

# Comparison between the effects of aerobic and resistive training on immunoglobulins in obese women

Gihan S. Mohamed<sup>a,b</sup>, Mona M. Taha<sup>a</sup>

<sup>a</sup>Department of Cardiovascular/Respiratory Disorder and Geriatrics, Faculty of Physical Therapy, Cairo University, Giza, Egypt,

<sup>b</sup>Faculty of Applied Science, Umm Al-Qura University, Mecca, Saudi Arabia

Correspondence to Mona M. Taha, PhD,  
2-Street, 9, Helwan, Cairo 11722, Egypt  
Tel: 0201111351518

E-mail: monady2007@yahoo.com

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## Background and purpose

Obesity has adverse consequences on the immune system, causing immunosuppression, and thus increasing the incidence of infections and certain types of cancer in obese individuals. The aim of this study was to compare changes in selected immune system responses after moderate aerobic and resistive training in obese women.

## Methods

In total, 40 obese women (age: 35–45 years) were selected and divided into two equal groups: the aerobic training group (A), in which women received moderate-intensity aerobic training; and the resistive training group (B), in which women received moderate-intensity resistive training. Blood sampling was carried out for immunoglobulin (Ig) M and IgG in the pretest and after the 12th week of training.

## Results

There was a significant increase in IgM and IgG in response to aerobic training, whereas no significant changes occurred in the resistive training group. There were significant differences in IgM and IgG between the two groups after training in favor of the aerobic training group (A).

## Conclusion

Regular moderate aerobic training seems to improve immunity compared with resisted training in obese women.

## Keywords:

aerobic training, immunoglobulins, obesity, resistive training

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## Introduction

As one or more of the immune system components are inactive, immunodeficiency occurs. The capability of the immune system to respond to pathogens is reduced in both young and elderly, with immune responses starting to decrease at the age of around 50 years in obese women due to immunosenescence [1].

Autoimmune diseases as inflammatory diseases and cancer may occur due to the immune system disorders. As the immune system is not as active as its normal state, immunodeficiency occurs, leading to recurring and life-threatening infections [2].

Decreased immune function has been observed in several studies on obese humans [3–7] and animal models [8–11], which include decreased antigen/mitogen response stimulation, reduced cytokine production, decreased dendritic cell and macrophage function, and impaired natural killer cells. The impaired immune response in obese host leads to an increased susceptibility to infection with various pathogens as influenza, community-acquired tuberculosis, *Mycobacterium tuberculosis*, *Helicobacter pylori*, coxsackie virus, and encephalomyocarditis virus. This immune response may be attributed to

increased inflammation, impaired adipokine signaling, different metabolic changes, and regulation of epigenetic [12]. Immunoglobulin (Ig) G antibodies are present in all body fluids, and are the smallest and the most common antibody, representing 75–80% of all antibodies in the bodies. IgG antibodies have a big role in attacking viral and bacterial infections. They are the only type of antibody that can pass the placenta in a pregnancy and help protect the fetus [13].

IgM antibodies are the largest antibody and the first antibody type secreted in response to an infection. They are present in the blood and lymph fluid. Moreover, they lead to other immune system cells to destroy foreign materials. IgM antibodies represent about 5–10% of the antibodies [13]. The humoral arm of adaptive immunity is generally accompanied with circulating antibodies/Igs. During a primary antibody response, T-cells and antigen activate naive B-cells, which then differentiate into long-lived plasma

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cells (3–4 months in mice), short-lived plasma cells, or memory B-cells [14]. The short-lived plasma cells are produced first and initiate the primary antibody response and provide a rapid antigen-specific defense. This includes an early rise in antibodies of the antigen-specific IgM class (isotype), followed by isotype switching, and a rise in antigen-specific IgG, IgA, and/or IgE antibodies. This response requires up to 10 days to become fully activated. However, secondary antibody response is initiated by the long-lived plasma cells during subsequent re-exposure to antigen. These provide an immediate high-affinity antibody response to an antigen in the circulation without the need for slower activation of memory B-cells [15]. A variety of exercise types have been used in studies conducted on older people including calisthenics [16–19] and strength training [20–23]. However, most of the studies used cardiovascular exercise at a moderate intensity. Generally, the longer-term interventions have resulted in improvements of the immune function. For example, 12 months of exercise including both resistance and endurance components resulted in increased salivary IgA levels [24]. Acute, high-intensity exercise may suppress the immune system for 1–6 h of recovery. High-intensity training, according to the ‘j-loop’ hypothesis, may increase the risk for upper respiratory tract infection. For example, a single strenuous bout of exercise may suppress the innate immune system for up to 24 h after exercise. During this ‘open window’ there may be an increased risk for illness [25].

The binding of Ig to its target antigen forms antibody–antigen complexes; Ig and antibody–antigen complexes circulate in the body fluids. The effect of exercise on humoral immune function has been evaluated through measurements of the serum and mucosal Ig concentration *in vivo* and serum immunoglobulin (S-Ig) synthesis following in-vitro mitogen stimulation [26]. Because the immune function is critical to host survival, it is difficult to detect how much immune function must be gained or lost to reveal changes in host disease susceptibility. There are numerous examples of exercise that alter the measures of immunity. Whether those changes are enough to alter host defense, disease susceptibility, or severity remains debatable [27–30]. It seems that highly conditioned participants have a relatively better-preserved immunity, but it is unclear which training is more favorable. Therefore, this study was designed to investigate the effect of moderate-intensity aerobic versus moderate-intensity resistive training on Igs in obese women.

## Participants

A total of 40 women from the ages of 35 to 45 years were recruited from the outpatient clinic of the Faculty of Physical Therapy, Cairo University. Each woman was briefed about the potential risks of the clinical trial; a signed informed consent was obtained from the women; the study was approved by an Ethical Committee of the Faculty of Physical Therapy, Cairo University.

Participants were assigned into one of the two groups (A or B).

- (1) Group A received moderate-intensity aerobic training.
- (2) Group B received moderate-intensity resistive training.

All women were required to meet a strict set of inclusion criteria: class I obese (BMI from 30 to 34.9), regularly menstruating, not pregnant, not currently lactating, and not participating in any structured physical training program more than two times per week for the preceding 6 months. All women were required to undergo a comprehensive physical examination by a physician to identify the following exclusion criteria: confounding endocrine, orthopedic disorders, or other adverse pathologies that would endanger the volunteers or impact their response to training.

## Methods: evaluative procedures

All patients underwent the following sequence:

- (1) Weight and height scale to calculate the BMI ( $\text{kg}/\text{m}^2$ ).
- (2) Laboratory investigation:

IgG and IgM were evaluated as antibodies produced by the immune system to fight antigens before conduction of the exercise program and after the completion of the study (for 12 weeks). Total S-IgG and S-IgM was drawn by a laboratory specialist and determined using a Roch-Cobas device with the Alastat Microplate total IgG and IgM kits by Diagnostic Products (Los Angeles, California, USA) according to the manufacturer’s instructions and by comparisons with a known range of standard IgG and IgM concentrations.

## Training procedure

### *Aerobic training group (A)*

In total, 20 women participated in a supervised aerobic exercise program that consisted of walking/running on a treadmill three sessions per week for 12 weeks. The total

aerobic exercise time was 40 min as follows: warm-up for 5 min at 40% of the predicted maximal heart rate (MHR), 30 min aerobic exercise at 60–75% of the predicted MHR (calculated as  $MHR=210-\text{age}$  for women), and, finally, 5 min of cool-down period [31].

#### Resistive training group (B)

A total of 20 women participated in a supervised resistance exercise program performed three times per week for 12 weeks by using free weights and dumbbells at 80% one repetition maximum for group B participants [32]. The strength training consisted of 10 resistance exercises (five repetitions for five sets per session) – that is, knee extensors, knee flexors, hip flexors, hip abductors, hip extensors, elbow flexors, elbow extensors, shoulder flexors, abdominal muscles, and back muscles exercises. The total duration of the session was 40 min, including 5 min of warm-up and cool down in the form of simple stretching exercises.

#### Statistical analysis

Statistical analysis was carried out using the statistical package for the social science software, version 16 (SPSS, Inc., Chicago, IL). Data were expressed as mean±SD. The results were analyzed by using Student's *t*-test. A *P*-value of less than 0.05 was considered statistically significant.

## Results

A total of 40 obese women were recruited and divided between two groups: 20 in group A (the aerobic training group) and 20 in group B (the resistive training group). Participants in group A adhered to a 12-week moderate-intensity aerobic training program, whereas participants in group B adhered to a 12-week moderate-intensity resistive training program. Table 1 shows the baseline characteristics of the participants at the beginning of the study. There were no significant differences between the mean age, weight, and BMI values between the two groups.

The *t*-test was carried out to examine and compare IgM and IgG mean values within and between groups. Results showed that before training, there was a nonsignificant difference in the IgM and IgG mean values between the two groups. The mean IgM and IgG values significantly increased by 2.62 and 0.78%, respectively, in the aerobic training group (A) ( $P=0.001$ ) when comparing pretraining and post-training values, whereas there was no significant change in the mean IgM and IgG values as observed in the resistive training group (B) when comparing

pretraining and post-training values (Tables 2 and 3). There were significant differences between the mean levels of IgM and IgG between the two groups after training in favor of the aerobic training group (A) (Tables 2 and 3).

## Discussion

An interesting finding in this study was the significant increase in the total S-IgG and S-IgM ( $P=0.001$ ) after aerobic exercise, whereas no significant change was observed after resisted exercise. Generally in exercise immunology, exercise prevents immunosuppression because of an increase in Ig concentration [15].

This study showed improvement in immunity after 12 weeks of aerobic training in obese women. It seems that research is conflicting; our results are in line with others studies such as that by Poortmans [33], who found a significant 12% increase in S-IgG immediately after nearly 21 min of the progressive cycle ergometer test up to fatigue.

Similar to the present results, Poortmans and Haralambie [34] reported that IgG had an improvement (about 7%) immediately after racing. Recently, Petibois *et al.* [35] recorded Ig changes over 12 months of training, and stated that IgG1, IgG2, and IgG4 levels rose because of rowing exercise training. But they found that IgG3 became low at the 18th training week and continued to be low from this point onward.

Nieman and Nehlsen-Cannarella [36] reported that during the 15 weeks of exercise of mild intensity, S-Ig levels improved by 20%. Thus, infection would be prevented. Hanns *et al.* [37], in their study, reported that the IgG, IgA, and IgM serum levels were elevated after the marathon and returned to normal in the recovery period; elevated changes in only IgA were due to plasma volume changes.

Andrew *et al.* [38] reported that a run on a treadmill (gradient -13.5%) at a speed eliciting 75% of their  $VO_2$  peak for an hour in 15 males caused the total IgG, IgG1, IgG3, and IgA levels to be significantly increased and IgG2 level significantly decreased.

Nieman *et al.* [39] demonstrated that an increase in IgM by 7.2% after running 1 h at sub-3 h marathon pace would take 21 h to return to the baseline levels. In their study, Petibois *et al.* [35] observed IgM concentration increase over 12 months in elite rowers after training.

**Table 1 Baseline characteristics of the study participants (mean±SD) in two groups**

Characteristics	Group A	Group B	t-value	P-value
Age (years)	37.75±2.291	38.60±2.062	-1.233	0.255**
Weight (kg)	89.59±3.820	90.40±4.297	-0.633	0.530**
BMI (kg/m <sup>2</sup> )	32.12±1.25	32.81±0.972	-1.931	0.061**

Level of significance at  $P < 0.05$ . \*Significant. \*\*Not significant.

**Table 2 Within and between groups comparison of IgM (mg/dl) (t and P values)**

	Group A (mean±SD)	Group B (mean±SD)	t-value	P-value
Pretraining	174.62±16.349	166.31±15.490	1.651	0.107**
Post-training	179.21±15.632	166.16±15.478	2.653	0.012*
t-value	-25.063	0.773		
P-value	0.001*	0.449**		

Ig, immunoglobulin. Level of significance at  $P < 0.05$ . \*Significant. \*\*Not significant.

**Table 3 Within and between groups comparison of IgG (mg/dl) (t and P values)**

	Group A (mean±SD)	Group B (mean±SD)	t-value	P-value
Pretraining	1436.45±8.383	1441.42±8.600	-1.852	0.072**
Post-training	1447.71±7.922	1441.76±8.962	2.224	0.032*
t-value	-11.229	-0.849		
P-value	0.001*	0.407**		

Ig, immunoglobulin. Level of significance at  $P < 0.05$ . \*Significant. \*\*Not significant.

In contrast, McKune *et al.* [15] found that IgM was significantly reduced (23%) 24 h after the marathon, which may be attributed to acute intense bouts of long-duration marathon (>2 h). Few studies have reported the level of IgM after ultra-endurance training. Mashiko *et al.* [40], in their study, reported a 15% decrease in IgM and a significant decrease of 28% in IgG after a rugby training camp for 20 days, training 120 min a day, and 6 days per week. This is possible because they performed more physically strenuous exercise (running) during the camp. In contrast, Imanipour *et al.* [41] in their 14-week study on 20 active men found significant decreases in the IgA and IgM serum concentrations; in addition, they reported a slight but insignificant increase in the IgG concentration between pretest and post-test. The author stated that the limitation of his study was the small sample size.

On the other hand, Verde *et al.* [42] reported that the IgG and IgM levels in 10 elite male runners that participated in increased training schedules by an average of 38% for 3 weeks decreased significantly. Moreover, Nieman and Nehlsen-Cannarella [36] observed that IgG reduced after running at the marathon pace for 3 h during recovery, reaching its least point at 1.5 h (27.6%) and increasing to baseline level after 21 h of exercise.

Mohebbi *et al.* [43] showed that high-intensity resistance training reduced S-IgG concentration and low-intensity training increased S-IgG concentration. One explanation that has been suggested for the rises in special antibodies after exercise is the flushing of nonsystemic Igs out of the secondary lymphatic sites and/or enter the circulation because of increased lymphatic flow [36].

The significant results of IgG obtained after a study of 3 months of application of moderate-intensity treadmill exercise program suggested that these changes may be because moderate-endurance training results in increasing capacity to generate interferon  $\gamma$  [44]. S-Ig concentration appears to remain either the same or slightly higher, in response to either brief or prolonged exercise. Mitogen-stimulated IgM concentration appears to increase in response to exercise independently of changes in T or B-cell number, although there are opposing findings concerning IgA and IgG [26].

Mohebbi *et al.* [43] showed that blood lactate levels after high-intensity resistance exercise see an increase compared with lower resistance, and this increase is due to the released lactate from the muscle into the blood. Lactate influence on the immune system cells and some immune reactions and its ability to be

mobilized during resistance training are associated with the anaerobic exercise intensity as reflected by lactate production. Staron *et al.* [45] found that 8–12 weeks of resistance training programs had minimal effects on resting inflammatory, innate, or acquired immune parameters, as assessed through an analysis of peripheral blood. Karacabey *et al.* [46] explained that the changes in the production of Igs have been related to the stimulation of the central nervous system and the increase in catecholamine. Therefore, it can be concluded that the differences among studies can be attributed to the difference in training protocol such as type, duration, intensity, and program of exercise or difference in subject adaptation to exercise or study sample as most of the previous studies were conducted on athletes.

The limitations of this study are the small number of participants and the lack of a control group. Future studies on a larger sample are required to confirm our results, and, also, further studies with different training protocols need to be conducted on sedentary individuals to show the favorable impact upon immunity.

## Conclusion

Despite these limitations, the study suggests that aerobic training is superior to resisted training in improving humoral immunity in obese females.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

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