

Impact of Airborne Pollutants Emitted from Cement

Plants on Liver Function: A Review

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Abstract

This literature review addressed the impact of air pollutants emitted from cement plants on liver functions, with a focus on the pathological mechanisms and the physiological and biochemical changes resulting from chronic exposure to industrial pollutants. Studies have shown that the cement industry is a significant source of air pollution due to the continuous emissions of fine particles, industrial dust, sulfur and nitrogen oxides, and heavy metals, which have serious health effects on humans. Recent research has indicated that the fine particles and heavy metals produced by cement plants can enter the body through the respiratory system, then move into the bloodstream and reach the liver, leading to multiple cellular and functional disorders. Studies also demonstrated a clear relationship between chronic exposure to cement dust and elevated liver enzymes such as ALT, AST, and ALP, in addition to increased indicators of inflammation and oxidative stress. Studies have also indicated that oxidative stress represents the primary mechanism in the occurrence of liver damage caused by air pollutants, as free radicals lead to damage to cell membranes and mitochondria and stimulate chronic inflammation within liver tissues. Some studies have also shown that long-term exposure to industrial pollutants may be associated with the occurrence of non-alcoholic fatty liver disease, liver fibrosis, and metabolic disorders. Professional studies have shown that workers in cement factories, especially in grinding, packing, and firing units, are most susceptible to health effects due to high levels of direct exposure to industrial dust and suspended particles. Therefore, this review emphasizes the importance of implementing environmental control measures, improving filtration and ventilation systems, and conducting regular medical examinations for workers to reduce health effects associated with industrial air pollutants.

Keywords: Pollution, cement plant, airborne, liver function test, ALT, AST, ALP.

INTRODUCTION

Pollution resulting from cement production is a prime source of uncontrolled emissions—including toxic aerosols—released into the atmosphere; processes involved in cement manufacturing generate environmentally harmful substances such as carbon dioxide and concrete dust [1,2]. Further highlighted the role of oxidative stress-induced cytotoxicity—stemming from exposure to fine particulate matter (PM)—in adverse health effects observed over several decades, particularly given the complex composition of the polluting gases and particles we inhale, Oxidative stress is often viewed as an integrative biological pathway to help bridge the causal gap between the initial molecular trigger and the adverse health outcome. Numerous purely chemical/abiotic models and in vitro models have been developed to estimate the ability of particles and gases to induce oxidative stress in living organisms. While this oversimplification of the in vivo situation has clearly been criticized, some useful readings have been obtained from population-level health effects [3]. Previous studies [4, 5] indicated that many of the observed effects of particles can involve either disrupting exogenous redox signaling or enhancing endogenous redox signaling of reactive oxygen species (ROS), leading to exaggerated responses. Endogenous ROS originates from various sources, such as the mitochondrial respiratory chain, NADPH oxidases (NOXs), nitric oxide synthesis (NOS), and cyclooxygenases, lipoxygenases, xanthine oxidase, and cytochromes P450. Other study [6] focuses on understanding the role of reactive biological mediators generated in response to air pollution exposure. Exposure to air pollutants adversely affects liver function in adults living in urban areas where ambient air pollution poses a serious public health issue; Notably, these adverse effects persist longer in high-pollution areas compared to low-pollution areas [7].

Effect of cement industry in air pollution

The rising rate of concrete recycling currently poses a risk to environmental health due to the damage caused by the release of pollutants into the environment, as well as factors involving economic considerations, public awareness, and legislation. Pollution associated with cement plants refers to their adverse environmental impacts; major pollutants resulting from the cement industry include radioactive dust, nitrogen oxides, hydrogen chloride, hydrogen fluoride, sulfur dioxide, organic compounds, dioxins, and carbon monoxide, furans and ammonia. Among the most common technologies for reducing combustion-generated pollutants are Selective Catalytic Reduction (SCR) systems—devices used to remove nitrogen

oxides—and electrostatic precipitators, which remove solid waste and aerosols; nevertheless, organic pollutants and other combustible compounds, such as carbon monoxide, can still be found in these emissions. Globally, concrete is the most widely used construction material, yet it is environmentally harmful and contributes significantly to greenhouse gas emissions during production; cement is a key component of concrete. It is the second-largest industrial source of carbon dioxide on Earth, and cement manufacturing is considered a major source of heavy metal pollution in the soil [2, 8, 9]. Furthermore, ambient air pollution affects blood metabolism [10].

Cement is manufactured from four primary ingredients: limestone, laterite, clay, and gypsum. The first three are obtained through quarrying operations; in most cases, the rock is broken down into small particles known as "quarry fines." Additionally, further processing is employed to convert these fine particles into a powdered form known as "quarry dust."

Cement is a dark gray, powdery substance composed of alumina, silica, lime, iron oxide, and magnesium oxide. Exposure to cement dust affects blood parameters, leading to adverse health effects [11, 12]. A study [11] recommended that management and workers in cement plants adopt preventive measures to minimize the inhalation of cement dust.

Gases emitted by cement manufacturing plants consistently contribute to global warming and climate change. These facilities employ energy-intensive production processes characterized by high fuel consumption to produce clinker, which in turn results in environmental emissions. Beyond fuel consumption, the calcination process exacerbates the generation of emissions such as nitrogen dioxide (NO₂), sulfur dioxide (SO₂), carbon dioxide (CO₂), and other particulate matter.

Cement is composed of various chemical substances—such as lime, silica, and others—that can cause health problems, particularly respiratory diseases; studies have indicated that ambient air pollution may lead to lung cancer [13,14,15]. A study [17] highlighted the environmental impact of cement production emissions. Furthermore, numerous research studies have shown that sub-Saharan African countries suffer the most from environmental pollution—particularly air pollution—with the behavioral patterns of workers and traders living near production plants identified as a contributing factor. The demand for cement, the availability of local raw materials, and the need for local content have led to an increase in the number of cement plants, exposing areas in and around these facilities to various air pollutants. These pollutants affect the lives and well-being of workers, children, and residents of nearby communities, as well as flora and fauna. Exposure to cement dust pollutants is also linked to

various health conditions, including chronic obstructive pulmonary disease (COPD), silicosis, preterm delivery, psychasthenia, endocrine disruption, cancer, and infertility. Additionally, other studies [8,22] have also highlighted the health risks associated with exposure to cement dust.

The relationship between oxidative stress and air pollution

Polluted air is a complex mixture of gases and particles derived from both human activities—such as transportation and industry—and natural sources, including windblown dust, sea salt spray, and volatile organic compound emissions from plants. This mixture includes solid and liquid particles known as Particulate Matter (PM)—airborne particles characterized by their aerodynamic diameter. Particles with an average diameter of less than 2.5 micrometers are designated as PM_{2.5}; those less than 10 micrometers are designated as PM₁₀; and those smaller than 100 nanometers are referred to as ultrafine particles or nanoparticles. These particles are structurally heterogeneous, exhibiting marked variations across different regions and exposure durations—whether over the course of a day or longer periods—reflecting changes in emission sources. Most studies highlight their adverse health effects and the strong link between short- or long-term exposure to air pollutants and a growing list of non-communicable diseases; furthermore, oxidative stress can induce cancer in brain cells and disrupt the expression of the N-methyl-D-aspartate (NMDA) receptor 1 protein, thereby contributing to the development of Alzheimer's disease [18, 19, 20].

The normal range for the AST enzyme is 13–31 IU/L, and it is considered a key indicator of liver function efficiency. Elevated levels suggest the presence of disease; very high concentrations are associated with viral hepatitis, severe hepatic congestion, and hemorrhage in the blood vessels supplying involuntary muscles. Conversely, more moderate elevations may indicate myocardial infarction or liver cirrhosis. While AST is similar to the ALT enzyme, it is less specific for diagnosing liver diseases because it is also produced by muscle tissue [21].

Conclusions

Elevated AST enzyme levels are observed in individuals residing near pollution sources—specifically cement plants—due to the impact of airborne pollutants emitted by these facilities. A slight rise in AST concentration serves as an indicator of hepatic metabolic dysfunction, fatty liver, and pancreatitis; furthermore, the AST enzyme plays a role in the metabolism of amino acids and urea, as well as in the tricarboxylic acid (TCA) cycle.

ETHICAL APPROVAL

The research protocol was approved by the Ethical Research Committee of the Kirkuk Education Department. We declare that the Helsinki Declaration had been followed in the study.

INFORMED CONSENT

Participants were aware of the purpose of the study and provided informed consent prior to the participations.

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