

Effect of exposure to petroleum products



Petroleum-derived products are chemicals (mostly volatile) that primarily used for internal combustion machines. These products are made up of hydrocarbon and non-hydrocarbon components; hydrocarbons consist of (aromatic, saturated and unsaturated) and non-hydrocarbons consist of (N, S, O₂, vanadium and nickel)(179, 180). Some individuals ought to have a bigger risk of revelation to gasoline fumes; those individuals may include gasoline trucks drivers, Petroleum refining factory workforce, filling-station staff and service station attendants, (181). In the present study, table 3-1 demonstrates that, the exposure of oil refineries working staff to petroleum products for long period of time may significantly elevates serum levels of copper, iron, and lead equated to control standards. It is well known that impairment of metal homeostasis in the biological system can be considered as a risk factor for many health disorders. In this regard, chronic elevation of free iron in biological fluids may lead to diabetes, cardiomyocytes damage, impaired sexual drive, liver cirrhosis, and kidney diseases. Meanwhile, free iron-induced tissue damage might be irreversible, and should be seriously considered by health care professionals due to the high risk of establishing many types of pathological disorders(182). In addition to the release from endogenous storage sites, heavy metals along with other pollutants are discharged to the environment through many industrial activities; during most of these activities, wastes are generated and discharged into the environment in volatile forms(181). The copper content in the human body represents an essential part of various significant enzymes that takes part in vigorous biological processes. Free form release can be done to catalyze ROS formation despite it is generally proteins bounded, including the most powerful and dangerous radicals of hydroxyl. Many cell culture and *in vitro*

studies collected data, support the idea that free form of Cu initiates oxidative stress, and interferes with important many cellular events(183). Moreover, exposure to exogenous sources and/or liberation of free copper from endogenous sources facilitates the rate of Fenton reactions that excessively generate free radicals(184). For a variety of diseases this is considered as a dynamic research zone, in which copper is playing a significant but yet extra delicate part than in acute toxicity(185). Lead, among the metals that elevated in the plasma of oil refineries workers, is the most dangerous one and massive exposure can lead to fatal consequences. In the present study, the mean blood level of lead is significantly elevated in exposed workers compared with that reported in control subjects.

The pathogenesis of lead poisoning is contributed by lead-induced oxidative stress, and increases liberation of ROS through different heterogeneous mechanisms, which may facilitate tissue damage. Among the mechanisms through which Lead induce tissue damage, is the damage of DNA and other proteins and enzymes that have critical roles in body homeostasis. Several target places such as: liver, brain, blood vessels, lung, sperm, and testes can be caused by different pathological conditions related to lead-induced oxidative stress (186). Since lead is one of the components of the petroleum products, especially automobile fuel, blood lead levels are the best indicator of lead exposure impact in the oil refinery and petrol station workers. Many chemicals and additive in Petroleum products however, anyone of the composition could be the cause for such deterioration in the homeostasis of certain metals. Accordingly, intervention may be improved to prevent or at least delay minor impairment development to fatal levels of lead and/or the

free forms of iron and copper in the occupational setting. This could be done by the identification of precise product and the categorization of risk exposed job . Moreover, reports regarding hazards in oil refineries may arise in different ways, such as: biological, psychological chemical, physical, and non-application of ergonomic principles, etc. Worldwide diseases and work-related hazards remain to be serious problems, due to the large number of hazards in such workplaces and the absence of consideration to health and safety measures to numerous workers. (187).

Although the urinary tract system is viewed as a target of heavy metal toxicity, the study showed that there is no obvious significant alteration in kidney function markers. In the present study, table 3-2 demonstrates a slight elevation in serum urea and microalbuminuria levels in the exposed personnel in comparison to non-exposed healthy individuals; although serum creatinine levels has not affected at all. Petroleum derivatives consist of aliphatic and aromatic hydrocarbons generally gasoline interrelated , these products are mostly toxic to the body organs and specially the kidney(188, 189), that could be referred to the risen liberated toxic metabolites, that includes reactive oxygen species. Although animal experimentations stated that inhaling the aromatic hydrocarbons toluene, styrene, and xylene was nephrotoxic(190), however, in humans it has not been verified(191). Both human and trial readings propose that the kidney may be affected by several chemicals (192). Of these chemicals, organic solvents' role in chronic kidney ailments, mainly chronic glomerulonephritis, has been debated for a long time (193). Moreover, the lead content of the volatile petroleum products can induce many enzymatic systems in the body; most of them liberate ROS

in many organs and tissues including the kidney and the cardiovascular tissues(194). Development in additional researches in this field was resulted by the acknowledgment that industrial and ecological causes may damage the renal functions. In the present study, the biomarkers of renal function (urea, creatinine and microalbuminuria) are not importantly high in oil refinery personnel; nevertheless, while they might be slightly raised, and still within standard figures, an inclination could be signified in the direction of initiation of renal diseases. Within the same context, former research has indicated that petroleum products exposition could have damaging impact on kidney functions(195, 196). Stengel *et al* ., based on a case control study, does not agree on the effect of petroleum exposure in occurrence of glomerulonephritis; but proposed a role in the progression to end stage renal disease(197). Jacob *et al* . indicated that while using an suitable cohort study design, exposition to petroleum products for long period is connected, with faster progress to end-stage renal ailment, in patients with IgA and membranous glomerulonephritis(198).

Regarding the liver function markers, the present study demonstrates a significant increase in the activities of ALT, AST, and ALKP in the serum of refinery workers, compared to that reported in non-exposed subjects. This result supports the possibility of the existence of liver damage induced by the long-term exposure to petroleum products. Some of the liver enzymes existed in the serum in quite low concentration. These enzymes' function in the serum is to offer hepatic functions information and the possibility of damage in hepatic tissues.

Raise of AST and ALT beside the increase in ALP action could imitate inflammatory diseases or liver injury. In this study, the maximum activity of ALKP found was very high, suggesting the likelihood of hepatic cell injury. Serum enzyme activity patterns reflect the organ's physiological state, as some investigators stated. For example the AST, ALT and ALKP activities rise in serum levels, was noticed in serum of mice inhaled petroleum vapors(199). Other studies indicate a rise in hepatic enzymes activities following albino mouse liver injury exposed to different types of toxic substances(200, 201). Moreover, albino rats serum and liver showed biochemical changes for rats inveterately exposed to gasoline, kerosene and crude petroleum revealed a dose-dependent increase in the activities of liver enzymes of rats(202). However, the Total and conjugated serum bilirubin remains unaffected. The present study results in tune with the findings. Evidence from the experimental data indicates that crude petroleum and/or its refined products could induce hepatic damage, and when exposure becomes high and chronic, it could render the liver dysfunctional.

The current study indicates that there is no significant effect of exposure to petroleum products on the lipid profile markers. Besides, the serum glucose level is non-significantly affected.

The present study reveals a weak significant decrease ($P= 0.04$) in the total antioxidant status (TAS) in the serum of oil refinery workers, compared with non-exposed subjects. Although exposure to the fumes of oil processing is considered as a powerful initiator of ROS generation, the studied workers show a very small remarkable decrease in this regard. This might be attributed to the effective compensatory mechanisms, which are still

function effectively in many of the workers included in the study.

Antioxidants, such as GSH, uric acid, ascorbate and α -tocopherol present in epithelial lining fluid could keep the tissues from oxidant damage made by air pollutants exposition including petroleum vapors(203). It has been reported that under normal physiological conditions, the biological system maintains a balance between generation and neutralization of ROS(204). However, when the biological system exposed to petroleum products, the rate of ROS production cells get increased, and depletion of antioxidant reserves may occur when compensation is not enough(205).

Our study shows a statistically significant reduction in all the measured parameters of lung function (except FEV1/FVC) of the oil refinery workers compared with the non-exposed subjects. This finding is in tune with that reported by others. An animal study has indicated that inflammation and/or injury of the airways can be resulted from diesel exhaust waste that establishes a significant fraction of particulate air pollution (206). The peak expiratory flow, forced expiratory mean flow, and forced vital capacity are significantly reduced in animals exposed to gasoline exhaust fumes. The existence of SO₂ is possibly behind respiratory impairment and the quality of the hydrocarbons in gasoline exhaust gases(207). Moreover, exposure to petroleum hydrocarbons significantly induces lipid peroxidation with a consequent increased MDA levels, and a decrease in SOD and CAT activities and GSH level. Such state of oxidative stress, due to exposure to petroleum hydrocarbons, also causes alteration in the histomorphology of lung tissues, and inhaled petroleum hydrocarbons are potential risk factor in the pathophysiology of pulmonary dysfunction(208). Histomorphologic

research stated that edema and hemorrhagic necrosis of the lung alveoli and parenchyma were caused by petroleum hydrocarbons.

The outcome of the effect of exposure to petroleum products on hematocrit and RBC count appears to be importantly diverse when matched to that of control group. The toxic ingredients, in petroleum vapors, have been testified to alternate the composition of blood. Most of subjects exposed, for longer than two years, to petroleum products had significantly lower figures of hematocrit, hemoglobin concentration, and red blood cell count than subjects for less than two years exposition. Subjects would become anemic progressively on exposure to petroleum fumes. These results indicate that the petroleum fumes cause a reduction in hematological indices, which worsens with prolonged exposure(209). The principal limitations of the current study, including small sample size, does not enable perfect prediction of the hematological changes in the oil refinery workers included in the study.

Moreover, the present study demonstrates significant elevation in serum interleukin- β 1 in the exposed workers compared with non-exposed subjects. Interleukin-1 β , also known as catabolin, is a mediator related to the family of cytokines . When the macrophage is activated, This cytokine is released as a proprotein, which is converted by proteolytic enzyme to its active form. This cytokine is considered a significant arbitrator of the inflammatory reaction, and plays a significant part in a diverse cellular events, such as cell distinction, proliferation, and apoptosis(210). Subjects with chronic obstructive pulmonary disease(COPD) and asthma , demonstrate high level of the inflammatory cytokine interleukin (IL)-1. The highly expression of IL-1 β

induced in the lungs of adult mice lead to pulmonary inflammation distinguished by neutrophil and macrophage infiltrates. IL-1 β affect distal airspace and cause swelling, consistent with emphysema. IL-1 β destroyed elastin fibers in alveolar septa and cause fibrosis in the pleura and airway walls. The thickness of conducting airways is elevated by IL-1 β , that lead to production of mucine, which in turn aggregates lymphocyte in the airways(211). Moreover, IL-1 β has great influence in the induction of murine emphysema and small airway remodeling. It is compared to TNF- α in this effect(212).

Conclusions:

Workers exposed to petroleum products for long-term are placed at oil refinery industry at continuous risk of development of defective organs' functions which necessitate a frequent medical examinations and application of suitable measures to prevent serious effects on different body organs of workers and employee at oil refinery industry.

Recommendations for Future Work:

- From my perspective , I do recommend people who work in oil refinery industry should take annual classes that provide valuable information regarding health and safety measures to reduce the risk .
- Providing information and education about air quality monitoring in and around refineries.