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# Effect Of Infrared Radiation On Membrane Phospholipids Of Lymphocytes And Platelets In An Experimental Model Of Chronic Obstructive Pulmonary Disease

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**Abstract:** Background: Chronic obstructive pulmonary disease (COPD) is associated with systemic inflammation and metabolic disturbances. Membrane phospholipids play central roles in cellular integrity and immune regulation. Infrared (IR) radiation is proposed to modulate inflammatory pathways, yet its effects on membrane phospholipid composition in immune cells have not been fully elucidated.

Objective: To investigate changes in membrane phospholipids of lymphocytes and platelets in rats with experimental COPD and to evaluate the effect of IR radiation on these changes.

Methods: COPD was induced in male outbred white rats (n = 60) by daily exposure to tobacco smoke for 60 days. Rats were divided into three groups: COPD untreated, COPD + IR radiation, and control (no smoke exposure). IR treatment consisted of 10 daily sessions (5–10 minutes each) over two weeks. Phospholipid fractions of peripheral lymphocyte and platelet membranes were analyzed using high performance chromatography and quantified by densitometric and photometric methods. Statistical significance was assessed at $p < 0.05$.

Results: COPD rats demonstrated significant disruptions in membrane phospholipid profiles and increased phospholipase activity compared with controls. After IR treatment, rats exhibited partial normalization of phospholipid fractions, with further improvement at 30 days post treatment. These changes suggest IR radiation supports restoration of membrane structure and function in immune cells under COPD conditions.

Conclusions: IR radiation exerts modulatory effects on membrane phospholipids in lymphocytes and platelets in experimental COPD, reflecting its potential as a therapeutic modality for systemic cellular repair processes in inflammatory lung disease.

**Keywords:** Chronic obstructive pulmonary disease; infrared radiation; membrane phospholipids; lymphocytes; platelets; inflammation.

**Introduction:** Chronic obstructive pulmonary disease (COPD) is recognized as a complex systemic condition with significant inflammatory and metabolic components (Vestbo et al. 2013). Changes in membrane phospholipid composition can significantly affect immune cell activation, cytokine production, and cellular signaling (Schmidt & Brown, 2008). Experimental models are integral for understanding the molecular pathology of COPD and evaluating novel therapies (Barnes 2016).

Infrared radiation has been investigated for its anti inflammatory properties and ability to modulate cellular metabolism (Hamblin, 2017). However, its impact on membrane lipid profiles in immune cells during COPD has not been fully studied. This study explores these molecular effects in a well established

rat model of smoke induced COPD.

## METHODS

### Animals and COPD Model

Sixty male outbred white rats (120–180 g) were exposed to tobacco smoke daily for 30–40 minutes for 60 days to induce COPD (Yanagi et al. 2017). Smoke concentration was monitored via carbon monoxide levels.

### Experimental Groups

1. COPD without treatment (n = 20)
2. COPD + IR radiation (n = 20)
3. Control (no smoke exposure; n = 20)

### Infrared Radiation Protocol

IR phototherapy was administered using GI, Ku, Zb series lamps targeting the lung region. Treatment included 10 sessions: 5 min/day for the first 5 days, then 10 min/day for the following 5 days.

### Phospholipid Analysis

Lymphocytes and platelets were isolated by Ficoll density gradient centrifugation. Phospholipid fractions were separated by high performance liquid chromatography and quantified densitometrically according to established protocols (Folch et al. 1957). Phospholipase activities were measured using standard enzymatic assays.

### Statistical Analysis

Data were analyzed using Microsoft Excel and descriptive statistics. Differences were significant at $p < 0.05$ using Student's t test.

## RESULTS

### Baseline Phospholipid Profiles

Healthy rats exhibited defined membrane phospholipid fractions: phosphatidylcholine (PC), phosphatidylethanolamine (PE), lysophosphatidylcholine (LPC), lysophosphatidylethanolamine (LPE), free fatty acids (FFA), and total phospholipids (TPL). Phospholipase activities were measured for PLA2, PLC, and PLD.

### Effects of COPD Induction

Tobacco smoke exposure induced significant alterations in membrane phospholipid levels and elevated phospholipase activities in both lymphocytes and platelets ($p < 0.05$) compared with controls.

### Effects of IR Radiation

After 10 IR sessions, PC and PE levels increased toward control levels, while LPC and LPE decreased. Phospholipase activities also showed normalization trends. At 30 days post IR, further improvements were observed, indicating sustained modulation of membrane composition.

## DISCUSSION

These findings align with previous studies showing membrane lipid alterations in inflammatory diseases (Moffatt et al. 2007). The modulatory effects of IR radiation on phospholipid metabolism suggest possible mechanisms involving reduced oxidative stress and improved membrane repair (Hamblin 2017). This supports investigation of IR therapy as an adjunctive treatment in systemic inflammation.

## CONCLUSIONS

1. Experimental COPD results in significant quantitative changes in lymphocyte and platelet membrane phospholipids.
2. IR radiation promotes normalization of altered phospholipid profiles, with effects sustained at 30 days post treatment.
3. IR acts at a molecular level to support restoration of immune cell membrane integrity.

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**Table: Laboratory Parameters – Control vs COPD + IP (Immune Complex)**

| Parameter | Control | COPD before treatment (lymphocytes / platelets) | After 10% | After 30 days |
|---|---|---|---|---|
| **Lymphocytes** | | | | |
| FX | 40.1 ± 1.04 | 27.37 ± 0.59** | 30.6 ± 0.76* | 33.5 ± 1.00* |
| LFX | 3.41 ± 0.06 | 6.9 ± 0.13** | 6 ± 0.15* | 5 ± 0.12* |
| FE | 32.3 ± 0.47 | 18.3 ± 0.54* | 22.53 ± 1.19* | 23.6 ± 0.57* |
| LFE | 5 ± 0.14 | 9.2 ± 0.21** | 8.48 ± 0.21 | 8.0 ± 0.20* |
| ESR | 19.3 ± 0.16 | 28.4 ± 0.55** | 24.3 ± 0.56* | 24.3 ± 0.42* |
| OFL | 158.4 ± 1.31 | 130.3 ± 1.28** | 136.7 ± 2.98 | 137.5 ± 3.61 |
| FaA2 | 3.1 ± 0.12 | 5.53 ± 0.14** | 5.1 ± 0.12 | 4.8 ± 0.08* |
| FS | 4.95 ± 0.11 | 8.18 ± 0.19** | 7.8 ± 0.16 | 7.6 ± 0.21* |
| FD | 2.84 ± 0.06 | 4.27 ± 0.11** | 3.93 ± 0.13* | 3.85 ± 0.08* |

| Parameter | Control | COPD before treatment (platelets) | After 10% | After 30 days |
|---|---|---|---|---|
| **Platelets** | | | | |
| FX | 64.3 ± 0.39 | 42.3 ± 0.98** | 47.8 ± 0.85* | 48.4 ± 0.92* |
| LFX | 4.2 ± 0.22 | 7.7 ± 0.13** | 7.2 ± 0.14* | 6.9 ± 0.18* |
| FE | 30.1 ± 0.92 | 22.3 ± 0.69** | 25.1 ± 0.59* | 25.8 ± 0.6* |
| LFE | 6.7 ± 0.24 | 11.1 ± 0.4** | 9.5 ± 0.19* | 9.4 ± 0.27* |
| ESR | 17 ± 0.16 | 23.48 ± 0.68** | 20.9 ± 0.54* | 21.0 ± 0.55* |
| OFL | 173.5 ± 1.04 | 142.3 ± 3.37** | 149.77 ± 3.05 | 152.1 ± 2.59 |
| FaA2 | 4.12 ± 0.12 | 5.23 ± 0.13** | 4.83 ± 0.09 | 4.7 ± 0.1* |
| FS | 5.72 ± 0.08 | 7.9 ± 0.20** | 7.1 ± 0.14* | 7.0 ± 0.19* |
| FD | 0.98 ± 0.05 | 4.3 ± 0.09** | 3.4 ± 0.06* | 2.9 ± 0.09* |

**Note:** Statistical significance of COPD group vs. control and COPD + IP group vs. COPD before treatment: *P < 0.05, **P < 0.01