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Bridging Experimental and Community Air Pollution Research: Pulmonary Effects of Cement Dust, Vehicular Emissions, and Coal Heating

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ABSTRACT

Air pollution is the most important environmental risk factor for respiratory health globally. The most significant pollutants in the atmosphere related to respiratory morbidity and lung damage are vehicle exhaust gases, industrial dust particles, etc. Experimental studies are essential to the knowledge of direct pathological effects of various pollutants on the respiratory system in a controlled manner. This experimental study evaluated and compared the histological effects of cement dust and vehicle exhaust exposure on respiratory tissues in 30 male white outbred rats. Animals were assigned to control, cement dust, or exhaust exposure groups. Following four weeks of inhalation exposure, lung and bronchial tissues underwent histological examination using hematoxylin–eosin staining. Histological analysis showed that there were no pathological changes in the lungs of controls, bronchial texture was preserved. The results of exposure to exhaust gases were vascular congestion, thickening of interalveolar septa, pulmonary edema, bronchial spasm, desquamation of epithelium, infiltration of lymphoid cells, and focal atelectasis. Comparative analysis showed that vehicle exhaust primarily affected pulmonary microcirculation and bronchial structures, whereas cement dust mainly induced chronic inflammatory and ventilatory changes. Both exposures caused significant pathological alterations in the respiratory system. The main impact of exposure to exhaust gas was acute inflammatory and microcirculatory disorders, and exposure to cement dust was the main cause of chronic inflammatory disorders and impaired airways of lung tissue. The results obtained were found to corroborate the deleterious impact of atmospheric pollutants on respiratory function and the need for environmental protection measures in industrial and urban areas.

Keywords: *Air pollution, Exhaust gases, Cement dust, Respiratory system, Experimental animals, Inflammation*

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INTRODUCTION

Air pollution is still a great environmental and public health challenge today. The World Health Organization claims that 7 to 8 million premature deaths occur annually due to exposure to polluted air (World Health Organization, n.d.). The major sources of atmospheric pollution are industrial enterprises and motor transport, which emit harmful gaseous pollutants and fine particulate matter into the atmosphere (National Institute of Environmental Health Sciences, n.d.). Pollutants such as carbon monoxide, nitrogen dioxide, sulfur compounds, and fine dust particles that can penetrate deep into the respiratory tract are the most dangerous. The respiratory system is especially susceptible to airborne pollutants because the toxic substances inhaled immediately impact the bronchi and lung tissue (Bălă *et al*., 2021). Many studies have shown that chronic exposure to polluted air is a risk factor for bronchial asthma, chronic obstructive pulmonary disease, inflammatory respiratory diseases, and decreased lung function. Fine particles and toxic gases can produce structural and functional changes in the lung, such as inflammatory reactions, bronchial spasm, vascular disorders, and changes in pulmonary ventilation ("The Impact of Airborne Particulate Matter-Based Pollution," n.d.). Experimental studies on laboratory animals are invaluable sources of understanding about the pathological effects of respiratory system polluting agents (Siddiqui *et al*., 2024). Such models can be used to assess direct impacts of pollutants in a controlled setting without social and behavioral factors influencing the impacts (Gilmour *et al*., 2001; Cissé *et al*., 2024). Histological investigation of the respiratory organs can give

useful information on the morphological alterations induced by toxic environmental agents.

Although numerous studies have been conducted on air pollution, less is known about the comparative effects of various air pollution components on respiratory organs (Siddiqui *et al*., 2022; Chen & Chen, 2023). In industrial areas, people are frequently subjected to industrial dust exposure and vehicle exhaust emissions. The pathological differences between these pollutants and their effects on lungs, however, need further experimental studies (Mack *et al*., 2019; Kariri *et al*., 2024). Toxic substances, which can impact the microcirculatory system and respiratory tract, are found in vehicle exhaust gases. Inhalation of exhaust fumes can cause congestion, edema, inflammatory changes, and bronchospasm (Sherbaevna *et al*., 2022; Singh *et al*., 2023; Tam *et al*., 2023). Meanwhile, the dust particles of industry may be deposited in the lungs and may lead to disorders of pulmonary airways and chronic inflammatory reactions (Vimercati, 2011; Hashem *et al*., 2024). The chemical and physical characteristics of gaseous pollutants and dust particles are very different, and so, too, may be the effects on the respiratory organs. A second key question is whether there is a need for controlled experimental models that mimic real environmental exposure scenarios. Epidemiological studies have shown associations between air pollution and respiratory diseases, but these studies cannot always be precisely used to assess direct morphological effects of the specific pollutants (Shetty *et al*., 2023; Csep *et al*., 2024). For this reason, continued experimental inhalation studies in laboratory animals are still needed to determine the structural changes induced in respiratory tissues by different environmental contaminants (Alam *et al*., 2022; Guzek *et al*., 2023). The present study is directed towards the experimental modeling of the effect of cement dust and vehicle exhaust gases on the respiratory organs. The novelty of the work is that for two different types of atmospheric pollutants, the comparative histological study of lung and bronchial changes is conducted under experimental conditions that are controlled.

This study is unique in that it looks at both gaseous emissions and industrial dust exposure in the same experimental design. Differences in the character of the injury to the respiratory tissue were identified by histological examination. Microcirculatory disturbances, bronchial spasms, edema, and inflammatory infiltration were the most frequent findings associated with exhaust gas exposure. Cement dust, on the other hand, led to impaired airiness of the lung tissue, deposition of foreign particles, activation of macrophages, and chronic inflammatory changes. The results acquired help to elucidate the pathological effects of air pollution on the respiratory system and validate the adverse effects of transport- and industrial-origin pollutants. The study also highlights the necessity of protecting the environment and reducing air pollution in industrial and urban areas to reduce risks to health due to the respiratory system.

MATERIALS AND METHODS

Experimental animals

The experimental study was carried out in 30 white outbred male rats weighing 150 – 250g. All animals were quarantined for two weeks prior to the experiment. The animals were split into three groups: a control group, a group exposed to cement dust, and a group exposed to vehicle exhaust gas (all 10 animals in each group). Experiments were conducted following the bioethical principles when dealing with laboratory animals.

Control group

The remaining birds in the control group were kept under normal husbandry as follows: in an environmentally protected environment free from dust and exhaust gases. Animals were held in normal housing throughout the experiment.

Cement dust exposure

In the second group, animals were exposed to cement dust, which was gathered around a cement plant. Dust collection was performed by installing dust collectors in the wind direction in the village located in the vicinity of the factory's cement production area. The height of the aspirator was set at the human respiratory tract level (1.5–2 m). The aspiration rate was kept constant at 20 liters per minute, and the collected particles were collected in hermetically sealed containers, which were then brought to the laboratory for analysis daily. The composition of dust and concentration in dust were determined by the X-ray fluorescence analysis apparatus (XL3t-960), and the mass of dust was determined by the gravimetric method. The animals were adapted in a modified dust chamber made on the basis of the design of V. Babchinsky and I.Z. Labunkova with the dust volume of 127,85dm^3. Cement dust was fed up to 5μm in size at a rate of 50mg/m^3. Exposure: 4 hours/ day, 5 days/ week, 4 weeks. Dusting was conducted at an intermittent dusting mode, consisting of alternately 20 minutes of sand production time and 20 minutes 100% clean air feeding. Gravimetric techniques and horizontal aspiration filters (HAF) were used to control the dust concentration in the chamber. Glass rotameters were used to measure air supply and discharge rates.

Exhaust gas exposure

In the inhalation chamber, "NOE" vehicle exhaust gases were produced using the fuel AI-92. Exhaust gases were added by means of fittings to a mixer, where they mixed with the ambient air and were finally added to the chamber. The harmful gases were prevented from leaking out by keeping the pressure in the chamber between 1 and 2 mm water column. The condition of the gas-air mixture was monitored using automatic gas analyzers and recording systems. The most important exhaust gas parameters measured were carbon monoxide, nitrogen dioxide, and sulfur dioxide. CO concentration was 21mg/m3, which is about seven times the maximum permissible atmospheric concentration. It was found that the concentration of nitrogen dioxide was 1.54 mg/m^3 and the concentration of sulfur dioxide was 0.073 mg/m^3. The weekly measurements of SO2 and NO2 were done by sorption and chemical analytical process. In the experiment, the temperature in the chambers was kept at 20-22°C and R.H. between 50-60%. Animals were housed in normal vivarium housing conditions between exposure sessions.

Histological examination

Animals were terminated in hexane anesthesia in compliance with bioethical needs, after the end of the experimental

exposure period. Lung and bronchus tissue were taken from the euthanized birds for histology. The specimens were fixed in 10% neutral buffered formalin and paraffin-embedded. The 4–5 µm thick histological sections were prepared, and hematozoa were stained with hematoxylin and eosin. The stained sections were examined under a microscope (BIOMED B-6). Histological examination comprised the examination of structural changes in the bronchi and lung tissue, such as changes in the vessels, infiltration of inflammatory cells, bronchial spasm, edema, and changes in pulmonary airways. Many of the experiments were conducted on rats that received the following treatment: máquinas de grunge. For the experimental portion of the study, 30 white outbred rats weighing 150–250 g were used. All animals were quarantined for two weeks prior to the experiment in normal vivarium conditions. Animals used in the study were in numbers compatible with the accepted bioethical guidelines for experimental investigations on rodents that stipulate a minimum of 8–10 animals per group for analysis. The study followed the standards of humane treatment of laboratory animals and also followed the guidelines of minimum animal use in the statistical evaluations. The animals were randomly assigned to three experimental groups. The animals belonging to the first category were fed as controls and housed in regular village laboratory containers in the village of Papan, subjected to environmentally safe air. Cement dust was used for animals in the second group, and vehicle exhaust gases for controlled inhalation in the third group, as shown in **Table 1**.

Table 1. Experimental groups and exposure conditions of animals included in the study.

Group	Number of animals	Exposure condition
Control group	n = 10	Standard atmospheric conditions
Dust exposure group	n = 10	Cement dust exposure
Exhaust gas exposure group	n = 10	Vehicle exhaust gas exposure

Cement dust exposure model

The dust exposure was carried out with dust particles of cement collected in the vicinity of the cement production plant in the village of Gulbaar. The installation of the dust collectors followed the wind directions to collect the dust. The aspirator was re-positioned at a height of 1.5–2 m, which is that of the human upper respiratory tract. All samples were collected in air-tight bottles and were stored in the laboratory daily until analyzed. All the samples were collected in airtight bottles that were later transferred for laboratory analysis, with the aspiration rate kept at 20L/min. The gravimetry technique was used to determine the mass of the particles, and the X-ray fluorescence method was used to determine the composition and concentration of the dust on the apparatus XL3t-960. All animals were put into a dust inhalation chamber modified on the basis of the model of V. Babchinsky and I.Z. Labunkova, with the chamber volume of 127.85 dm^3. The geometric median diameter of the dust particles was up to 5 µm, and their concentration was 50 mg/m^3. A 4-week experimental period with 5 days/week of exposure, 4 hours/day was used. Dust administration was done in a defined intermittent manner, which involved alternating periods of dust-air mixture supply and periods of clean air supply, each of 20 minutes duration. The concentration of dust inside the chamber was measured through gravimetric processes and with the use of AFA filters. The aspiration device and glass rotameters were used to control air supply and discharge rates.

Exhaust gas exposure and histological examination

Animals of the 3rd experimental group were exposed to vehicle exhaust gases emitted by AI-92 fuel in the vehicles parked inside the inhalation chamber "NOE". This chamber was a two-level, removable, stainless steel exposure system. Monitoring and automatic control of aerosol and environmental parameters such as temperature, humidity, oxygen concentration, carbon dioxide concentration, pressure, and gas distribution were provided by the system. The design of the chamber also included exhaust gas purification systems. Newcomer exhaust was fed into the mixer, where it was mixed with atmospheric air before going into the chamber. Harmful gases were prevented from leaking into the chamber by maintaining a negative pressure of 1-2 mm H2O in the chamber. Using automatic gas analyzers and recording systems, the gas-air mixture was monitored. The concentrations of the main gaseous components were 21 mg/m^3 of CO, 1.54 mg/m^3 of NO2, and 0.073 mg/m^3 of SO2. The concentration of carbon monoxide was about seven times greater than the Maximum allowable concentration in the atmosphere. The relative humidity of the chambers was kept from 50-60% and the temperature was between 20-22°C. Animals were killed, following the bioethical requirements, under hexane anesthesia, at the end of the experimental period. Lung and bronchial tissues were taken for histological examination. The collected samples were preserved in 10% neutral formal saline and then processed into one-inch-thick paraffin blocks, and then cut into slices of 4–5 µm in thickness and stained in hematoxylin and eosin. The stained sections were submitted to histological examination using a microscope BIOMED B-6, and the micrographs were obtained in the process of microscopic examination.

Ethical statement

The study was approved by the Ethical Review Board of Osh State University, Osh city, Kyrgyzstan, and was conducted in accordance with institutional guidelines for the care and use of laboratory animals and the principles of the Declaration of Helsinki (World Medical Association, 2013; Chen & Chen, 2023; Salikhova *et al*., 2023). All experimental procedures followed accepted bioethical standards to ensure humane treatment and minimize animal suffering. Animals were maintained under standard vivarium conditions and euthanized under anesthesia at the end of the experiment.

RESULTS AND DISCUSSION

Results of experimental work

The experimental portion of the study was performed to create a model of the changed conditions of the respiratory system under the influence of various concentrations of air pollution. The experimental model aimed to assess the direct action of major atmospheric pollutants on the respiratory organs in a

controlled environment. The study had two objectives: The goal of the first was to explore the potential morphofunctional modifications in the respiratory system after intentional exposure to air pollutants. The second was to control for social, behavioral, psychological, and other factors that may impact human populations that reside in environments contaminated at different levels. Histological tissues were taken from sexually mature rats from the laboratory, which were exposed to the irritation of cement dust and vehicle exhaust gases under standard vivarium conditions. The histopathological examination of the respiratory organs in the normal control animals showed the normal structure of the lungs and bronchi. Multilayer non-keratinized epithelium without pathological changes was present on the bronchial wall. The mucous membrane was intact, and there were no vascular changes. Lung tissue was observed descriptively and showed a complete retention of architectural and aieriness features indicative of a lack of any pathological process in the respiratory organs. The structure of bronchial mucosae was normal, indicating the normal structure of epithelial tissue and the protection of the respiratory mucosa against the external environment. There was no inflammatory infiltration, edema, or epithelial desquamation seen in the control animals. The vascular network of the lungs and bronchi remained the same, and the lungs have a normal blood supply and normal physiological respiratory function.

Histological changes following exposure to exhaust gases

In animals exposed to vehicle exhaust gases, considerable structural changes in the respiratory organs were found in the histology. The greatest changes were seen in the pulmonary microcirculatory bed. The blood was found to be voluminous, and a dynamic slowing of blood flow occurred in the pulmonary vessels. Several areas of the lung tissue showed interalveolar septal thickening. Fluid buildup in the tissues was representative of pulmonary edema in some parts. There was also pronounced bronchial spasm noticed. Mucosa of the bronchi was moderately edematous, and in some areas, cells from the mucosal layer were found in the bronchial lumen. In preserved areas, the bronchial epithelium was still in a high cylindrical structure. Inflammatory changes were often observed in the form of lymphoid infiltrates in the bronchial wall, especially in larger bronchi. Atelectasis was present in many small areas of the respiratory portions of the lungs. Exposure to exhaust gases overall had a negative impact on the pulmonary microcirculatory system and bronchial system (airway) and caused vascular congestion, edema, bronchospasm, and inflammatory changes with secondary structural changes of pulmonary tissue.

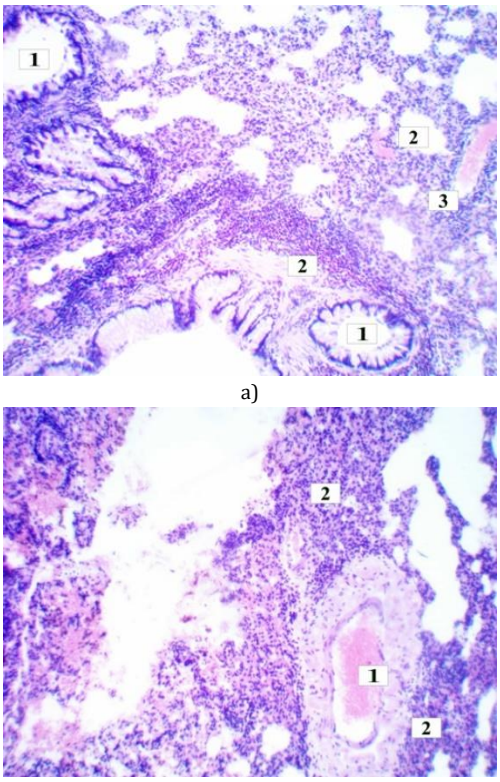

Figure 1. a): cross-section of the lungs and bronchi. Uv. x 100. Bronchial spasm-1, atelectasis-2, plethora-3. b): Lungs. Uv. x 100. Plethora-1, lymphoid infiltrate-

Histological changes following exposure to cement dust

Histological analysis of animals that were exposed to cement dust showed that there was extensive damage to their lung tissue. The main changes in the lungs of animals in the exposure group were related to changes in lung airiness and inflammatory changes in the lungs. Unlike the group with exposure to exhaust gas, the main changes were found in the pulmonary airways and inflammatory changes in the lungs of the animals. Although moderate vascular plethora was seen, the most important pathological changes were those of ventilatory dysfunction and structural changes of the pulmonary tissue. The main histological finding was that areas of distelectasia, in which the normal airiness of the lungs is lost, were found. Reduced air content was seen in some areas of the lungs, potentially leading to defective gas exchange.

Unbalanced ventilation (airless and air over-ventilated areas) within the lung tissue was found throughout the tissue, suggesting irregular distribution of ventilation and disturbance of pulmonary ventilation-perfusion relationships. Numerous alveolar epithelial cells exfoliate, and macrophages were found in the alveoli. Inflammatory and protective cellular responses within the lungs were activated, as evidenced by the presence of macrophages. Foreign particulate matter was also demonstrated in the pulmonary stroma, which is further evidence of penetration and deposition of inhaled dust to the lungs. The presence of lymphoid infiltrates in the bronchial walls and in the pulmonary parenchyma suggested the presence of chronic inflammatory reactions as a response to long-term exposure to inhaled pollutants. Overall, the histological analysis

showed that cement dust exposure leads to severe structural changes in lung tissue relevant to the reduction of airways and inflammatory changes, as well as deposition of inhaled particulate matter, suggesting the adverse effects of particulate pollutants in the atmosphere on the respiratory system **(Table 2).**

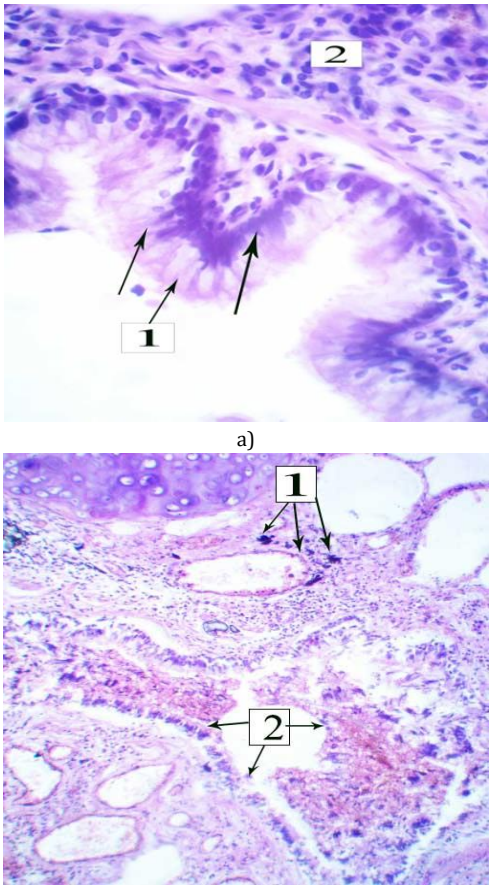

Figure 2. a) - Bronchial. Uv. Hyperfunction x 100 of the bronchial epithelium, mucus production-1, lymphoid infiltrate-2. b) - Figure in the bronchi and Lungs on a cross-section. UV x 100 -1 Foreign particle, bronchial mucus-2.

Table 2. Histological changes observed in the respiratory organs of experimental animals following exposure to exhaust gases and cement dust.

Experimental group	Histological findings
Control group	Normal structure of lung tissue and bronchi was preserved. The epithelial lining, vascular network, and pulmonary architecture showed no pathological alterations.
Exposure to exhaust gases	Fullness of pulmonary blood vessels and vascular stagnation; thickening of interalveolar septa; pulmonary edema; bronchial spasm and desquamation of bronchial epithelium; lymphoid infiltrates and focal areas of atelectasis.
Exposure to cement dust	Distelectasis of individual lung regions; alternating zones of hypo- and hyperventilation; large numbers of macrophages and desquamated alveolar epithelium; presence of foreign particles within lung tissue; lymphoid infiltrates indicating chronic inflammatory changes.

Both vehicle exhaust and cement dust were found to be effective in causing pathological changes in the respiratory organs of laboratory animals as a result of the present experimental study. Meanwhile, the type of tissue damage varied according to the type of inhaled pollutant. Histological findings showed that the pulmonary microcirculatory system and bronchial structures were mostly affected in the case exposed to the exhaust gases, while in the case exposed to the cement dust, the most prevalent changes were those in the structure of airiness in the lungs and the chronic inflammatory changes in the lung tissue. Vascular congestion, stagnation of blood in the pulmonary vessels, thickening of interalveolar septa, pulmonary edema, bronchial spasm, and inflammatory infiltration were the findings in the exhaust gas exposure group. The changes suggest that the pulmonary microcirculation is impaired and acute inflammatory reactions in the respiratory tract are present (Nyamagoud *et al*., 2024; Petronis *et al*., 2025). Bronchospasm and edema are noted, and it is possible that this causes narrowing of the lumen of the bronchi and diminished respiratory function. Further, focal atelectasis indicates secondary ventilation disturbances, presumably due to the toxic effects of gases on the respiratory system. The results obtained show that few studies were compared to those reported in an earlier international study on the biological impact of vehicle emissions on lung tissues. Other reportable changes are those seen by SS Taha *et al*. (2025), who reported that exhaust-related pollutants such as carbon monoxide (CO) and nitrogen dioxide (NO_2) lead to hypoxia, inflammatory responses, and structural changes of the pulmonary tissue (Cakmak *et al*., 2024; Manfredini *et al*., 2024; Taha *et al*., 2025). The microcirculatory changes detected in the present study confirm this concept that gaseous pollution acts predominantly on the regulation of the vascular system, and that bronchial reactivity is altered.

By contrast, exposure to cement dust caused other patterns of histological reactions. The most common observations were areas of distelectasis, hypo-hyperventilated areas, accumulation of desquamated alveolar epithelial cells and macrophages, presence of foreign particles in the pulmonary tissues, and lymphoid infiltrates warranting chronic inflammatory activity, as also shown in a study by AH Rahmani *et al*. (2018) , Çınaroğlu *et al*. (2023), and Leadbeatter and Tjaya (2024). The present findings illustrate the ability of fine particulate matter to reach the lower respiratory tract up to the lung parenchyma. A great number of alveolar macrophages is a sign of alveolar defense mechanism activation as a result of inhaling particles. Macrophages are important cells for the process of phagocytosis and the elimination of foreign particles, but as their activation persists for longer periods, they may be responsible for chronic inflammatory reactions and interfere with the normal ventilatory process in the lungs (Lendeckel *et al*., 2022; Bratt & Naimi-Akbar, 2023; Muthanandam *et al*., 2024). Point-by-point deposition of cement dust particles in the respiratory part of the lungs is confirmed by the finding of

foreign particles in the lung tissue in the present study.

The results obtained confirm the ones obtained by R Ahmad *et al*. (2021), who noted the occurrence of chronic inflammatory lesions and occupational respiratory diseases among people working in the industrial production of cement and sand dust (Lendeckel *et al*., 2022; Çınaroğlu *et al*., 2023). Other works, including Y Shi *et al*. (2025) and Y Fan *et al*. (2020), have indicated that chronic exposure to dust particles from industry can lead to chronic bronchial and pulmonary diseases such as pneumoconiosis and impaired lung function. These observations are confirmed by chronic inflammatory infiltrates seen in our model and are suggestive that chronic exposure to particulate matter can have a harmful effect on respiratory tissues. The present study also corroborates the findings of RB Hamanaka *et al.* (2025), which showed an association between long-term exposure to fine particulate matter and inflammatory diseases and disorders of pulmonary gas exchange. The changes in airiness and ventilation that were seen in the lungs in the present experiment after exposure to cement dust might be related to impaired efficiency of the respiratory function of the lungs due to the deposition of inhaled dust in lung tissue. Comparisons between the two exposures indicate that there may be separate pathogenetic mechanisms for respiratory injury resulting from gaseous vs. particulate pollutants. Bronchospasm, pulmonary microcirculatory disturbances, vascular congestion, and pulmonary edema were the main effects of gaseous pollutants. Cement dust exposure, on the other hand, caused most often chronic inflammatory reactions, foreign particle deposition, activation of macrophages and reduction of airiness of the lungs. The results suggest that the mechanism and severity of injury to the respiratory tissue depends on the physical and chemical properties of the pollutants present in the air. Review of overall results obtained indicates the role of any pollution of the atmosphere from both the transport system and industrial activities is detrimental to the respiratory system and the need for measures in order to control the environment and reduce toxic exposure to the atmosphere in industrial and urban areas.

The results of the present experimental study can also be interpreted against the backdrop of the general background of environmental and domestic air pollution in Kyrgyzstan. Chronic exposure to coal-based heating emissions may cause long-term health effects on the respiratory and systemic systems for homemaking women in Southern Kyrgyzstan, as it does for the rats used in the experimental setting following exposure to cement dust and vehicular exhaust. Fine particulate matter and toxic compounds found in industrial pollutants and/or domestic combustion emissions are capable of either causing inflammatory changes or oxidative stress, both of which have an impact on pulmonary function. Women are especially vulnerable in areas that experience extreme weather conditions, where coal continues to be a major fuel source, and where the air inside the home is poorly ventilated, allowing them to be exposed to the coal smoke for extended periods of time. Thus, the histological changes observed in the present experimental model could be used as a biological tool to learn the mechanisms involved in the pathogenesis of respiratory morbidity in human populations using environmental and indoor air pollution. All of the above studies highlight the need for coordinated public health strategies to address both the outdoor industrial emissions and the indoor household air pollution exposures to alleviate the increasing public health burden of air pollution-related respiratory and cardiovascular diseases in Kyrgyzstan.

CONCLUSION

In our study, the following pathological changes were demonstrated in experimental modeling, showing significant changes in the respiratory organs as a result of vehicle exhaust gases and cement dust exposure. The lungs had the most severe effects, revealed in histological examination as primarily damage to the pulmonary microcirculatory system and bronchial structures caused by the exposure to exhaust gas, which led to the presence of congested blood vessels with edema, bronchospasm, and acute lesions in lung tissue. Cement dust, on the contrary, mainly induced chronic inflammatory changes, which correlated with the changes in the pulmonary airiness, the foreign particle accumulation in the alveolar tissue, the activation of alveolar macrophages, and the disturbance of gas exchange processes. These results suggest that particulate pollutants cause chronic inflammatory responses and lung structural alterations in the respiratory parts of the lungs. The results obtained are in accordance with the fact that gaseous emissions from transport and the dust pollution in the industry constitute serious threats to the health of the respiratory system, especially in areas with high industrial and atmospheric pollution. Study highlights the need for environmental protection measures and minimization of air pollution exposure to avoid the development of respiratory conditions related to the intake of harmful gases formed in the atmosphere.

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CONFLICT OF INTEREST: None

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ETHICS STATEMENT: The study was approved by the Ethical Review Board of Osh State University, Osh city, Kyrgyzstan, and was conducted in accordance with institutional guidelines for the care and use of laboratory animals and the principles of the Declaration of Helsinki. All experimental procedures followed accepted bioethical standards to ensure humane treatment and minimize animal suffering. Animals were maintained under standard vivarium conditions and euthanized under anesthesia at the end of the experiment. The corresponding author may be contacted for further information regarding the ethical approval and experimental procedures.

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