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SHORT COMMUNICATION

Effects of inhaled methyl methacrylate monomers from acrylic prostheses on mouse lung tissue

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Abstract

Introduction: Materials based on polymethyl methacrylate (PMMA) have become more widely used because of their many benefits in various medical specialties, such as maxillofacial surgery, ophthalmology, and orthopedic prostheses. Since methyl methacrylate (MMA), the monomer from which PMMA is made, is extremely volatile and breathed during handling, inhalation is the main way that laboratory and healthcare workers are exposed to MMA. This compound is toxic and can irritate the respiratory mucosa, respiratory system, skin, nervous system, and other organs.
Objective: This study aims to assess the effect of MMA inhalation on lung tissue histology.
Methods: This *in vivo* experimental study used 15 white mice, which were randomly assigned to three groups: A negative control, a group exposed to MMA for 1 week, and a group exposed for 3 weeks. Mice were subjected to MMA inhalation for 6 h/day. Following exposure, lung tissues were processed for histological examination, including fixation, dehydration, paraffin embedding, sectioning, and hematoxylin-eosin staining. Lung injury was evaluated using a semi-quantitative histological scoring system and by measuring alveolar diameter in five fields of view.
Results: No lung damage was observed in the control group; lung histology remained within normal limits. The group exposed to MMA for 1 week showed mild to moderate lung damage, whereas the group exposed for 3 weeks exhibited moderate to severe histological damage. Measurements of alveolar diameter showed a decrease in both MMA-exposed groups.
Conclusion: Inhalation exposure to MMA causes significant histological damage to lung tissue in mice.

Keywords: Methyl methacrylate; Inhalation; Toxicity; Lung; Histology

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1. Introduction

Methyl methacrylate (MMA) monomer is widely used in dentistry as the main component for making dental prostheses, including bases of full and partial dentures,

denture relining and repairs, obturator prostheses, maxillofacial defect prostheses, facial prostheses, splints, removable orthodontic appliances, space maintainers, artificial teeth, veneers, and temporary crowns and bridges. In medicine, MMA is used in bone cement for fixation of orthopedic implants, in ophthalmic restorative resins (such as intraocular lenses), and in plastic and maxillofacial reconstructive surgery.[1-4] Acrylic powder and MMA monomer are combined during the dental prosthesis manufacturing process.[5] MMA is a colorless, volatile, and pungent-smelling liquid, which is metabolized into methacrylic acid.[3] MMA is potentially toxic and can affect the respiratory system, skin, oral mucosa, nervous system, liver, and gastrointestinal system.[1] Frequent exposure to the substance occurs among dental technicians.[2]

Occupational exposure to MMA is of considerable concern, as inhalation represents the primary route of toxicity and predominantly targets the respiratory system.[2] Inhaled MMA has been shown to alter neuronal activity, particularly by increasing the excitability of neurons in the area postrema, a chemoreceptor-trigger zone located in the medulla oblongata that lacks a blood-brain barrier and is responsible for initiating emetic responses.[6] This neural activation suggests that vagal afferent pathways may play a role in the systemic effects of MMA, linking inhalation not only to local respiratory irritation but also to central nervous system-mediated responses. In addition, methacrylate monomers are capable of causing oxidative DNA damage and inducing genotoxic effects through mechanisms involving oxidative stress.[2] Such DNA lesions can lead to somatic mutations, which, when accumulated, may contribute to malignant transformation and ultimately increase the risk of cancer development.[7]

Despite these concerns, the toxicological impact of MMA on pulmonary tissue has not been extensively studied. Previous work by Goenharto *et al.*[8] demonstrated that exposure to 150 ppm of MMA in mice produced clear histological evidence of lung inflammation, suggesting that the respiratory tract is particularly vulnerable to methacrylate monomers. However, detailed characterization of pulmonary injury, including structural alterations, inflammatory infiltration, and morphometric changes, remains limited in the literature.[2] Given the increasing occupational use of MMA in medical, dental, and industrial applications, understanding its impact on lung tissue is crucial. The present study was therefore designed to investigate the histopathological consequences of MMA inhalation, with particular attention to cellular damage, inflammatory processes, and structural changes in the alveolar architecture. This approach allows for a more comprehensive evaluation of MMA-induced respiratory injury and provides experimental evidence that may inform occupational health and safety practices.

2. Materials and methods

2.1. Materials

2.1.1. Animals

A total of 15 healthy male white mice (*Mus musculus*, DDY strain), aged 3 months and weighing 20–30 g, were obtained from the Pharmacology Laboratory, Faculty of Pharmacy, Andalas University, and used in this study.

2.1.2. Animal care

The mice were acclimatized for 1 week prior to the experiment. During this period, all animals received a standard diet comprising 67.2% carbohydrates, 12.7% protein, and 5.3% fat, at 10% of their body weight. Food and water were provided *ad libitum* throughout the study.

2.1.3. Inhalation exposure chamber

Mice were exposed in a glass chamber (approximately 60 L) routinely employed in the laboratory for inhalation experiments. The chamber size was sufficient to accommodate 5–10 mice without overcrowding and had been used in the facility for similar studies. The chamber was maintained under controlled room conditions with a temperature of around 22–24°C and a relative humidity of 50–60%. The procedure was performed by trained laboratory staff following standard operating procedures. Detailed records of airflow and potential adsorption to chamber surfaces were not available, and this was considered a limitation of the study.

2.1.4. MMA

MMA, a self-curing acrylic compound manufactured by S. Court Limited (England), was used as the test substance in this study.

2.1.5. Equipment and Reagents

The equipment and reagents used in this study included an Olympus CX33 light microscope (Japan) for histological observation and a Sony Beta microscope camera equipped with a 3.1 MP Sony Exmor CMOS sensor (Japan) for image acquisition. Image processing and measurement of alveolar diameters were performed using the BetaViewInk software (Sony, Japan). Histological preparations of lung tissue were stained using hematoxylin and eosin (H&E) reagents, which enabled detailed visualization of cellular morphology, tissue architecture, and pathological alterations. These tools and reagents were essential for ensuring accurate assessment and documentation of lung histopathological changes following MMA exposure.

2.2. Experimental design

Randomization was conducted at the individual animal level using a simple random number generator, and the animals were housed five per cage with randomized cage assignment. Histological scoring and alveolar diameter measurements were performed by two independent observers who were blinded to the treatment groups, with a high level of consistency between their assessments ($\kappa = 0.81$). This study employed a laboratory posttest-only group design. A total of 15 mice were randomly allocated into three groups ($n = 5$): Group 1, negative control (0 ppm MMA); Group 2, exposed to MMA for 1 week; and Group 3, exposed to MMA for 3 weeks.

2.3. MMA inhalation exposure procedure

Mice were exposed to MMA by inhalation for 6 h/day, 5 days a week. Each day, the MMA weight was measured before and after exposure to assess the exposure dose, with the average daily exposure calculated at approximately 23.9 ppm. This value represents an estimated mean based on monomer weight loss, rather than a direct, time-resolved measurement of gas concentration.

2.4. Euthanasia and tissue collection

Mice were ethically euthanized under anesthesia, followed by cervical dislocation. Lung tissues were harvested and fixed for histological analysis.

2.5. Histological procedures

2.5.1. Histological processing and staining

Histological processing included dehydration, clearing, embedding, sectioning, and mounting on glass slides. H&E staining involves using hematoxylin for nuclear staining (blue/purple) and eosin for cytoplasmic and extracellular matrix staining (pink/red), with steps for washing, dehydration, and mounting. After staining, the slides were examined under a microscope to assess the extent of lung injury and any pathological changes. The examination of histological lung injury was then completed.

2.5.2. Histological analysis

Histological analysis of lung injury was conducted using a semi-quantitative scoring system to assess tissue damage. Observations were made at ×400 total magnification (×40 objective lens) across five fields of view per sample, and the results were reported as mean scores. In addition, the diameters of alveoli were measured at the same magnification, with a minimum of 50 alveoli evaluated per sample to determine the mean alveolar diameter (μm). The scoring and measurement criteria followed the inflammation scoring system established by Pham *et al.*[9] The lung damage scoring system is presented in Table 1.

2.6. Statistical analysis

The results of the study were statistically analyzed using one-way analysis of variance (ANOVA) for normally distributed data and Kruskal-Wallis test for non-normally distributed data, followed by *post hoc* comparisons using least significant difference and Mann-Whitney *U* tests.

3. Results

3.1. Histopathological evaluation of lung injury

Histological analysis demonstrated clear differences in lung tissue integrity between control and exposure groups. Group 1 (control) exhibited normal alveolar architecture with thin septa and no evidence of edema, congestion, or inflammatory cell infiltration (Figure 1 and Table 2). In contrast, Group 2 (1-week exposure) displayed mild to moderate alterations, including interstitial edema and scattered inflammatory cell infiltration. More severe changes were observed in Group 3 (3-week exposure), including moderate to severe inflammation, alveolar wall thickening, and disruption of normal tissue structure. Statistical analysis confirmed these observations: The Kruskal–Wallis test revealed significant overall group differences (p=0.002), and Mann–Whitney *U* tests showed significant pairwise differences between Groups 1 and 2, Groups 1 and 3, and Groups 2 and 3 (all p=0.008). These results indicate a progressive, exposure-dependent pattern of lung injury following MMA inhalation.

3.2. Alveolar diameter measurements

Morphometric analysis revealed a progressive reduction in mean alveolar diameter across the experimental groups. In the control group (Group 1), alveoli displayed uniform morphology with an average diameter of 34.5 μm (Figure 2 and Table 3). A decrease was observed in Group 2 (27.7 μm), although this reduction did not reach statistical significance compared with controls. The most pronounced narrowing was observed in Group 3, with an average diameter of 22.1 μm, reflecting a more extensive structural alteration. One-way ANOVA indicated a significant overall difference among groups (p=0.013). *Post hoc* analysis using the least significant difference test confirmed a significant reduction between Groups 1 and 3 (p=0.004), while comparisons between Groups 1 and 2 (p=0.072) and between Groups 2 and 3 (p=0.133) were not significant. These findings suggest that substantial reductions in alveolar diameter occur primarily after prolonged exposure, corresponding with the development of more severe histological injury.

Table 1. Lung damage scoring system

Score	Parameters of lung damage level	Extent of inflammation	Level of inflammation
0	Thin alveolar walls, very few alveolar macrophages, inflammation (–), Normal respiratory epithelium, with a thickness of 1 cell layer. Smooth muscle, submucosal layer, and blood vessel endothelium are normal	No inflammation	Normal
1	Few macrophages/monocytes in alveolar, submucosal, and perivascular areas. PMN (–). Almost all perivascular connective tissue is visible	<25% lung inflammation	Mild inflammation
2	The respiratory tract is slightly thickened, with a moderate distribution of PMN/neutrophil phagocytes, eosinophils, and macrophages in the alveolar and perivascular areas. Most of the perivascular connective tissue is still visible	25–50% lung inflammation	Moderate inflammation
3	Dense distribution of phagocytes/PMN cells, foamy cells, alveolar macrophages, and perivascular forming cell clusters. Alveolar/airway walls are thickened. Most of the perivascular connective tissue is faint	>50% lung inflammation	Severe inflammation

Abbreviation: PMN: Polymorphonuclear cells.

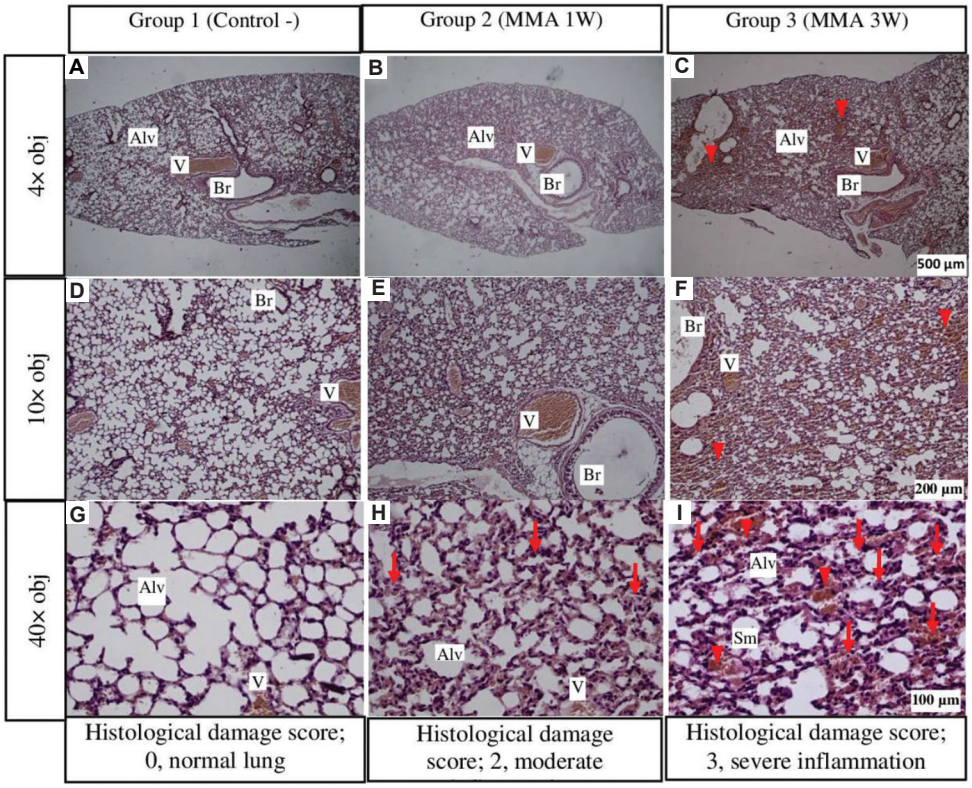

Figure 1. Histopathological features of mouse lung tissue stained with hematoxylin and eosin following MMA exposure, shown at (A-C) ×40 magnification (scale bar: 500 µm), (D-F) ×100 magnification (scale bar: 200 µm), and (G-I) ×400 magnification (scale bar: 100 µm). Representative micrographs show bronchioles (labeled as Br), blood vessels (labeled as V), and alveoli (labeled as Alv). (A, D, G) Group 1 (control) exhibits normal alveolar morphology with thin septa and intact air spaces, without inflammatory changes. (B, E, H) Group 2 (1-week exposure) demonstrates early inflammatory changes, including thickened alveolar septa and perivascular regions due to inflammatory cell infiltration (red arrows). Several alveoli contain inflammatory cells, and multiple blood vessels appear dilated and hemorrhagic (red triangles). (C, F, I) Group 3 (3-week exposure) shows more pronounced lung injury, characterized by diffuse septal thickening, marked inflammatory cell infiltration (red arrows), vascular congestion, and focal hemorrhage.
Abbreviation: MMA: Methyl methacrylate.

In addition to the reduction in alveolar diameter, qualitative differences in lung architecture were observed across the groups. In the 1-week exposure group, edema was localized and inflammatory cells were scattered within the alveolar septa, whereas in the 3-week exposure group, the inflammation was more diffuse, extending into perivascular and peribronchiolar areas. Vascular congestion and occasional hemorrhage were also evident in the prolonged exposure group, suggesting that microvascular injury accompanies epithelial and alveolar damage. These pathological changes correspond with the increasing histological damage scores and provide

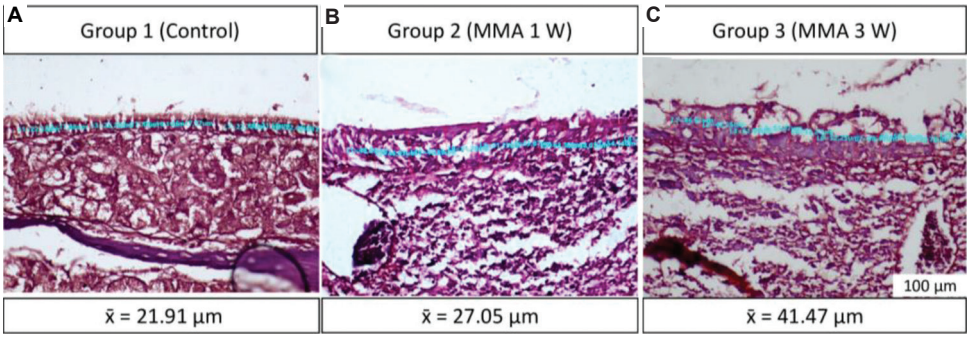

Figure 2. Alveolar morphology and diameter measurements in mouse lung tissue following MMA exposure for (A) Group 1 (control), (B) Group 2 (1-week exposure), and (C) Group 3 (3-week exposure) (scale bar: 100 µm; magnification: ×400). Alveolar diameter was measured from a minimum of 50 alveoli per group, and values are reported as mean diameter (µm). Group 1 shows normal alveolar architecture with thin septa and air-filled spaces. Group 2 displays smaller alveolar diameters with septal thickening and inflammatory cell infiltration. Group 3 exhibits more pronounced narrowing of alveolar spaces compared with Group 2, reflecting progressive injury.
Abbreviation: MMA: Methyl methacrylate.

Table 2. Histological assessment of mouse lung tissue after exposure to methyl methacrylate

Group/exposure duration	Histological damage score		Statistical analysis		
	Mean±standard deviation	Level of damage	Kruskal-Wallis test	Group comparisons	Mann-Whitney test
Group 1 (control)	0.24±0.17	Normal	p=0.002*	Group 1 and 2	p=0.008*
Group 2 (1-week exposure)	1.76±0.26	Slight-to-moderate damage		Group 2 and 3	
Group 3 (3-week exposure)	2.52±0.18	Moderate-to-severe damage		Group 3 and 1	

Note: *p<0.05.

Table 3. Measurement of the alveoli diameter of mouse lung tissue after exposure to methyl methacrylate

Group/exposure duration	Alveolar diameter (µm)			
	Mean±standard deviation	One-way ANOVA	Group comparisons	Least significant difference
Group 1 (control)	34.50±3.44	p=0.013*	Group 1 and 2	p=0.072
Group 2 (1-week exposure)	27.71±5.68		Group 2 and 3	p=0.133
Group 3 (3-week exposure)	22.16±6.69		Group 1 and 3	p=0.004*

Note: *p<0.05.
Abbreviation: ANOVA: Analysis of variance.

supportive evidence that lung injury progresses in both severity and spatial extent with longer MMA exposure. The combined qualitative and quantitative analyses highlight the multifaceted impact of MMA inhalation, affecting not only alveolar dimensions but also the integrity of surrounding vascular and connective tissues.

4. Discussion

In this study, we histopathologically evaluated the effects of MMA inhalation on lung tissue and observed clear differences between the control and exposed groups. Mice exposed for 1 week (Group 2) showed early signs of injury, including alveolar narrowing, septal thickening, and localized inflammatory cell infiltration in both alveolar and perivascular regions. These changes suggest that short-term exposure is sufficient to trigger an acute inflammatory response, likely reflecting the irritant properties of MMA vapors. In contrast, prolonged exposure for 3 weeks (Group 3) resulted in more extensive damage, including diffuse inflammation, alveolar edema, vascular congestion, and occasional hemorrhage. The presence of inflammatory infiltrates in perivascular and airway connective tissue indicates a progression from localized to widespread tissue involvement. Consistent with these qualitative observations, quantitative measurements showed a progressive reduction in alveolar diameter, with the most significant narrowing observed after 3 weeks of exposure. Together, these findings demonstrate that MMA inhalation induces time-dependent pathological alterations in the lung, ranging from mild inflammatory changes to more severe structural damage.

Histological assessment showed that exposure to MMA caused significant lung damage (Kruskal-Wallis, p=0.002). Pairwise comparisons demonstrated that both exposure groups had significantly higher histological damage scores than controls, and the 3-week exposure group exhibited more severe damage than the 1-week exposure group (Table 2 and Figure 1). In terms of alveolar diameter, the omnibus test indicated an overall difference among groups (ANOVA, p=0.013). However, pairwise analysis revealed that only the 3-week exposure group had a significantly smaller alveolar diameter compared with controls (p=0.004). The 1-week exposure group showed a reduction in mean alveolar diameter, but this did not reach statistical significance compared with controls (p=0.072) or with the 3-week group (p=0.133) (Table 3 and Figure 2). Taken together, these findings suggest that MMA inhalation induces progressive histological lung injury, and the severity of injury increases with longer exposure duration, with the most consistent and significant effects observed after 3 weeks of exposure.

Our findings are in line with previous animal studies demonstrating the pro-inflammatory effects of MMA on lung tissue. Goenharto *et al.*[8] reported that short-term MMA exposure in mice increased the number of inflammatory cells within the lung parenchyma after only 120 min of exposure. Although their study did not employ a histological damage scoring system, the presence of inflammatory cells confirmed the irritant effect of MMA vapors on respiratory tissue. In contrast, our study not only confirmed inflammatory cell infiltration but also quantified the extent of tissue injury using a standardized histological scoring method, complemented by morphometric assessment of alveolar diameter. This multiparametric approach allowed us to demonstrate a more progressive and measurable pattern of lung injury, from mild infiltration at 1 week to widespread alveolar and vascular disruption after 3 weeks of exposure.

The histopathological changes observed in our study are highly relevant in the context of occupational exposure. At a concentration of 125 ppm, MMA vapor is known to cause acute symptoms in humans, including coughing, throat irritation, and watery eyes.[10] Due to its irritant properties, the United States Occupational Safety and Health Administration (OSHA) has set a permissible exposure limit of 100 ppm for MMA over an 8-h workday.[11,12] In our study, however, the estimated daily MMA exposure per mouse was approximately 23.9 ppm. Despite being far below the OSHA threshold, this concentration was sufficient to cause moderate to severe histopathological lung damage after 1 and 3 weeks of exposure. These results indicate that even relatively low concentrations of MMA may have cumulative toxic effects when exposure is repetitive, emphasizing the need for strict occupational safety measures.

At the cellular and molecular level, MMA monomers have been shown to interfere with essential biological processes, including differentiation, proliferation, and apoptosis. Previous studies demonstrated that MMA can inhibit osteoblast proliferation, induce apoptosis, and trigger necrosis in osteoblast cell lines. Similarly, fibroblast cells exposed to MMA or poly-MMA have exhibited DNA damage, genotoxic effects, and alterations in cell cycle progression.[13] Although evidence on developmental and reproductive toxicity, such as skeletal deformities and low birth weight, has been reported, these findings remain debated and have not been consistently confirmed at occupational exposure levels.[12]

Several studies have demonstrated that MMA possesses cytotoxic, genotoxic, and even estrogenic potential. One important mechanism underlying its toxicity is glutathione depletion, which increases the generation of reactive oxygen species and consequently elevates oxidative stress within cells.[14] Reactive oxygen species are well known to cause nucleotide oxidation, DNA strand breaks, protein misfolding, and disruption of DNA and RNA synthesis, in addition to interfering with antioxidant defense systems.[15,16] These molecular disruptions can initiate and promote carcinogenesis. Moreover, oxidative stress has been implicated in the pathogenesis of numerous diseases, including ischemia-reperfusion injury, endothelial dysfunction, systemic inflammation, sepsis, diabetic retinopathy, cancer, cataracts, cardiovascular disease, and cognitive impairment.[7] These mechanisms provide a strong biological rationale for the progressive tissue damage observed in our experimental model.

The acute histological changes observed in our study are more consistent with toxic inhalation injury than with chronic degenerative alterations. Acute exposure to volatile chemicals is well recognized to cause airway inflammation and chemical pneumonitis, often presenting with features of acute bronchitis such as airway irritation, alveolar edema, and inflammatory cell infiltration.[17] Clinically, Chao *et al.*[18] described a case of vanadium pentoxide inhalation leading to severe acute chemical pneumonitis with bronchitic manifestations and pulmonary infiltrates, which improved following corticosteroid treatment. These clinical and experimental observations support our interpretation that MMA inhalation for 1–3 weeks primarily induces acute inflammatory and bronchial changes, characterized by alveolar septal thickening, inflammatory infiltration, vascular congestion, and narrowed alveolar spaces. Thus, the reduced alveolar diameter in our study should be

viewed as a reflection of acute inflammatory injury rather than chronic emphysematous changes.

Exposure to chemicals, pollutants, irritants, allergens, viral infections, bacterial infections, tissue injury, inhaled hazardous particles, and, in rare cases, autoimmune conditions can lead to inflammation of the respiratory tract.[19] In this context, acrylate monomers such as MMA represent an important occupational hazard. Preventive strategies are therefore critical. To minimize risk, protective masks should be worn during the preparation and processing of MMA-based materials, and adequate ventilation in dental laboratories and industrial settings must be ensured.[1] These preventive measures can substantially reduce acute irritant effects and help prevent long-term cumulative damage to lung tissue.

This study has several limitations that should be acknowledged. First, alveolar diameter was used as a morphometric parameter, but this is a relatively coarse surrogate of lung structural changes and may not fully capture the complexity of tissue injury. In addition, we did not employ inflation fixation or control for lung inflation pressure during tissue preparation, which may have influenced the measurement of alveolar dimensions. Second, MMA chamber concentrations were estimated indirectly rather than measured in real time with calibrated gas detectors, limiting the precision of exposure characterization. Third, the relatively short exposure duration (1–3 weeks) does not allow conclusions about whether acute inflammatory changes might evolve into chronic remodeling or fibrotic processes. Another limitation of this study is the absence of recovery groups to assess whether the observed lung changes were reversible or permanent. Finally, the study was conducted in a small animal model, and while murine data provide important insights, direct extrapolation to human occupational exposure should be made cautiously. These limitations should be addressed in future studies to obtain a more comprehensive understanding of MMA-induced pulmonary toxicity.

5. Conclusion

This experimental study demonstrates that inhalation exposure to MMA induces progressive lung injury in a time-dependent manner. Histological scoring revealed significant differences between both exposure groups and controls, while morphometric analysis confirmed a significant reduction in alveolar diameter after prolonged exposure. The findings indicate that even relatively low levels of MMA can provoke acute inflammatory and bronchiolar changes, leading to tissue disruption and compromised alveolar architecture. By integrating histological and morphometric endpoints, this study provides novel evidence of the multifaceted nature of MMA-induced lung injury and emphasizes the importance of strict occupational and laboratory safety measures to minimize inhalation exposure.

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Conflict of interest

The authors declare no competing interest.

Author contributions

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Ethics approval and consent to participate

This study was approved by the Research Ethics Committee of the Faculty of Pharmacy, Andalas University (Approval No. 48/UN16.10.D.KEPK-FF/2024).

Consent for publication

Not applicable.

Availability of data

Not applicable.

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