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Article

Genotoxicity evaluation in workers occupationally exposed to the pollutants of Dhi- Qar oil refinery

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ABSTRACT
Background: Workers are occupationally and environmentally exposed to diverse forms of xenobiotics as gases and vapors. Career publicity to those compounds can trade the prooxidants/oxidants and antioxidants equilibrium throughout oxidative toxic stress (OTS). Pollutants might also cause genotoxicity by oxidative DNA. This work was conducted in AL-Nasiriya at the Thi-Qar Oil refinery in Thi-Qar province, south of Iraq. Samples have been amassed from the refinery workers and Campus college students and workforce as control. Seventy blood samples from the refinery people running within the asphalt, laboratory, and refining units and samples from Campus students and staff as control have been collected. The 8- OHDG was measured using the ELISA technique. The serum 8-OHdG of refinery workers was notably higher than that of individuals of control subjects ($p< 0.001$*). A higher level of serum 8-OHdG was quantified in the blood samples of all exposed subjects. The significant feature that resulted in an enhanced genotoxic potential was the years of exposure to the oil refinery environment, which led to an increase in the 8-OHdG formation.

Keywords: Genotoxicity ; workers occupationally ; pollutants ; Dhi- Qar ; oil refinery

Introduction

Refinery Environmental

Rapid international populace expansion, mixed with a choice for material abundance, has resulted in commercial and agricultural productivity advances over many years. The multiplied energy usage and waste production that has resulted has positioned a prime stress at the natural environment, causing adjustments inside the composition of the ecosystem, land, and freshwater assets [1]

Chemical pollution from refineries is widespread, affecting workers and the ecosystem [2].

Petroleum is a naturally oily, flammable liquid made in large part of hydrocarbons obtained with the aid of digging wells underneath the surface of the Earth. Crude oil is the term for unrefined petroleum, initially called rock oil [3.]

Refinery operations had been connected to a spread of air pollution, inclusive of criterion air pollutants, risky natural compounds, unsafe air pollution, and other pollutants [4]

Other pollutants resulting from oil refineries include heat and noise pollution [5].

Petroleum Mixture

Petroleum is defined as a complex combination of domain names of compounds in carbon range from C6 to C60 that implements aliphatic hydrocarbons (n-alkanes, iso-alkanes, and cycloalkanes), polycyclic aromatic hydrocarbons (PAHs), and derivatives of those are alkylated, and compounds of polar which can be typically made up of compounds for heterocyclic (S, N, and O) have aromatics [6]

Due to refinery emissions, there are more excellent severe pollution standards (particulate rely on PM and sulfur dioxide) and unsafe air pollutants (polycyclic aromatic hydrocarbons PAH) [7].

Human activities, including fossil fuel burning and manufacturing operations, are the ecosystem's number one source of SO2 (e.g., oil refineries, coal combustion, and biomass)[8].

The gases released by way of oil companies are labeled into three corporations: Suffocating gases (H2, CH4, CO2) soak up areas in the working environment's air, lowering the percentage of oxygen, disturbing gases (Cl2, F) cause infection and irritation in each skin and breathing tissues, resulting in tissue erosion and cell death, and toxic gases (Cl2, F) cause infection and irritation in both pores and skin and respiration tissues, ensuing in mobile dying. Pb, Cu, Ni, Cr, Cd, S, P, and CCl4 are some heavy metals and other compounds that cause persistent poisoning in workers because of their exposure to them within the place of work dust, smoke, or steam vapors. Harm is caused both via inhalation or accumulation [9].

Heavy metals are the most accomplishable pollutants within the surroundings of an oil refinery. Steel particle inhalation may be more harmful than different pathways, including the digestive system or the skin [10].

Physical and chemical agents produced by way of human activity might also have A ramification of results on human beings, even as certain chemical substances do not purpose harm, others may be accountable(if publicity is high sufficient) for impairing easy strategies such as boom and development, environmental situations may frequently have a helpful effect or prepathological, on this regard, the presence of those hazards in distinctive devices of the refinery has to be taken extraordinarily serious [11].

Occupational & Health Hazard of Refiner:

In the workplace, millions of people are exposed to dangerous chemicals, dust, or fibers(Abed et al., 2015). Benzene, heavy metals, and mineral fibers have all been related to a higher chance of developing various illnesses at work. Some of these chemicals may cause genetic changes in exposed employees' somatic or germ cells [12,14]

Work is such an essential element of a person's life that it must be safely provided to them. Workers are nevertheless exposed to industrial dangers, such as hazards and chemical poisons [15].

Polycyclic aromatic hydrocarbons (PAH) cause mutations and

Cellular inflammation in the cardiovascular and respiratory systems,

Posing a risk to those who are exposed. Skin, bladder, lungs, and gastrointestinal tract cancers have become more common among exposure workers [16,19]. Air pollution is one of the leading causes of death in humans, but it also threatens food security, the economy, and the ecosystem. Human exposure to lead can have various biological consequences, including toxic effects on the hematological, cardiovascular, neurological, and reproductive systems [20,22]. Long-term coke dust, silica, and hydrogen sulfide

exposure can lead to chronic lung disease. Lead alkyls used as gasoline additives can cause psychosis and peripheral neuropathies. Fine and ultrafine PM have a higher chemical composition and toxicity [23]. Particulate matter (PM) in outdoor air pollution was classified as a Group I carcinogen by the International Agency

for Research on Cancer (IARC). Sulfur dioxide causes respiratory irritation, bronchoconstriction, and the potential for respiratory and pulmonary alterations, as well as cardiovascular disorders in humans and animals (23-26). Polycyclic aromatic hydrocarbons (PAHs) are considered a priority by the United States Environmental Protection Agency (USEPA) is Carcinogenic and mutagenic for humans and are released chiefly from anthropogenic sources [27]. Several studies have also discovered significant links between SO2and mortality [28]. The major routes of exposure, especially for occupationally exposed subjects, are inhalation and ingestion. Lead is taken into the bloodstream and accumulates in the bones and soft tissues, especially the liver and kidney. As a result, guide poisoning is extremely dangerous to these organs [29].

Free Radical and Oxidative stress

Oxygen is a necessary component of life. Because of the mitochondria's synthesis of ATP (adenosine triphosphate), they produce free radicals when cells use oxygen to cause energy [30]. When a molecule is attacked by a free radical (FR) in the cell, it loses an electron. It becomes another free radical [31] because FR is an atom or molecule containing one or more unpaired electrons in a valence shell or an outer orbital and the ability to exist independently [32]. Reactive oxygen species(ROS) can be created from different sources. Reactive oxygen species from endogenous sources, including various cellular organs such as mitochondria, peroxisomes, and endoplasmic reticulum, consumed much oxygen [33].

Oxidative stress is created when there is an imbalance between the systemic expression of reactive oxygen species (ROS) and a biological system's ability to detoxify reactive intermediates or repair the harm they cause 34 quickly.

Air Pollution Exposure and Induce Oxidative Stress Xenobiotics' environmentally and occupationally exposure may increase oxidative toxic stress (OTS), which can change the balance of Oxidants /prooxidants and antioxidants [35]. Modifications in oxidative stress were the first pathophysiologic

mechanism identified in humans and animal models in response to air pollution exposure [36]. Evidence suggests that chemical exposure in the workplace is mediated through oxidant signaling pathways [37].

Oxidative toxic stress is described as the excessive exposure of a molecule, cell, or tissue to oxidants, particularly free radicals like superoxide or hydroxyl radicals, generally called ROS [38]. Metals can generate free radicals directly or indirectly through various mechanisms [36].

Free Radical, Oxidative Stress and Effects Health

Increased cardiovascular and pulmonary morbidity and death are linked to air pollution. Oxidative stress can trigger redox-sensitive pathways that lead to inflammation and cell death [39]. Oxidative damage might result from excessive production of ROS. Increased concentrations of ROS and/or RNS induce structural alterations in nucleic acids, proteins, and lipids [40]. When the antioxidant systems are depleted in diseased situations, Cancer, neurological disorders, atherosclerosis, hypertension, ischemia/perfusion, diabetes, acute respiratory distress syndrome, idiopathic pulmonary fibrosis, chronic obstructive pulmonary disease, and asthma are all associated with OS and typical aging are all diseases linked to redox imbalance [41]. The accumulation of ROS/RNS can lead to lipid peroxidation, protein oxidation, and DNA damage [42].

Effects of Oxidative Stress and Pollution on Different Immune Cell Types Innate immune cells in the lungs act as 'first responders,' responding quickly to inhaled pathogens. When they contact pathogens, they can activate complicated adaptive immune responses [43].

In addition to being airborne pollutants (PAHs), particulate matter (PM) and its constituents (organic carbon (OC), elemental carbon (EC), total carbon (TC), and

polycyclic aromatic hydrocarbons) all play a significant role in the environment in promoting the development of allergic sensitization and disease [44].

As a result of detecting phagocytosing inhaled PM, macrophages get activated and produce a variety of cytokines, including TNF-α, IL-6, IL-12, and IL-10 [45]. Eight-Hydroxydeoxyguanosine(8-OHG) Markers for Nucleic Acids Damage. The products of petroleum and its fumes created by the combustion of organic matter can have toxic and carcinogenic things. The International Agency for Research on Cancer (IARC) has included it in group 1 [46]. Hydrocarbon aromatic chemicals can produce ROS in the body, which interacts with DNA and causes DNA damage [47]. Long-term exposure to low quantities of petroleum products such as benzene may reduce antioxidant capacity and increase the risk of DNA damage in refinery workers [48]. PM2.5 can cause genotoxic DNA damage and epigenetic modification in direct and indirect ways [15]. The direct action is usually linked to a genotoxic substance interacting with DNA and causing damage [22]. Hydroxyl radical (HO•) is the most essential oxygen freer radicals causing damage to DNA, which attacks DNA strands and leads to the addition of DNA bases and new radicals that form a diversity of oxidation products [5]. The interlinkage of HO• with nucleobases of the DNA strand, such as guanine, leads to the formation of 8-hydroxy2'-deoxyguanosine due to this reaction [19]. The oxidized metabolite 8-hydroxy-2-deoxyguanosine (8-OHdG) is one of the most researched oxidized metabolites and is a biomarker for DNA oxidative damage [32]. Excessive concentrations of 8-OHdG have been linked to disorders like cardiovascular disease and chronic obstructive pulmonary disease (COPD) (26), as seen in Figure(2-4). Smoking, aging, or occupational exposure to physical, chemical, or biological substances lead to an increase in 8-OHG levels [29]

Aims of study

Find out whether pollutants in the oil refinery affect Nucleic Acids.

Materials and Methods

This study was conducted in AL-Nasiriyah at the Thi-Qar Oil Refinery in Thi-Qar province, south of Iraq. From November 2020 to April 2021, samples were collected from the refinery workers and Campus students and staff as control. Seventy blood samples from the refinery workers working in the asphalt, laboratory, and refining units, and 20 samplesCampus students and staff as control have been collected. Where a butterfly has drawn venous blood of 8 ml from both two groups [23]. A blood sample (5 ml) was put in a Gel tube and left to clot for half an hour to get the serum. This serum has been used to assess the level of 8-OHdG by ELISA assay.

Results

Comparison between the Exposed Workers at the refinery and non-exposed Subjects according to genotoxicity indicator. The current study data show a significant increase in the 8-OHG mean of exposed employees in comparison with non-exposed subjects (70.49±21.41 vs. 52.54±11.27) (p=0.01*) at (p≤0.05) as shown in Table (1).

Parameter	Workers (N=70), Mean±SD	Control (N=30), Mean±SD	P.Value
8-Ohd	70.49 ± 21.41	52.54 ± 11.27	**0.001***

Table 1. Correlation assessment between the age and service with 8-OHG.

df=128 ,*p.value ≤0.05 Significant

Comparison of the Exposed Workers in refinery and Non-exposed according to immunological indicators

The current study's data show a significant increase in the mean of IL-6 among exposed workers compared to the control (53.18±15.15 vs 16.55±7.43) (p=0.00). On the other hand, there is a significant increase in the mean of IL-10 of exposed workers compared to non-exposed subjects (50.55±16.75vs vs. 26.49±11.04) (p=0.00).

Parameter	Workers (N=70), Mean±SD	Control (N=30), Mean±SD	t.value	P.Value
IL-6	53.18 ± 15.15	16.55 ± 7.43	8.67	**0.00***
IL-10	50.55 ± 16.75	26.49 ± 11.04	5.63	**0.00***

Table 2. Comparison between the Exposed Workers and Non-exposed according to immunological indicators.

Correlation assessment between the age and service with 8-OHG, IL-6, and IL-10

The current study data show a positive correlation of 8-OHG with age and service. There is a positive correlation of IL-10 with age and service. There is a negative correlation between IL-6 with age and service, as shown in Table 3.

	Age	Service	8-Ohd	IL-10	IL-6
Age	1.000				
Service	.720**	1.000			
8-OHdG	-0.029	-0.131	1.000		
IL-10	0.124	.271*	-0.832**	1.00	
IL-6	-0.059	-0.198	0.946**	-0.903**	1.00

****. Correlation is significant at the 0.01 level (2-tailed).**

***. Correlation is significant at the 0.05 level (2-tailed).**

Table 3. Correlation assessment between the age and service with 8-OHG, IL-6, IL-10.

Discussion

Data from the current study demonstrated an increase of 8-OHdG exposed workers compared to subject control [22]. It may be due to exposure to pollutants and increased oxidation [43].

Although the carcinogenic mechanism is unknown, petroleum vapors appear to be implicated in generating ROS and electrophilic intermediates that can induce DNA breakage, adduct formation, and the oxidation of nucleic acid bases through their metabolites [32]. The consequences of this work show that workers at refineries exposed to modest doses of pollutants suffered from oxidative DNA damage. The idea that enzymatic bioactivation of benzene plays a substantial role in benzene-initiated toxicity through oxidative DNA damage is widely accepted [44]. The construction of free radicals can be induced by benzene metabolism, producing a prooxidant/antioxidant imbalance and oxidative stress [36]. Oxidative stress disrupts intracellular metabolic processes that result in changes such as DNA oxidation and subsequent long-term modifications. Agreement with numerous studies [44]. In this work, the products of oxidation of DNA damage were found, introduced in the serum as 8-OHdG. DNA damage has been shown to grow with age as repair mechanisms become less effective, where the correlation was stronger according to years of work seniority. The result is in agreement with previous works [23]. The current study shows a significant increase

in IL-6 & IL-10 in exposed workers compared to controls. The results were consistent with [22].

Our data show that exposure to particulate matter causes the production of IL-6. The goal of this study, which used IL-6 as a surrogate marker, was to see if the lungs are a significant source of systemic inflammatory mediators through pollutant inhalation. These findings back our theory that PM10 exposure raises pulmonary inflammatory mediators, which circulate in the bloodstream, contributing to systemic inflammation [46]. Inhibition phagocytosis of apoptotic cells by macrophages increases the potential for an inflammatory response by a decrease that the dying cells do not eliminate before their intracellular contents are released, which leads to increased cytokines level [19]. In addition, the study supposes activated transcription factors such as NF-κB by exposure increase that leads to OS [11]. Chapter Five Discussion 77 Increase IL-6 and decrease IL-10 of exposed workers compared with control subjects; it may be due to Th-1 suppressor and altered immune response toward Th-2 [38]. There is clear evidence that cytokines influence can modulate prooxidant and antioxidant activities [13]. Thus, it is reasonable to infer that hematological cancers arise due to a genotoxic operation or stimulation of immunosuppression. Furthermore, acquired epigenetic alteration may contribute to the development of leukemia, as pollutants in the refinery alter nuclear receptors and cause protein modifications, altering the activity of regulatory proteins such as oncoproteins, tumor suppressor proteins, and transcription factors [25]. Thus, stricter rules and requirements may be required to safeguard the evolving professional population.

Conclusions

The higher serum of 8-HdG concentration in workers exposed compared to the control group appears to reflect DNA oxidative increase at some stage in occupational exposure to pollutants in oil refineries.

Compliance with Ethical Standards statements

Ethical approval:

The manuscript is written in original, and all the data results about this manuscript are original according to the research performed. The authors followed academic integrity and did not copy any content/results from another source.

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V. All the authors of this study contributed equally in terms of performing the research and preparing the manuscript. All the authors of the study followed the guidelines of the corresponding author. Any query/suggestion related to the manuscript can be sent to the corresponding author

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