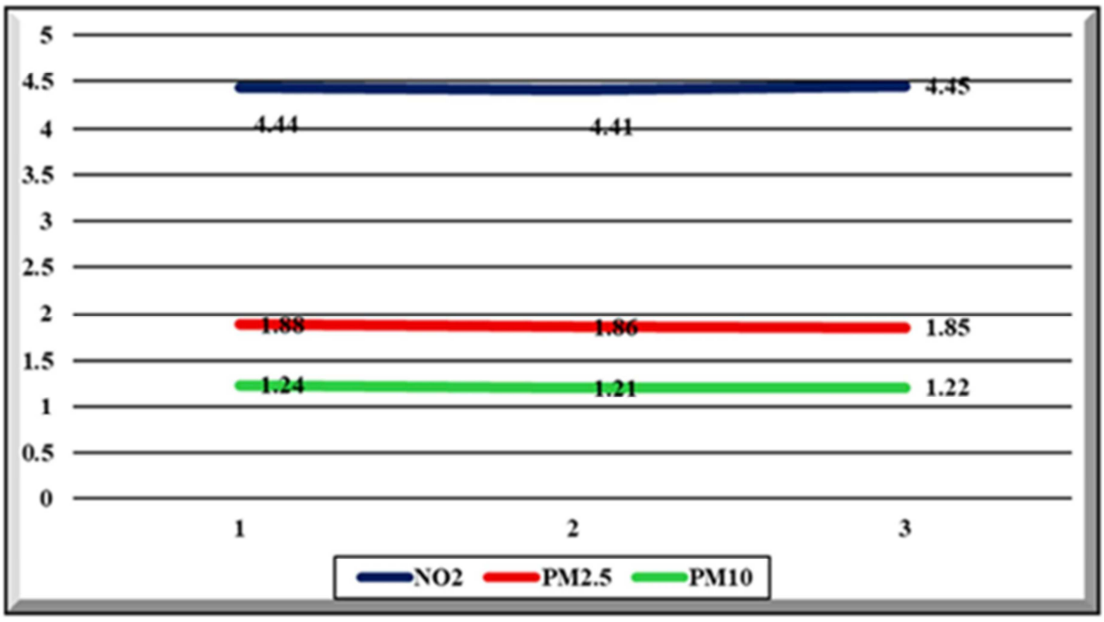

Figure 2: Shows (spring) the average of PM2.5, PM10 and NO_2

3.2. By analyzing Blood samples, it was as follow

Table (3) illustrate there is a significant difference at level less than (0.01) between (control) and (case) of Creatine Kinase (ck-MB) where (Z) value was (4.903) which significant at level less than (0.01) the mean of (control) was (19.05) and the mean of (case) was (39.35). This means as indoor air pollution increases the risk to cardiovascular diseases increases with the increase of creatine kinase mb and the risk of myocardial enzyme elevation as air pollution exists while in control samples to those who are not exposed to indoor air pollution the level of creatine kinase mb remains the same. Results suggest that occupational exposure to NO_2, $PM_{2.5}$ and PM_{10} is associated with increased risk of CK-MB elevation reaction that in healthy individuals is catalyzed by creatine kinase.For a long time, the primary physiological function of CK was thought to be the preservation of energy balance in areas with high energy turnover, including rapidly contracting skeletal muscle cells. The idea that fosfocreatine had several purposes in skeletal muscle was sparked by the finding that distinct cellular sites contained creatine kinase isoenzymes. Maintaining steady levels of ATP and ADP protected the cell from the quick depletion of ATP, which was one of these roles. Since different isoenzymes are associated with sites of ATP production and consumption, acting as a transport mechanism for high energy phosphates, the discovery of the mitochondrial isozymes demonstrated that CK was located in different compartments, leading to the development of the concept of a creatine phosphocreatine shuttle, which is a system power transmission between the local

production of ATP (mitochondria) and the place of use (generally the myofibrils). As a result, when creatine kinase increases, the opposite would occur in workers exposed to air pollution [21]. This result agreed with [22,23] in that occupational exposure to air pollutants cause elevation of creatine kinase. When CK is highly expressed in excitable tissues that require large energy fluxes and plays a significant role in the energy homeostasis of these tissue cells that causes cardiovascular diseases.

Table 3: Mann-Whitney Test to illustrate the difference between samples of Creatine Kinase (ck-MB) (n=20)

Variables	Mean ± SD		Mean Rank		Sum of Ranks		Z	P-value
	Control	Case	Control	Case	Control	Case		
Creatine Kinase (ck-MB)	19.05 ±3.68	39.35± 11.43	11.45	29.55	229.00	591.00	4.903	HS

HS: Highly significant at level less than (0.01)

Table (4) illustrate there is a significant difference at level less than (0.01) between (control) and (case) of Tumour necrosis alpha factor TNf-ALPHA where (Z) value was (2.670) which significant at level less than (0.01) the mean of (control) was (6.05) and the mean of (case) was (9.30). This means as indoor air pollution increases the risk to inflammation that causes cardiovascular disease as tumor necrosis factor alpha increases when workers exposed to indoor air pollution (NO_2, $PM_{2.5}$ and PM_{10})while humans that are not exposed to this air pollution the levels of tumor necrosis alpha are normal. One of the main contributing factors to the development of metabolic syndrome symptoms, such as dyslipidemia and impaired glucose tolerance, is inflammation. These metabolic alterations serve as the foundation for the development of insulin resistance and atherosclerotic plaque later on. TNF-a appears to be an important component in the pathophysiology of these disorders among inflammatory indicators. Acute circulatory TNF-a-increasing situations benefit the host from lipid changes; however, chronic TNF-induced lipid alterations raise the risk of cardiovascular disease, stroke, and the ensuing morbidity and mortality.

This was consistent with the minor elevation of diastolic blood pressure that was noted, and endothelial dysfunction does not provide a significant risk to healthy individuals. However, it can cause ischemic events in those who are vulnerable by aggravating pre-existing atherosclerotic plaque instability and/or by reducing myocardial perfusion. The correlations between $PM_{2.5}$ and exacerbations of heart failure and strokes may also be explained by the rise in blood pressure. Furthermore, it is highly likely that patients with cardiovascular disease or risk factors who already have an endothelial dysfunction or ANS imbalance that reduces the effectiveness of counterbalancing mechanisms will exhibit an exacerbated version of these responses. For instance, it has been demonstrated that individuals with hypertension respond to ambient $PM_{2.5}$ exposures with a higher rise in blood pressure than do normotensives.

Compared to healthy individuals, diabetics are more susceptible to endothelial dysfunction caused by air pollution. These results corroborate reports that susceptible people with pre-existing heart disease are at far higher acute risk from $PM_{2.5}$ [24].

Table 4: Mann-Whitney Test to illustrate the difference between samples of Tumour necrosis alpha factor TNf-ALPHA (n=20)

Variables	Mean ± SD		Mean Rank		Sum of Ranks		Z	P-value
	Control	Case	Control	Case	Control	Case		
Tumour necrosis alpha factor TNf-ALPHA	6.05±3.18	9.30±4.18	15.58	25.43	311.50	508.50	2.670	HS

HS: Highly significant at level less than (0.01)

Table (5) illustrate there is a significant difference at level less than (0.01) between (control) and (case) of catalase test where (Z) value was (4.291) which significant at level less than (0.01) the mean of (control) was (21.65) and the mean of (case) was (16.15). When the Twenty workers were exposed to indoor air pollution, the level of catalase enzyme decreased. While the control cases the level of catalase enzyme remained at normal levels .this means that when humans exposes to air pollution oxidative stress increases.

This indicates that while workers exposed to indoor air pollution have lower levels of catalase enzyme (the enzyme that catalyzes H $_2O_2$ Decomposition to oxygen and water), those who are not exposed to such air pollution have normal levels of catalase enzyme. Indoor air pollution increases the risk of oxidative stress, which causes cardiovascular disease. As a consequence of aerobic respiration, humans produce H $_2O_2$, which requires catalase to maintain redox balance. Moreover, catalase can: (a) function as an oxidase; (b) generate ROS in the presence of UV light; (c) lower free NADPH to avoid inactivation; and (d) be controlled by ROS, reactive nitrogen species, and even H_2S through post-translational modification. Reduced catalase results in an increase in free radicals, which can lead to heart attacks and stroke.

This agreed with **[25]** when workers exposed to indoor air pollution, the mechanism as follows .Additionally, **our** findings concurred **[25]**. Control typical twenty cases, catalase functions normally in the following manner: Using a ping-pong mechanism, catalase carries out the dismutation reaction of H_2O_2 in two steps. A molecule of H_2O_2 oxidizes the enzyme's heme iron group in the first stage, producing an oxyferryl group and a porphyrin cationic radical [Por- FeIV= O], while a molecule of H_2O is released into the medium. Compound I refers to this intermediate state of catalase. In the second stage, a second H_2O_2 molecule lowers Compound I, bringing the enzyme back to its starting point while also producing a second H_2O and an O $_2$ molecule. Catalase is a special enzyme in terms of its kinetics since it deviates from the Michaelis-Menten model's typical scheme. First, under experimental conditions, the enzyme cannot be saturated by substrate concentration. Second, catalase's own substrate, H_2O_2, has the ability to inactivate it (a process known as suicide substrate inactivation). In particular, compound I can undergo a one-electron reaction to become compound II [Por - FeIV= O], which can subsequently undergo a two-electron reaction with H_2O_2 to become compound III.Catalase can be inactivated either irreversibly or reversibly by these one- or two-electron processes.

$$\text{Catalase} + H_2O_2 \xrightarrow{k_I} \text{Compound I} + H_2O \quad \text{(R.1)}$$

$$\text{Compound I} + H_2O_2 \xrightarrow{k_{II}} \text{Catalase} + H_2O + O_2. \quad \text{(R.2)}$$

$$\text{Compound I} + e^- \rightarrow \text{Compound II}\,(inactive) \quad \text{(R.3)}$$

$$\text{Compound II} + e^- \rightarrow \text{Catalase}\,(active) \quad \text{(R.4)}$$

$$\text{Compound II} + H_2O_2 \rightarrow \text{Compound III}\,(inactive). \quad \text{(R.5)}$$

Table 5: Mann-Whitney Test to illustrate the difference between samples of catalase test (n=20)

Variables	Mean ± SD		Mean Rank		Sum of Ranks		Z	P-value
	Control	Case	Control	Case	Control	Case	4.291	HS
Catalase test	21.90±2.15	16.15±3.34	28.35	12.65	567.0	253.0		

HS: Highly significant at level less than (0.01)

4. Conclusion

In summary, indoor air pollution has a major role in the development of human diseases. There is a robust association between air pollution and atherosclerotic cardiovascular diseases, including myocardial infarction and stroke. Short exposure to NO_2 and particulates ($PM_{2.5}$, PM_{10}) may increase the risk of heart attack and stroke, increase hospitalization, and raise death rates. Stroke and cardiovascular diseases remain one of the leading causes of morbidity and mortality worldwide. Mitigation measures must be taken to reduce the concentration of indoor air pollutants in the investigated site (that factory) in order not to exceed in the following years. Such reduction may be achieved by using suction fans to increase air flow rates in the workshop to keep pollutants far from the standards.

Ethical consideration

The Study was approved by Research Ethical Committee, Faculty of post graduate childhood studies, Ain Shams University, starting

from14/10/2021 and for one year during which the Human blood samples were taken and analyzed. ApprovalNo.:FRGCSASUREC/RHDIRB2020110401/MSDFC-3

5. Conflicts of interest

There are no conflicts to declare.

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