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Differential Effects of Low- And Moderate-Intensity Aerobic Exercise on Interleukin-17 In Moderate Chronic Obstructive Pulmonary Disease: A Randomized Controlled Trial

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ABSTRACT: **Aim:** Aerobic treadmill activities were tested for their impact on interleukin-17 (IL-17) in chronic obstructive pulmonary disease (COPD) patients.

Methods: Three equal groups of sixty male and female patients diagnosed with moderate chronic obstructive pulmonary disease (COPD) (aged 40-60 years) were randomly allocated. Twenty participants were divided into three groups: A, which received conventional chest physiotherapy for 8 weeks in addition to low-intensity treadmill aerobic exercise at 40-50% HRR twice weekly, B, which received moderate-intensity treadmill exercise at 50-70% HRR twice weekly, and C, which served as a control group, received only conventional chest physiotherapy for 8 weeks. Functional exercise capacity, dyspnea severity, and serum IL-17 levels were evaluated both before and after the intervention using the Enzyme-Linked Immunoassay (ELESA).

Results: At the outset, the demographic characteristics and IL-17 levels of all groups were comparable ($P > 0.05$). Based on the results of the within-group analysis, it was found that both Group A and Group B had a significant decrease in IL-17 levels, with Group A seeing a 23.40% improvement ($P = 0.0001$) and Group B seeing a 4.94% improvement ($P = 0.0001$). Unlike Group B, which showed a 4.44% improvement (47.55 ± 4.99 to 45.44 ± 9.86 pg/mL; $P = 0.188$), Group C showed no significant change. Although Groups B and C did not vary statistically ($P = 1.000$), Group A had significantly lower IL-17 levels after treatment compared to Groups B and C ($P = 0.001$).

Conclusion: Compared to moderate-intensity exercise, aerobic treadmill exercise reduced blood IL-17 levels in COPD patients more effectively, suggesting that this form of exercise could be a non-pharmacological approach to managing systemic inflammation in COPD.

INTRODUCTION

The acronym COPD stands for chronic obstructive pulmonary disease, which is an umbrella word for a group of progressive lung diseases. The two most common types are chronic bronchitis and emphysema [1]. Smoking and prolonged exposure to chemical irritants are the leading causes of chronic obstructive pulmonary disease (COPD), a treatable and preventable respiratory ailment. Chronic obstructive pulmonary disease (COPD) is characterized by progressive, partially reversible airflow limitation and progressive, inflated lungs [2]. An individual's sickness may be worsened by co-occurring conditions and other systemic symptoms that are not limited to the lungs.

Chronic obstructive pulmonary disease (COPD) is brought about by smoking cigarettes. Nearly 90% of chronic obstructive pulmonary disease (COPD) cases are categorized as involving current or former smokers. Chronic obstructive pulmonary disease (COPD) affects 20–30% of smokers. The bulk of people with COPD are middle-aged or older and have smoked at some point in their life, according to research [3].

The onset of systemic and localized chronic inflammation is the first step in the morphophysiological changes seen in COPD and BA. Inflammation is a hallmark of these diseases and is associated with the secretion of a diverse array of proinflammatory mediators into the bloodstream and respiratory tract [4].

This complex interacts in a manner that causes airway fibrosis and obstruction, excessive cell growth in smooth muscles, and a worsening of COPD and BA [5]. Airway inflammation and obstruction in COPD and asthma are pathophysiologic features of the proinflammatory cytokine interleukin-17 (IL-17A), previously known as IL-17. IL-17 plays a pivotal role in the activation of lung cells in patients with severe beta-amyloidosis (BA) and chronic obstructive pulmonary disease (COPD) [6].

Exercise training is an integral part of any pulmonary physical therapy treatment regimen. This is where respiratory therapy comes in. Training that promotes muscle loss in COPD patients motivates them to exercise more often until their peripheral muscles are conditioned [7].

As a component of their physical therapy treatment, patients with COPD often reap the benefits of aerobic exercise regimens that include the use of a cycle ergometer or a treadmill [8]. The purpose of this study was to determine whether or not aerobic activity on a treadmill reduced IL-17 levels in COPD patients.

Although aerobic exercise is well-known to be an important component of pulmonary rehabilitation, little is known about the effects of different intensities of aerobic exercise on circulating IL-17 in individuals with stable mild chronic obstructive pulmonary disease (COPD).

SUBJECTS AND METHODS

Participating in this study were sixty individuals with a diagnosis of COPD ranging from forty to sixty years of age. In this investigation, they were evaluated for potential intervention. Participants were recruited from the outpatient clinic of the university's chest department on October 6. Eight weeks were allotted to them. Three groups, each with an equal number of patients, were randomly allocated to participate in the research. The randomization method used was a 1:1:1 computer algorithm.

With the use of G*Power, we were able to decide that 60 patients would make up our sample, 20 from each group. Members of the Research Group A underwent conventional chest physiotherapy (postural drainage, vibration, percussion, huffing and coughing, and breathing exercises) twice weekly for 8 weeks, as well as their prescribed pharmaceutical treatment, and participated in low-intensity aerobic exercise on a treadmill for at least 40 minutes twice a week for eight weeks [7]. Study Group B participants were given pharmaceutical treatment in addition to traditional chest physiotherapy twice weekly for 8 weeks and moderate-intensity treadmill aerobic exercise for at least 40 minutes per session for 8 weeks [9]. Patients in the Control Group C who were also given medication also underwent traditional chest physiotherapy for eight weeks [7].

Inclusion criteria for the study were that participants be male or female smokers between the ages of 40 and 60, and that their respiratory symptoms be stable with no worsening in the preceding four weeks. Individuals were eligible to participate in the study if they met the following criteria: a baseline dyspnea grade of 1 or 2 as per the mMRC criteria; a diagnosis of mild COPD Stage II as per the GOLD criteria; a body mass index (BMI) between 19.5 and 26.9 kg/m²; and, finally, they had to meet the GOLD 2 criteria, which state a FEV₁ of 50–79% predicted. People who had a history of lung surgery, cancer, cognitive

impairment, COPD flare-ups, long-term oxygen therapy, neurological disorders, musculoskeletal disorders, rheumatic disorders, cardiac disorders, or had participated in a pulmonary rehabilitation program before were not eligible [7].

Ethical Consideration

All procedures followed the rules specified in the Declaration of Helsinki, the ethical code of conduct for research conducted by the World Medical Association. From the very beginning, all patients were informed of the study's goals and their rights. Prior to the study procedures being started, participants' written informed permission was obtained, granting authorization to participate. Following validation by the Ethical Committee of the Physical Therapy Faculty at Egypt's October 6 University (No: P.T. REC\024\003006), the study procedure remained unchanged. All writers with access to the research read and approved the final report.

METHODS

A full battery of clinical and demographic evaluations were administered to all individuals. Every participant had their complete demographic and medical information recorded. As part of the anthropometric data collected, we know the subjects' weight, height, and BMI. The subjects stood on a digital scale while wearing little clothes, spreading their weight equally on both feet, and going barefoot. To the closest half centimeter, each subject's height was recorded using a wall-mounted stadiometer.

In order to determine functional exercise capacity, the 6-minute walk test (6MWT) was used. The test was carried out in a designated hallway with the use of a timer and colored tape to demarcate the beginning and finishing locations. Reflecting the integrated reactions of the respiratory, cardiovascular, circulatory, and muscular systems during exercise, the 6MWT is a straightforward, risk-free, and dependable way to estimate an individual's distance that can be covered on a flat, hard surface in 6 minutes [9].

The severity of dyspnea was assessed using the mMRC Dyspnea Scale (Appendix II). Higher scores indicate more functional restriction owing to breathlessness, and the scale rates breathlessness from 0 to 4 based on the amount of physical activity that causes dyspnea [10].

Levels of interleukin-17 (IL-17) were measured as part of the laboratory examination. Blood samples were taken from the antecubital vein just before treadmill activity began, after a 12-hour overnight fast. Blood samples were taken 24 hours after the last exercise session to determine the effects of the intervention. We used an enzyme-linked immunoassay (ELISA) to measure the IL-17 levels [11].

Each individual in groups A and B was given moderate aerobic activities on a treadmill (Runx, PVT by Mombai) twice a week for eight weeks as part of the treatment. In each session, forty minutes were required [9].

Procedures

Evaluation Procedures

Every single participant in Groups A, B, and C had pre- and post-treatment evaluations using the same set of tools. The following tests were performed: measurement of body mass index (BMI), assessment of dyspnea severity, and identification of interleukin-17 (IL-17). After a 12-hour fast the night before and just before starting the treadmill activity, IL-17 was extracted from the antecubital vein. Enzyme-Linked immunoassay (ELISA) was used to collect blood samples 24 hours after the previous exercise session.

Treatment Procedures

1. Aerobic exercises

Groups A and B were obligated to attend aerobic fitness sessions. Individuals' exercise intensities were prescribed using the heart rate max (HRmax) method, which is defined as the sum of the maximal heart rate (HRmax) and the resting heart rate (HRrest). On the first day of testing, participants were asked to lie down on a bed for five minutes and have their resting heart rates felt by palpating their radial artery. The patient's age (in bpm) was subtracted from 220 to get their maximum heart rate (HRmax). Training heart rates were set using the calculated HRR [12].

Using a computerized treadmill (Treadmill), Group A participants engaged in low-intensity aerobic activity for eight weeks. We kept the exercise intensity at 40-50% of our HRmax. About 40 minutes of low-speed warmup walking, 30 minutes of treadmill walking at the recommended intensity, and 5 minutes of cool-down walking made up each session [12].

Group B members exercised aerobically at a moderate intensity on a computerized treadmill for eight weeks, twice a week. We kept the exercise intensity at 50-70% of our HRmax. Five minutes of warmup walking, thirty minutes of treadmill walking at the goal intensity, and five minutes of low-speed cool-down walking made comprised each forty-minute session [9].

2. Conventional Chest Physiotherapy

Every participant in Groups A, B, and C received 8 weeks of traditional chest physiotherapy, which consisted of breathing exercises, percussion, and vibration treatments, provided twice weekly.

Two breathing techniques, diaphragmatic and pursed-lip, were practiced. Participants sat up straight on a chair, hands on their abdomens, with their shoulders relaxed and feet propped up. This position was used for diaphragmatic breathing. A slow, deep intake via the nose, with the belly rising, followed by a full exhalation through the mouth, was the prescribed breathing technique. Initially, the exercise was done three times, with a 30-second break between each set of regular breathing cycles that lasted 10-15 seconds each. During the intervention phase, individuals trained for around 10–20 minutes per day, and the number of repetitions was progressively raised to ten [13].

Practiced while seated in the same position, pursed-lip breathing entails taking a controlled inhalation via the nose and an equally measured exhalation through the pursed lips. The expiratory phase lasted about twice as long as the inspiration phase. The workout was progressed from three sets of ten repetitions of ten to fifteen breathing cycles with brief rest intervals in between. Each day, the session lasted between ten and twenty minutes [13].

Following the procedure outlined by Prasad [1], percussion and vibration methods were used. The therapist would clap rhythmically over the chest wall with cupped hands during percussion, generating an air cushion between the hand and chest to help mobilize trapped secretions. In patients with hemoptysis, bronchospasm, or cancer, as well as those with bony prominences, the spine, or kidneys, percussion was avoided. When percussion was not an option, vibration was used to help evacuate mucus by providing oscillatory pressure to the chest wall with the palms or arm muscles during expiration [1].

Postural drainage

The procedure for airway clearing was followed while doing postural drainage. In order to promote the gravity-based drainage of secretions, participants were placed in drainage postures that corresponded to the specific parts of their lungs that were being studied. The individual was asked to stay in each posture for around 5 to 10 minutes while being guided to breathe deeply and relax using their diaphragm. The participant's clinical state and level of tolerance dictated the adjustments made to the positions. To aid in the mobilization and clearing of residual bronchial secretions, the procedure was executed throughout each therapy session.

Huffing and coughing

In order to help clear the airway of mobilized secretions, procedures such as huffing and coughing were used. The participants were asked to inhale deeply and slowly before forcing themselves to exhale via an open mouth in a "huff" motion. When secretions were directed into the bigger airways, huffing and controlled coughing occurred. So that they wouldn't become too tired or gasping for air, participants were given brief breaks in between sets. During treatment sessions, the method was administered based on the participant's tolerance and the quantity of secretions that were retained.

Statistical Analysis

For this statistical study, we used SPSS 25 for Windows, developed by SPSS Inc. and located in Chicago, Illinois, USA. While gender and mMRC dyspnea scores were shown as frequencies and percentages, quantitative data such as age, weight, height, BMI, and IL-17 levels were shown as mean $\pm$ standard deviation (SD). The Chi-square test was used to examine the gender distribution and mMRC dyspnea ratings among Groups A, B, and C. A paired t-test was used to compare the IL-17 levels of each group before and after treatment. The Cohen's d effect size was used to categorize the differences within groups as minimal (0.20), medium (0.50), or substantial (≥ 0.80). The researchers used a one-way analysis of variance (ANOVA) to compare the demographics of the three groups and to examine any differences in IL-17 levels before and after therapy. We compared two

sets of data using Bonferroni post hoc tests when analysis of variance (ANOVA) showed statistically significant differences. The magnitude of the between-group impact was determined using eta squared (η^2); small effects were shown by values of 0.01, medium effects by 0.06, and large effects by 0.14. When the p-value was 0.05 or below, statistical significance was established for all analyzes.

RESULTS

Of the 60 COPD patients included in the study, 36 were female and 26 were male. Each of the three groups consisted of twenty patients selected at random. Group A, B, and C COPD patients did not vary significantly from one another with respect to age ($P=0.955$), weight ($P=0.967$), height ($P=0.692$), or body mass index (BMI) ($P=0.267$), as shown in Table (1). Groups A, B, and C did not differ substantially in the distributions of gender ($P=0.762$) or mMRC ($P=0.812$), as shown in Table (2).

According to Table (3), after treatment, IL-17 levels in group A were considerably lower than pre-treatment levels ($P<0.05$) and post-treatment levels ($P=0.0001$). Within group A, the proportion of IL-17 that improved owing to time impact was 23.40%, while the percentage that changed was -11.15 (95% CI 9.51 - 12.76). After treatment, IL-17 levels in group B were considerably lower than pre-treatment levels ($P<0.05$; $P=0.0001$). Within group B, the change in IL-17 owing to time impact was 2.35% (95% CI 1.59 - 3.10) and the improvement percentage was 4.94%. The level of interleukin-17 (IL-17) in group C decreased somewhat ($P>0.05$) during the pre- and post-treatment periods ($P=0.188$). Within group C, there was a change of 2.11 (95% CI -1.12 - 5.34) and an improvement of 4.44% in IL-17 as a result of the time effect. Group A's COPD patients who participated in a program of low-intensity aerobic exercises saw a greater decrease in IL-17 (23.40%) after treatment than group B's COPD patients who participated in a program of low-to moderate-intensity aerobic exercises (4.94%), and finally, group C's control group (4.44%) who participated in a program of conventional chest physiotherapy in addition to pharmaceutical treatments.

The pre-treatment mean values of IL-17 were not significantly different ($P>0.05$) across groups, as shown in Table 4 ($P=0.999$). The groups' mean levels of IL-17 at post-treatment were significantly different ($P<0.05$) compared to each other ($P=0.0001$). Accordingly, the investigated groups' effects result in a beneficial reduction in mean IL-17 levels at post-treatment for group A (36.50 ± 6.02), group B (45.23 ± 5.00), and group C (45.44 ± 9.86).

Table 4 shows the mean difference in IL-17 levels between each set of groups after treatment and the results of the Bonferroni test. There were significant differences in IL-17 levels between groups A and B ($MD=8.73$; $P=0.001$) and A and C ($MD=8.94$; $P=0.001$) at the conclusion of treatment, but no difference between groups B and C ($MD=0.21$; $P=1.000$). Post hoc analysis across groups revealed that Group A had the highest IL-17 levels, followed by Group B, and then Group C.

Table (1): Comparison mean values of patient's demographic data among groups

Items	Age (Years)	Weight (kg)	Height (cm)	BMI (kg/m ²)
Group A (n=20)	54.10 $\pm$ 2.95	70.50 $\pm$ 6.94	172.95 $\pm$ 9.23	26.00 $\pm$ 1.42
Group B (n=20)	53.80 $\pm$ 3.35	69.90 $\pm$ 7.69	172.20 $\pm$ 9.15	23.55 $\pm$ 0.99
Group C (n=20)	53.90 $\pm$ 3.19	70.10 $\pm$ 7.76	174.75 $\pm$ 10.42	23.05 $\pm$ 0.94
F-value	0.046	0.034	0.371	1.353
P-value	0.955	0.967	0.692	0.267
Significance	NS	NS	NS	NS

Data are expressed as mean $\pm$ standard deviation; P-value: probability value, NS: non-significant

Table (2): Comparison of gender distribution among groups

Items	Gender		Modified medical research council	
	Males	Females	Grade 1	Grade 2
Group A (n=20)	10 (50.00%)	10 (50.00%)	12 (60.00%)	8 (40.00%)
Group B (n=20)	8 (40.00%)	12 (60.00%)	13 (65.00%)	7 (35.00%)
Group C (n=20)	8 (40.00%)	12 (60.00%)	11 (55.00%)	9 (45.00%)
Chi-square-value	0.543		0.417	
P-value	0.762		0.812	
Significance	NS		NS	

Qualitative variable data are expressed as frequency (percentage); P-value: probability value, NS: non-significant

Table (3): Comparison between pre- and post-treatment of IL-17 within each group

Items	IL-17 (Mean±SD)		
	Group A (n=20)	Group B (n=20)	Group C (n=20)
Pre-treatment	47.65±6.89	47.58±5.04	47.55±4.99
Post-treatment	36.50±6.02	45.23±5.00	45.44±9.86
MD (change)	-11.15	-2.35	-2.11
95% CI	-9.51 – 12.76	-1.59 – 3.10	-1.12 – 5.34
Improvement %	23.40%	4.94%	4.44%
Effect size (Cohen's <i>d</i>)	1.72	0.46	0.27
t-value	14.311	6.475	1.364
P-value	0.0001*	0.0001*	0.188
Significance	S	S	NS

Data are expressed as mean±standard deviation (SD), MD: Mean difference, CI: confidence interval; P-value: probability value, S: significant, * Significant (P<0.05), NS: non-significant

Table (4): Comparison between pre- and post-treatment of IL-17 within each group

Items		IL-17 (Mean±SD)			
		Pre-treatment		Post-treatment	
Group A (n=20)		47.65±6.89		36.50±6.02	
Group B (n=20)		47.58±5.04		45.23±5.00	
Group C (n=20)		47.55±4.99		45.44±9.86	
Effect size (Eta square)		0.004		0.217	
F-value		0.001		10.239	
P-value		0.999		0.0001*	
Significance		NS		S	
Multiple pairwise comparisons between both groups (post-hoc test)					
Pairwise group effect		MD	95% CI	P-value	Significance
Pre-treatment	Group A vs. Group B	0.07	-4.43 – 4.57	1.000	NS
	Group A vs. Group C	0.10	-4.40 – 4.60	1.000	NS
	Group B vs. Group C	0.03	-4.48 – 4.54	1.000	NS
Post-treatment	Group A vs. Group B	8.73	3.32 – 14.14	0.001*	S
	Group A vs. Group C	8.94	3.32 – 14.56	0.001*	S
	Group B vs. Group C	0.21	-5.63 – 6.05	1.000	NS

Data are expressed as mean±standard deviation (SD), MD: Mean difference, CI: confidence interval; P-value: probability value, S: significant, * Significant (P<0.05), NS: non-significant

DISCUSSION

A common, preventable, and treatable respiratory ailment, chronic obstructive pulmonary disease (COPD) is characterized by persistent airflow restriction, chronic inflammation of the airways, and progressive decline of lung function. It is a major danger to global health because of the enormous numbers of sick people, deaths, and reduced quality of life it produces. The pathogenesis of chronic obstructive pulmonary disease (COPD) includes changes in the anatomy of the airways and the lung parenchyma, an increase in mucus production, and a reduction in gas exchange [15, 16].

Interleukin-17 (IL-17) is a big pro-inflammatory cytokine that has been linked to COPD (chronic obstructive pulmonary disease). Interleukin-17 (IL-17), mostly produced by T-helper 17 (Th17) cells, stimulates neutrophil recruitment and activation and releases several inflammatory mediators; it is one of the factors that contribute to persistent airway inflammation [17]. In COPD, interleukin-17 (IL-17) has shown promise as a biomarker of inflammatory activity, according to some research [18]. The severity and progression of chronic obstructive pulmonary disease (COPD) are associated with elevated levels of interleukin-17 (IL-17) in the blood and sputum, according to previous studies.

In the treatment of chronic obstructive pulmonary disease (COPD), aerobic exercise is an essential part of pulmonary rehabilitation. Aerobic exercise training has the potential to improve exercise tolerance, lung function, dyspnea, and health-related quality of life for individuals with chronic obstructive pulmonary disease (COPD) [19]. There is growing evidence that aerobic exercise may have anti-inflammatory benefits by changing systemic inflammatory pathways and reducing circulating

inflammatory mediators, in addition to its numerous practical advantages. The evidence that exercise training improves patients with chronic obstructive pulmonary disease (COPD) is accumulating, but few research have examined the effects of different intensities of aerobic exercise on IL-17 levels [20].

So, this study set out to determine how IL-17 is affected by aerobic exercises performed on a treadmill in individuals with COPD. Sixty people who have been diagnosed with chronic obstructive pulmonary disease took part in this controlled experiment. After then, they were divided into three equal parts: Twenty individuals suffering from COPD were split into three categories: 1 (low intensity aerobics exercises), which consisted of two treadmill sessions per week for eight weeks; 2 (moderate intensity aerobics exercises), also consisting of two treadmill sessions per week for eight weeks; and 3 (control group), which consisted of twenty COPD patients who, in addition to their pharmaceutical treatments, received conventional chest physiotherapy twice per week for eight weeks.

Supporting the use of parametric statistical analyzes, the Shapiro-Wilk test verified that age, weight, height, BMI, and IL-17 were normally distributed among the three groups in the present investigation. After randomly assigning 60 COPD patients to one of three groups, researchers found no significant changes in the patients' age, weight, height, or body mass index (BMI) at baseline. The gender and mMRC grade distributions were also similar across all groups, with no significant variations. These results prove that the research groups were similar before the start of the trial, which is crucial for comparing the effects of different treatments.

Consistent with this finding, he et al. [21] conducted a prospective multicenter randomized controlled experiment to determine the ideal intensity of PR exercise training for patients with moderate-to-severe COPD. By comparing normally distributed baseline demographic characteristics using the Shapiro-Wilk test, they were able to ascertain whether the continuous data were normally distributed.

Moreover, Jiang et al. [22] shown associations between expression levels of interleukin (IL)-17, IL-10, and IL-35 in blood and clinical variables of patients with stable chronic obstructive pulmonary disease (COPD). Participants were 30 healthy individuals (15 men and 15 women) and 75 patients (47 men and 28 females) with stable chronic obstructive pulmonary disease (COPD). No statistically significant differences were found in terms of gender, age, or smoking status when comparing the observation group to the control group.

Also, Zou et al. [23] conducted a prospective study to evaluate the levels of serum IL-1 β and IL-17, and the connections between these two crucial mediators and clinical markers in COPD patients. Sixty individuals with stable chronic obstructive pulmonary disease (COPD), sixty individuals with COPD who were undergoing an acute exacerbation, and forty healthy controls had their blood samples taken. Their results indicated that there was no statistically significant difference in age or body mass index across the categories. The levels of IL-17 (in pg/mL) did vary significantly across treatments.

At the outset, there were no discernible variations in the IL-17 levels of the therapy groups compared to one another. In terms of IL-17 level reduction after the intervention, Group A (low-intensity treadmill aerobic exercise) performed the best, with a 23.40% improvement; Group B (moderate-to-low cardiovascular activity) came in second, with a 4.94% improvement; and Group C (conventional chest physiotherapy) showed a 4.44% improvement, which was not statistically significant. Comparisons made after treatment showed that Group A had a considerable advantage over Groups B and C, but Groups B and C did not vary significantly from one another. Results show that aerobic exercise on a low-intensity treadmill was the best strategy for lowering IL-17 levels in chronic obstructive pulmonary disease patients.

Several interrelated mechanisms, such as the suppression of Toll-like receptor (TLR) signaling, the reduction of inflammatory mediators derived from adipose tissue, the enhancement of regulatory T-cell activity, and the increased production of anti-inflammatory cytokines, are responsible for the pro-inflammatory to anti-inflammatory milieu shift that exercise induces [24, 25]. The main cellular source of IL-17, Th17 lymphocytes, are inhibited in their development and activation by these adaptations taken together. Aerobic exercise, when practiced regularly, reduces systemic production of pro-inflammatory cytokines by downregulating inflammatory pathways and maintaining immunological homeostasis.

Possible changes in the inflammatory load caused by exercise intensity might explain why the low-intensity exercise condition showed a higher decrease in IL-17 compared to the moderate-intensity program. Both types of exercise have their benefits, although the immediate cytokine response, reactive oxygen species generation, and temporary activation of inflammatory pathways are all amplified with increasing exercise intensity. Aerobic exercise at lower intensities stimulates the body enough to

induce anti-inflammatory adaptations without significantly increasing exercise-induced inflammatory stress, as has been shown to happen with higher exercise intensities [26–28].

This jibes with the findings of Zeng et al. [9], who looked back at the literature to see if aerobic exercise training helped stabilize-stage COPD patients. As a result of their findings, aerobic exercise training may be safely added to the therapeutic arsenal for stable COPD. These studies demonstrate that aerobic exercise training improves lung function, reduces inflammatory factor expression, and reduces clinical symptoms.

Beyond that, Ayari et al. [25] conducted a comprehensive analysis to determine which aspects of chronic exercise—namely, the type, frequency, duration, intensity, and volume—may be more influential in lowering pro-inflammatory cytokines and increasing anti-inflammatory cytokines in research involving both animals and humans suffering from mild cognitive impairment (MCI) or dementia. Running on a treadmill, doing strength exercises, and swimming all reduced pro-inflammatory cytokines while increasing anti-inflammatory ones, according to their research.

Additionally, Nambi et al. [29] conducted a randomized control experiment with the purpose of determining and evaluating the psychological and clinical benefits of low- and high-intensity aerobic exercise, strength training, and community-dwelling older men exhibiting signs of post-COVID-19 sarcopenia. Psychological (kinesiophobia) and clinical (muscle strength) metrics were better improved by low-intensity aerobic training sessions than by high-intensity aerobics.

Sankar et al. [30] shown that low-intensity aerobic exercise produces better results than standard therapy for COPD patients in terms of patient satisfaction and quality of life. In a prospective study of COPD patients, Zou et al. [23] confirmed our findings by measuring blood IL-1 β and IL-17 levels and investigating the connections between these two crucial mediators and clinical markers. Because elevated IL-17 levels are associated with inflammation and disease severity in COPD patients, their results provide support to the notion that therapeutic interventions, such as aerobic exercise, might reduce IL-17 levels, which may have biological implications.

CONCLUSION

Patients with chronic obstructive pulmonary disease (COPD) had a significant decrease in blood IL-17 levels after engaging in treadmill-based aerobic activity. The most effective kind of aerobic exercise was low-intensity exercise, which reduced IL-17 levels more than low-to-moderate aerobic exercise or traditional chest physiotherapy. As a result, individuals with chronic obstructive pulmonary disease (COPD) may benefit from non-pharmacological interventions such as low-intensity treadmill aerobic exercise to decrease systemic inflammation and improve inflammatory status.

LIMITATIONS

The single-center design of this investigation constituted a limitation. Although aerobic exercise may reduce inflammation in COPD patients over the long run, the short duration of the intervention may have obscured any benefits. Lacking evaluation of other inflammatory biomarkers, the research only focused on serum IL-17 levels.

RECOMMENDATIONS

To increase the reliability of the results, future research should use bigger samples and be carried out at more than one location. Researchers discovered that patients with COPD who participated in pulmonary rehabilitation programs that included low-intensity treadmill aerobic exercise had a decrease in blood IL-17 levels, which is a marker of inflammation. Healthcare providers should also recommend supervised aerobic exercise on a regular basis to patients as a supplement to pharmaceutical therapy for systemic inflammation management and better patient outcomes.

CONFLICT OF INTEREST

Regarding this review's subject matter, none of the writers have any conflicts of interest.

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