Research Article

Omega-3 Administration Suppresses Changes in the Lungs of D-Galactose-Mediated Aged Rats

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Abstract | The present research seeks to look into the potential benefits of omega-3 in mitigating disruptions in lung performance, redox homeostasis, and inflammatory criteria in D-galactose-treated rats. Sixty adult male rats were randomly assigned to four experimental groups as follows: (1) Control group (T1); (2) T2 group, injected with D-galactose and administered omega-3 for 90 days; (3) T3A group, injected with D-galactose for 60 days followed by omega-3 administration for 30 days; and (4) T3B group, injected with D-galactose for 90 days. Blood samples were collected from anesthetized rats to estimate blood gases, and bronchoalveolar lavage fluid (BALF) samples were collected to estimate the levels of malondialdehyde (MDA), superoxide dismutase (SOD), Tumor necrosis factor-alfa (TNF-α), and Interleukin-1β (IL-1β) concentrations. Additionally, the quantity of mononuclear cells in the BALF and peritoneal fluid was counted. Furthermore, lung tissue samples were collected for histopathological investigation and to measure the expression of the lung IL-10 mRNA gene. The findings indicated a notable ($P < 0.05$) decrease in partial pressure of oxygen (PO_2), pH of blood and BALF SOD, along with a significant ($P < 0.05$) elevation in partial pressure of carbon dioxide (PCO_2), BALF MDA, BALF TNF-α, and BALF IL-1β levels in the T3B group. In addition, omega-3 significantly ($P < 0.05$) improved lung structural integrity, elevation in the expression of the IL-10 gene, enhanced blood gases, lung barrier integrity, and decreased oxidative stress. In conclusions, omega-3 has preventive action against lung dysfunctions in aged rats induced by D-galactose.

Keywords | Aging, Lung, BALF, Oxidative biomarkers, Omega-3, D-galactose

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INTRODUCTION

In humans and animals, aging is a contributing factor to the appearance of multiple diseases (Budinger *et al*., 2017). As we age, the lungs' mechanical properties and physical surroundings change, making them more susceptible to infections (Schneider *et al*., 2021; Hader *et al*., 2023). Respiratory system alterations by aging include irregularities in ventilation and gas exchange, a reduction in exercise tolerance, damage to the airway nerve, and a loss of respiratory muscle flexibility and strength (Schneider *et al*., 2021). The lung innate immune responses are essential for controlling respiratory immunity and serve as a crucial initial line of defense against infection; however, little is known about how innate respiratory immunity varies with aging (Haynes, 2020; Schneider *et al*., 2021). As the lung ages, the hematopoietic and non-hematopoietic cells would take part in the lung's innate immune response, in both structure and function (Krausgruber *et al*., 2020). A stronger pro-inflammatory phenotype has been reported

by some researchers, whereas others have documented an age-related decrease in the quantity and capabilities of alveolar macrophages (AMs), such as phagocytosis and cytokine generation (McQuattie-Pimentel *et al*., 2021).

The omega-3 polyunsaturated fatty acid (PUFA) family of fatty acids displays potential and positive effects in modifying a number of disease processes in the general population. This involves cardiovascular diseases, cardiac arrhythmias, and immunological and inflammatory disorders. The scientific community has shown a great deal of interest in omega-3(n-3) polyunsaturated fatty acids, especially eicosatetraenoic (EPA) and doceohexaenoic fatty acids (DHA) found in fish and fish oils, because dietary ω-3 fatty acids are essential for human health and cannot be produced in mammalian tissues (Silva-Neto *et al*., 2022).

EPA and DHA supplements have been shown to have ameliorative effect on lung aging by decreased oxidative stress (Korpak *et al*., 2024). Omega-3 fatty acids inhibit the pro-inflammatory nuclear factor-kappa B (NF-κB) pathway and increase the production of specialized pro-resolving lipid mediators (SPMs), including resolvins, which promote tissue resolution and are implicated in lung protection. These mechanisms contribute to reducing inflammation by modulating immune cell function and the inflammatory response (Tian *et al*., 2024). Omega-3 acts as an anti-inflammatories and pro-resolving properties through mediators such as protectins, maresins, and resolvins that support their protective effects against a range of illnesses (So *et al*., 2021). This study aimed to assess the role of omega-3 fatty acids in reducing the oxidative stress and lung inflammation in aging rats induced by D-galactose.

MATERIALS AND METHODS

Animal experiment

Omega-3 supplement capsules (Ferrer company, Spain) and 99.5% pure D-galactose (C6H12O6) with a molecular weight of 180.16g/mol (Rpicorp, USA) were utilized in the present study. The study used sixty adult male Wistar rats aged 4-4.5 months with body weight of 250–300 g. During the study, the animals were given free availability of food pellets and drinking water. Prior to the trial, the rats were acclimated for 15 days. The control group (T1) were treated by intraperitoneal (i/p) injections of normal saline and given orally oil soy bean, group T2 received 150 mg/kg of D-galactose (i/p) with 75 mg/kg omega-3 orally for 90 day, while group (T3A) received (i/p) injections of 150 mg/kg D-galactose for 60 day then given orally 75mg/kg omega-3 for 30 day and rats in group (T3B) received D-galactose (i/p) at the same dose for 90 day. Blood samples were collected after 60 and 90 days of the experiment by heart puncture technique. After all animals were anesthetized by ketamine at a dose of 100 mg/kg (i/p) and xylazine at a dose of 10 mg/kg (i/p), then sacrificed, and blood samples were collected and placed in heparinized tubes to estimate blood gases like partial pressure of oxygen and carbon dioxide (PO_2, PCO_2) and pH using blood gas analyzer tubes (OPTI Medical SN.OP6-005567, USA). Total RNA was extracted from the lung tissue using the genaid Korea RNA extraction kit, and samples were collected to evaluate the IL-10 mRNA gene expression. Gene expression was assessed using the IL-10 gene-specific forward primer (5-′ CCTTACTGCAGGACTTTAAGGGTT-3′)andreverse primer (5-CTGGGGCATCACTTCTACCAG-3’) (Hortobagyi *et al*., 2024). For this work, AddBio, Korea's AddScript RT-qPCR Syber master kit was used according to (Schmittgen and Livak, 2008) recommendations. In addition, qPCR was performed with an initial denaturation at 95 ˚C for 20 s, followed by 40 cycles of amplification (55 ˚C for 30 s and 72 ˚C for 30 s). The RT-qPCR results were normalized to the GAPDH gene and subsequently evaluated. Hematoxylin and eosin (H and E) were used to stain the tissue sections for histopathological analysis after the tissue had been fixed in 10% normal saline solution, embedded in paraffin wax, and thin sliced with 5 micrometers thickness (Suvarna *et al*., 2018). For preparation and cell counting in bronchoalveolar lavage fluid (BALF) and the peritoneal cavity, the trachea was carefully sutured before the surgical removal of the lungs and trachea to facilitate BALF collection. The trachea was then injected with PBS, and the supernatants containing cytokines were separated. The collected BALF was centrifuged. Following centrifugation, the cells in the tubes were counted using an autoanalyzer to determine the number of infiltrating mononuclear cells. The cytokines found in BALF, such as TNF-α and IL-1β, as well as BALF MDA and BALF SOD, were identified using the enzyme-linked immunosorbent test (ELISA) (Mohammed *et al*., 2020). In addition, peritoneal cavity exudates were analyzed following previous work (Alghetaa *et al*., 2023). In addition to measurement of lung vascular leakage as an intravenous injection of 100 microliters of Evans blue dye (Sigma-Aldrich, USA) was given to the experimental animals (five rats per group) two hours before their sacrifices (Al-Khaqani and Mohammed, 2024). Then the whole lung lobes were extracted and stored in formamide (Fisher Scientific, USA) at 37°C for 48 hours.

Statistical analysis

Statistical package for the social sciences SPSS computerized program was used to do statistical analysis, and the data was performed on the basis of analysis of variance (ANOVA) (one-way analysis of variance) in SPSS (Version 22). When comparing groups, the least

significant difference (LSD) was used. The level of P<0.05 was considered significant.

RESULTS AND DISCUSSION

Blood PO_2 and PCO_2

When evaluated against the control group, Figure 1A demonstrated a substantial drop in blood PO_2 for each treatment group. In contrast, there was a noticeable rise in blood PO_2 for the T2 and T3A groups as compared to the T3B group. A gradual significant (P ≤ 0.05) increase in oxygen concentration in T3A group as compared with T3B was observed. Conversely, the results in Figure 1B showed a statistically significant (P ≤ 0.05) elevation in blood PCO_2 levels in the aged group (T3B) in comprised with the rest of the experimental groups (T1, T2, T3A). Additionally, non-significant (P > 0.05) differences were observed between T2 and T3A. Moreover, blood pH results (Figure 1C) revealed significant reduction in its values in all study groups in comparison with the control group. Statistical analysis showed a significant increase in this parameter for the T2 group relative to T3A and T3B groups. The mean values were 7.33± 0.06, 7.25± 0.04 and 7.19 ± 0.01 for T2, T3A, and T3B respectively.

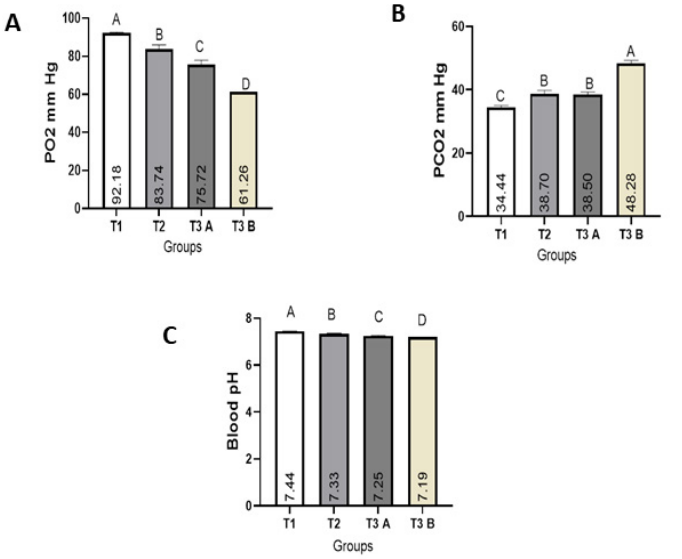

Figure 1: Effect of D-galactose, omega-3, and both on (A) PO_2, (B) PCO_2, and (C) blood pH of control and treated adult male rats after 90 days of treatment.
T1 Group = Control group. T2 Group = D-galactose and given omega-3 for 90 days. T3A Group = D-galactose for 60 days, then given orally omega-3 for 30 days. T3 B Group = D-galactose for 90 days. Uppercase letters depict significant differences among the groups. Significant threshold set on P<0.05.

Exposure to D-gal results in overexpression of the advanced glycation end product receptor (RAGE), which glycates adjacent proteins and creates AGE, which attaches to its receptor RAGE, starting NADPH oxidase and the generation of ROS (Zhang *et al*., 2023). These findings are consistent with Goodchild and DuRant (2020) who found that hemoglobin breakdown products attached to RBC membranes may indirectly alter the integrity of RBC membranes, causing significant alterations of RBC membrane redox status as seen by a decrease in the amount of sulfhydryl groups and an upsurge in lipid peroxidation activity which enhanced Hb glycation leading to early aging in erythrocytes (Remigante *et al*., 2022). These processes may cause damage and weaken red blood cells (Nagababu *et al*., 2013). Besides, D-galactose administration may induce the destruction of lung tissues, leading to an increase in inflammatory reactions, ROS induction, mitochondrial malfunction, and apoptosis (Goh *et al*., 2023). Inactivation of type II alveolar epithelial cells (AT2), causes irreversible tissue injury. Because these cells are unable to develop into Type I alveolar epithelial (AT1) cells, they impede the healing of damaged alveolar epithelium and eventually influence pulmonary gas exchange (Quan *et al*., 2024). In addition, along with aging lungs, the alveoli become larger and decrease their elastic recoil characteristics, which could make it easier for airways to close (Azman and Zakaria, 2019). These changes may include a decrease in gas exchange and an increase in the pH of blood. Omega-3 fatty acids may repair cell membrane abnormalities, decrease alveolar permeability, and decrease the production of adhesion molecules and pro-inflammatory cytokines (Huang *et al*., 2020).

Mononuclear cells counts in BALF and Peritoneal wash, and redox status of BALF

Figure 2A and 2B show that rats in the T3B group have significantly (P ≤ 0.05) more infiltrating mononuclear cells in their peritoneal fluid and BALF than rats in the T1, T2, and T3A groups. Additionally, when comparing T2 and T3A, the results showed insignificant (P ≤ 0.05) changes in the infiltrating mononuclear cells in BALF and intraperitoneal fluid. Furthermore, there was a noteworthy rise in T2 and T3A groups in comparison to the control group in both BALF and peritoneal wash. D-galactose-treated group (T3B) recorded a significant up regulation of BALF MDA values when compared within the others, as shown in the Figure 2C. However, there was a significant (P ≤ 0.05) decrease in BALF MDA concentrations in group T2 and T3A, in contrast to group T3B. Besides, the findings revealed non-significant (P > 0.05) differences in MDA concentration between T1 and T2 groups. In Figure 2D concerning the SOD concentration in BALF, group T2 recorded a high rise in BALF SOD concentration in comparison with T3A and T3B. The findings revealed a non-relevant distinction in BALF SOD level between T3A and T3B, as well as in T1 and T2, in this criterion.

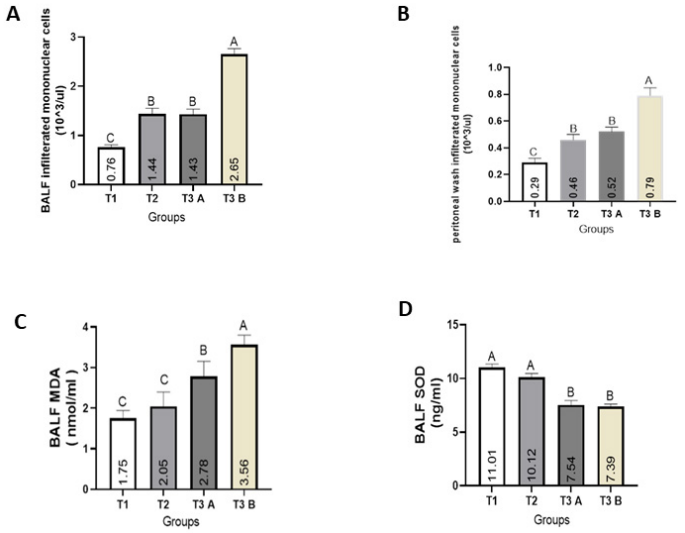

Figure 2: Effect of D-galactose, omega-3, and both on (A) infiltration of mononuclear cells in BALF and (B) infiltration of mononuclear cells in peritoneal wash, (C) BALF MDA, and (D) BALF SOD activity in treated adult male rats after 60 and 90 days.
T1 Group = Control group. T2 Group = D-galactose and given omega-3 for 90 days. T3A Group = D-galactose for 60 days, then given orally omega-3 for 30 days. T3B Group = D-galactose for 90 days. Uppercase letters depict significant differences among the groups. Significant threshold set on P<0.05.

Our results agree with (Ji *et al*., 2023), who reported that D-galactose induced aging in beagle dogs, suggesting that aging dogs have more oxidative stress with less antioxidant enzymes to handle. This stress results in serious lung tissue damage in the form of infiltration of inflammatory cells and alveolar wall breakdown. The production of pro-inflammatory substances from lung epithelial cells may also play a role in these alterations in the immune cell composition in BALF lungs (Mohammed*et al*., 2020; Cai *et al*., 2022). Administration of omega-3 supplements showed the greatest increase in antioxidant and anti-inflammatory profile of lung tissues via stimulating anti-inflammatory cells. Additionally, omega-3 plays a significant role in enhancing the immune system and stimulating the lymphocytes and neutrophils (Awad *et al*., 2015). Furthermore, omega-3 fatty acids can reduce inflammatory response by minimizing the effector T-cells as well as macrophages (Bodur *et al*., 2025). Furthermore, the exacerbating of oxidative biomarker MDA and reduced SOD levels in BALF due to D-galactose administration came aligned with our previous work (Al-Okaily, 2024). Oxidizing of D-gal at large concentrations into aldehydes and H_2O_2 contributed to the generation of ROS and LPO (Al-Tamemi and Al-Okaily, 2024). These results agreed with studies (Al-Kurdy, 2020; Feng and Huang, 2022; Khudair and Al-Okaily, 2022; Peng *et al*., 2023; García-Trejo *et al*., 2024) show a strong association between the generation of ROS (mostly H_2O_2) in cells and mitochondrial dysregulation or damage, via D-gal could be reduced galactitol leading to osmotic stress and disrupt redox status balance. Whereas our findings show that omega-3 supplementation increases the efficacy of lung antioxidant capacity and free radical scavenger activity, as confirmed by an increase in BALF SOD concentration, and decreasing LPO. The BALF redox status returned to near normal levels by improving total antioxidant capacity (Chautan *et al*., 1990).

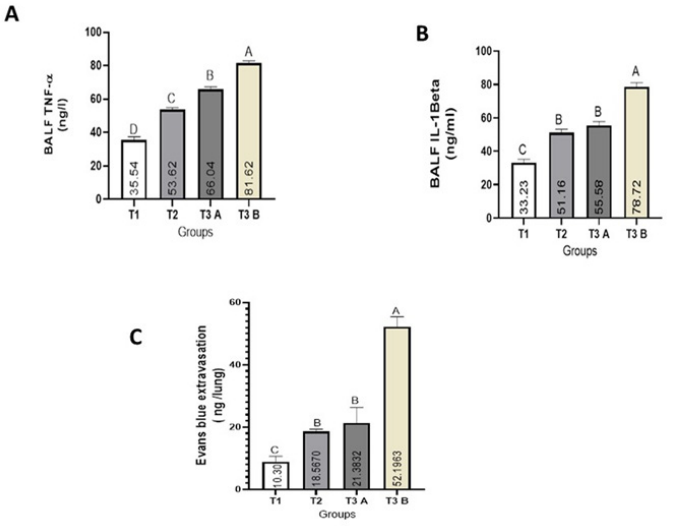

Figure 3: Effect of D-galactose, omega-3, and both on (A) BALF TNF-α and (B) BALF, IL-1β, and (C) lung-blood barrier integrity of treated adult male rats after 60 and 90 days.
T1 Group = Control group. T2 Group = D-galactose and given omega-3 for 90 days. T3A Group = D-galactose for 60 days, then given orally omega-3 for 30 days. T3B Group = D-galactose for 90 days. Uppercase letters depict significant differences among the groups. Significant threshold set on P<0.05.

Pro-inflammatory factors and lung tissue integrity

Figure 3A findings revealed a substantial ($P \leq 0.05$) rise in BALF TNF-α level for all groups that received therapy in contrast to the untreated group. Current results demonstrated that this value was substantially lower for T2, T3A groups as compared with T3B. In addition, TNF-α levels had been statistically ($P \leq 0.05$) declined in BALF of T3A group, while T3B had shown elevations in this cytokine. Figure 3B reveals that IL-1β cytokines in BALF were dramatically elevated in the three treated groups (T2, T3A, and T3B) compared to the control group. Additionally, non-significant differences in BALF IL-1β concentration between T2 and T3A were observed. As displayed in this figure, the administration of omega-3 to D-galactose stressed rats (T3A and T3B groups) showed a significant ($P \leq 0.05$) decrement in BALF IL-1β

B concentration versus T3B-treated rats. In the rat lungs of group T3B, the level of Evans blue extravasation was considerably rise than that of the T1, T2, and T3A groups, as illustrated in Figure 3C. Additionally, compared to the T1 group, T2 and T3A groups showed statistical rise of extravasated Evans blue dye concentrations. However, insignificant changes were recorded in the concentration of this dye between T2 and T3A groups.

The current study found that groups treated with D-galactose have profound proinflammatory cytokines, namely TNF-α and IL-1β in BALF. Inflammation is a main factor in inducing aging, and exorbitant inflammation will lead to the imbalance of homeostasis (Arosio *et al*., 2023). Inflammasomes generate IL-1β, which plays key role in mediating inflammation through activated macrophage (Lin *et al*., 2018; Alghetaa *et al*., 2023). Macrophages also generate different cytokines such as TNF, IL-1, IL-6, and IL-8, which damage tissue and cause cell death by binding to two receptors (TNFR1 and TNFR2), which play a pivotal role in organizing an inflammatory response (Wang *et al*., 2020). Additionally, D-gal produced chronic inflammation via the nuclear factor (NF)-κB signaling pathway by activating NF-κB in schwann cells and increasing the expression of pro-inflammatory cytokine genes such as TNF-α and IL-1β in cochlear tissues and decrease in anti-inflammatory gene like IL-10 (Wei *et al*., 2025). Omega-3 reduce inflammation by stabilizing inhibitor of nuclear factor-κB (IκB) and suppressing the activation of NF-κB signaling pathway, which consequently leads to down-regulated expression of tumor necrosis factor (TNF)-α and interleukin IL-1β along with up-regulated expression of IL-10 (Zou *et al*., 2025). This anti-inflammatory effect of omega-3 was in agreement with Bodur *et al*. (2025). Also, this effect can be attributed to decreased Nrf-2 activity as reported by El-Far *et al*. (2024). Also, omega-3 may lessen the detrimental effects of pro-inflammatory cytokines in lung BALF by decreasing production of inflammatory mediators originated form arachidonic acid such as prostaglandins, leukotrienes as well as thromboxane (Chen *et al*., 2021; Balachandar *et al*., 2023). In addition to forming a physical barrier, lung epithelial cells are essential for immune cell recruitment and pathogen identification. The major components of this barrier are claudin-1 and occludin, which serve as important markers for evaluating permeability and barrier function (Bustani *et al*., 2024). A significant elevation in concentration of Evans blue extravasation in rats receiving D-gal may be due to oxidative stress, causing breakdown of up lung-blood barrier (LBB), which accelerates age–related lung disease. This result was in agreement with (Hamady and Al-Okaily, 2022; Li *et al*., 2025). D-galactose can induce oxidative stress, increase levels of pro-inflammatory cytokines and tissue structural alterations, as well as increase the permeability of the barrier (Zhao *et al*., 2025). The results showed that omega-3 protected the blood-lung barrier by reducing its permeability, as indicated by a decrease in Evans blue extravasation concentration. The anti-inflammatory regulatory effects of EPA and DHA on cytokine dynamics and cellular mediator pathways may indirectly contribute to the preservation of the lung cellular barrier and improve the integrity and decrease its permeability (Hong *et al*., 2015; Chelakkot *et al*., 2018). Additionally, omega-3 inhibits the synthesis of pro-inflammatory cytokines and eicosanoids, prevents alterations in epithelial permeability, and stimulates the production of docosanoids and anti-inflammatory eicosanoids. Changes in the expression or activity of proteins involved in inflammatory signaling, such as nuclear factor kappa-light-chain-enhancer of activated B cells, have been linked to altered inflammatory indicators (Durkin *et al*., 2021).

IL-10 GENE EXPRESSION IN THE LUNG

IL-10 gene expression showed significant (P ≤ 0.05) down regulation in T3B group in comparison with the rest of the experimental groups (Figure 4). A notable rise in the fold change of the IL-10 gene expression was noted in group T2 when compared with the control (T1) and aged (T3B) groups.

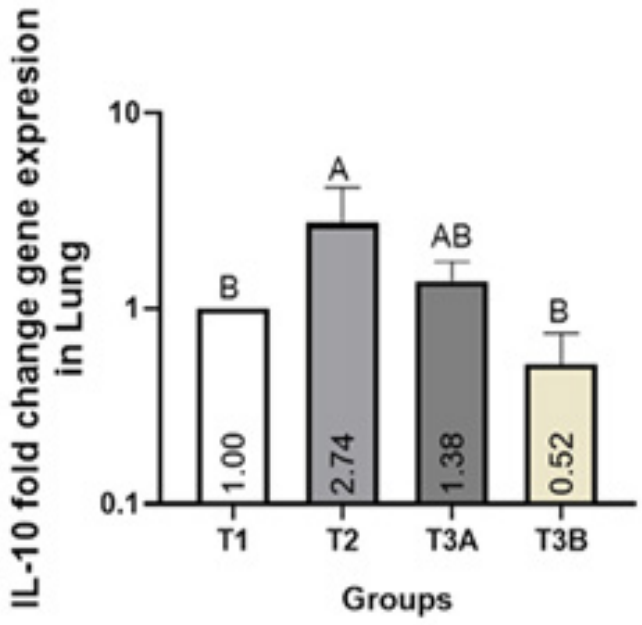

Figure 4: Effect of D-galactose, omega-3 and both on IL-10 gene expression in adult male rats lung tissue after 90 days.
T1 Group = Control group. T2 Group = D-galactose and given omega-3 for 90 days. T3A Group = D-galactose for 60 days, then given orally omega-3 for 30 days. T3B Group = D-galactose for 90 days. Uppercase letters depict significant differences among the groups. Significant threshold set on P<0.05.

Exposure to D-gal in T3B group promotes the expression of pro-inflammatory molecules and activates cytokines such as IL-1β and TNF-α and decrease anti-inflammatory cytokines like IL-10 that can induce oxidative stress and inflammation in lung, which are key molecular mechanisms of D- gal induced lung injury (Ma *et al*., 2024). Omega-3 fatty acids contain mediators that reduce inflammation called resolvins and protectins, which modify the balance between pro-inflammatory and anti-inflammatory cytokines, change the host's reaction to pulmonary infection, and influence the early course of infection. These mediators exhibit strong anti-inflammatory effects in various disease models, including lung injury (Liput *et al*., 2021). IL-10 expression can reduce numerous causes that lead to fibrosis, which is characterized by tissue damage caused by an excess of extracellular matrix components, such as collagen (Mohammed *et al*., 2020; Ahmed and Mohammed, 2022). Omega-3 fatty acids, especially EPA and DHA, have the ability to raise IL-10, an anti-inflammatory cytokine. It does this by lowering proinflammatory cytokines and encouraging inflammation-resolving pathways, which adds to omega-3 overall anti-inflammatory effects. Treatment of inflammatory and autoimmune diseases may benefit from the rise in IL-10, which is seen in a variety of cells and is associated with a more balanced immune response (Jannas-Vela *et al*., 2023).

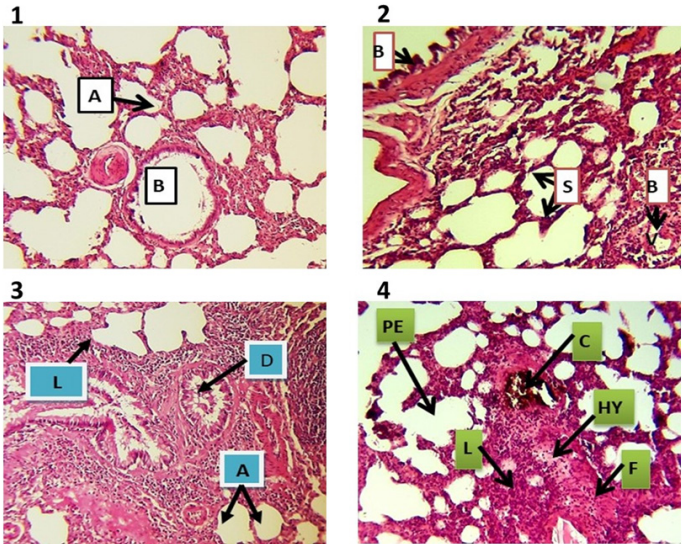

Figure 5: Histopathological changes in rat lung tissue. 1-control group, (A) Alveoli, (B)Bronchioles. 2-T2 group. (BV)Blood vessels, (B) Bronchioles, (S) Interstitial tissue. 3-T3A group. (D) Desquamation, (L) Lymphocytic cells, (A) Alveoli. 4- T3B group, (HY): hyaline degeneration, (F) Fibrosis, (L) inflammatory cells, (C) Congestion of blood vessels), (PE) Pulmonary emphysema. H and E stain X100.

Histopathological analysis

The results of microscopic examination of the lung tissue of rats of the control group showed there is normal pulmonary tissue which includes normal alveoli, which are lined by single alveolar cells and normal bronchioles (Figure 5). Also, the histological section of lung tissue in the rat of the T2 group shows normal blood vessels and normal columnar epithelium of bronchioles, thin interstitial tissue among alveoli (Figure 5). While the histological section of lung tissue in the rat of the T3A group shows there is desquamation of the epithelial cells that line bronchioles. Infiltration of lymphocytic and plasma cells in the pulmonary tissue. But there are normal thin-walled alveoli (Figure 5). In addition, the histological section of lung tissue in the rat of the T3B group shows there is hyaline degeneration with fibrosis and infiltration of inflammatory cells in the interstitial tissue of the lung, also there is congestion of blood vessels and pulmonary emphysema in alveoli (Figure 5).

Lung aging caused by D-galactose results in a decrease in the number of type II alveolar epithelial cells (AT2). By quickly changing the immunological environment in the lungs, damaged AT2 cells have the ability to recall the inflammatory cells to the alveolar spaces lead to inflammatory cells infiltrate, and many cytokines are produced (Katsura *et al*., 2019). Alveolar cells type-1 which regulate gas exchange, are able to transform into AT2 cells when they are wounded, are unable to heal themselves. The loss and degradation of AT2 stem cells by aging may have a direct effect on alveolar repair and the regeneration of epithelial tissue (Angelidis *et al*., 2019). Chronic inflammation, which has been connected to elevated oxidative stress and pro-inflammatory cytokines, is often the cause of lung fibrosis. Furthermore, a rise in hydroxyproline (Hyp) levels indicated that D-galactose treatment improved the lung fibrotic condition. As a result, in terms of fibrotic conditions, elevated oxidative stress, and low-grade chronic lung inflammation, D-galactose therapy appears to mimic the aging process (Makena *et al*., 2023; Al-Tamemi *et al*., 2024). The findings are in line with a study by Gammone *et al*. (2018) who showed that omega-3 therapy not only rapidly reduces the inflammatory response of lung tissue but also converts the inflammatory reaction-causing LTB4 series into the less active LTB5 series, which enhances pulmonary vascular permeability and dramatically lowers pulmonary edema. This is further validated by of results of lung scoring regarding emphysema, inflammatory cells, and fibrin (Figure 6) that indicated the protective effect of omega-3 in lung tissues. Additionally, omega-3 fatty acids can limit the body's over reactive inflammatory response during infections and injuries, which in turn regulates the activity of lipid mediators, cytokines, and endothelial cells (Gutiérrez-Delgado *et al*., 2019).

CONCLUSION

The current study indicates that omega-3 exerts enhanced

protective effects against aging in D-galactose-induced rat models. Overall, these findings indicate that oral administration of omega-3 supplements after the induction of aging in animal models exhibits strong anti-inflammatory and antioxidant activities. Moreover, the current results highlight the potential of omega-3 as a promising anti-aging therapeutic approach, particularly in the context of lung aging and related diseases.

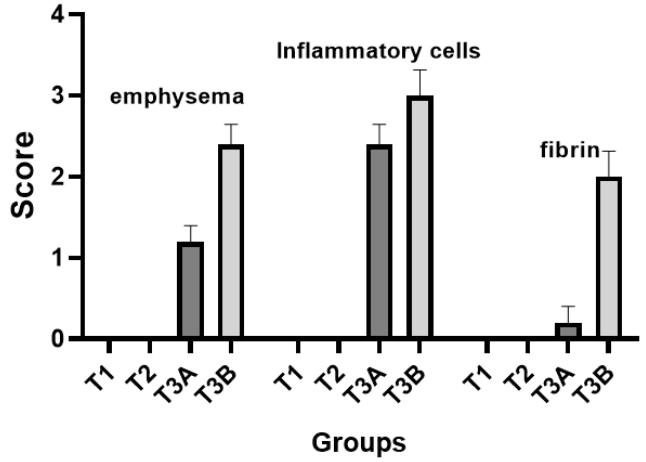

Figure 6: Quantitative histopathology for the lung with scoring for Emphysema, inflammatory cells, and fibrin.

ACKNOWLEDGMENT

Not applicable.

NOVELTY STATEMENT

This research article provides deep insight into the physiological roles of the aging process induced by D-galactose in the modulation of vital organs' function, such as the lungs.

AUTHOR'S CONTRIBUTION

BA and AM designed, conceptualization, reviewed the draft, and the last version of this manuscript. BS did all the experiments and wrote the draft, revised and wrote the last version of this manuscript. BA, AM, BS, and NA have reviewed and approved the submitted version of the manuscript.

Data availability

The data is available from the corresponding author upon reasonable request.

Ethical approval

In full compliance with the ethical guidelines for animal research, all animals used in this study were cared for and treated at the College of Veterinary Medicine, University of Baghdad. The ethical permission was got prior to experimentation (Approval No: P.G./552, date 10-3-2024).

Generative AI and AI-assisted technology statement

The authors declare that no Genrative AI was used in the creation of this manuscript.

Conflict of interest

The authors have declared no conflict of interest.

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