

## Aerobic vs anaerobic exercise training effects on the cardiovascular system

Harsh Patel, Hassan Alkhawam, Raef Madanieh, Niel Shah, Constantine E Kosmas, Timothy J Vittorio

Harsh Patel, Department of Internal Medicine, State University of New York at Brooklyn - School of Medicine, Brooklyn, NY 11203, United States

Hassan Alkhawam, Department of Medicine, Icahn School of Medicine at Mount Sinai (Elmhurst), Elmhurst, NY 11373, United States

Raef Madanieh, Niel Shah, Timothy J Vittorio, St. Francis Hospital - the Heart Center®, Center for Advanced Cardiac Therapeutics, Roslyn, NY 11576, United States

Constantine E Kosmas, Icahn School of Medicine, Mount Sinai Hospital Center, New York, NY 10029, United States

**Author contributions:** Patel H and Vittorio TJ conceived with an idea about this work and drafted the outline and main manuscript; Alkhawam H, Madanieh R, Shah N and Kosmas CE were responsible for critically reviewing the manuscript for intellectual content, reviewing of the literatures and editing a draft of the main manuscript.

**Conflict-of-interest statement:** None.

**Open-Access:** This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>

**Manuscript source:** Invited manuscript

**Correspondence to:** Timothy J Vittorio, MS, MD, St. Francis Hospital - the Heart Center®, Center for Advanced Cardiac Therapeutics, 100 Port Washington Blvd., Roslyn, NY 11576, United States. [t\\_vittorio@hotmail.com](mailto:t_vittorio@hotmail.com)  
Telephone: +1-516-6292166  
Fax: +1-516-6292094

Received: September 13, 2016

Peer-review started: September 14, 2016

First decision: October 21, 2016

Revised: November 22, 2016

Accepted: December 7, 2016

Article in press: December 9, 2016

Published online: February 26, 2017

### Abstract

Physical exercise is one of the most effective methods to help prevent cardiovascular (CV) disease and to promote CV health. Aerobic and anaerobic exercises are two types of exercise that differ based on the intensity, interval and types of muscle fibers incorporated. In this article, we aim to further elaborate on these two categories of physical exercise and to help decipher which provides the most effective means of promoting CV health.

**Key words:** Cardiovascular; Exercise; Training; Aerobic; Anaerobic

© **The Author(s) 2017.** Published by Baishideng Publishing Group Inc. All rights reserved.

**Core tip:** As the association between physical inactivity and the increased risk of cardiovascular morbidity solidified, further data and studies supported the advantages of exercise on physical well-being. Anaerobic and aerobic exercise have a favorable effect on lipid metabolism, anaerobic exercises have been shown to have a positive influence on the lipid profile.

Patel H, Alkhawam H, Madanieh R, Shah N, Kosmas CE, Vittorio TJ. Aerobic vs anaerobic exercise training effects on the cardiovascular system. *World J Cardiol* 2017; 9(2): 134-138 Available from: URL: <http://www.wjgnet.com/1949-8462/full/v9/i2/134.htm> DOI: <http://dx.doi.org/10.4330/wjc.v9.i2.134>

### INTRODUCTION

More than 250000 yearly deaths in the United States are

attributed to cardiovascular (CV) disease resulting from a lack of physical activity. On the other hand, physical inactivity is estimated to cause 30% of ischemic heart disease<sup>[1]</sup>. The association between physical inactivity and CV disease gained a foothold in the medical community in 1996, when the American Heart Association (AHA) published information advocating the benefit of physical exercise in regards to improvements in hemodynamic, hormonal, metabolic, neurological and respiratory function<sup>[2]</sup>. As the association between physical inactivity and the increased risk of CV morbidity solidified, further data and studies supported the advantages of exercise on physical well-being. The 2010 recommendations by the World Health Organization (WHO) provided activity recommendations based on three different age groups: Ages 5-17, 18-64, and > 64 years of age. In the age group of 5-17 years, individuals should accrue at least 60 min of moderate activity daily. Those in the group of 18-64 years should perform at least 150 min of moderate activity or at least 75 min of vigorous activity throughout the week. Finally, individuals above the age of 65 years are recommended similar length and intensity exercise programs as the prior group, but with a focus on activities to help enhance balance and to prevent falls<sup>[3]</sup>.

The inherent advantages of physical exercise stem from an increase in the cardiac output and an enhancement of the innate ability of muscles to extract and to utilize oxygen from the blood. This benefit is further compounded by the benefit physical exercise has on high-density lipoprotein cholesterol (HDL-C)<sup>[4]</sup>, adipose tissue distribution<sup>[5]</sup>, increased insulin sensitivity<sup>[6]</sup>, improved cognitive function<sup>[7]</sup>, enhanced response to psychosocial stressors<sup>[8]</sup>, as well as determent of depression<sup>[9]</sup>. With the benefit of physical exercise well established, the question remains which type of exercise provides the most effective and efficient means to help deter CV disease.

A recent meta-analysis published showed a decrease in the risk of all CV outcomes and diabetes mellitus incidence with increasing levels of physical activities<sup>[10]</sup>. Another meta-analysis suggested that high level of leisure time physical activity had a beneficial effect on CV health by reducing the overall risk of incident CHD and stroke among men and women by 20% to 30%, while moderate level of occupational physical activity might reduce 10% to 20% risk of CVD<sup>[11]</sup>.

Furthermore, cardiac rehabilitation, which is physical exercise based, is a promising field which showed a favorable outcome among patients with heart failure and post-CVD events<sup>[12,13]</sup>.

## AEROBIC EXERCISE

The American College of Sports Medicine (ACSM) defines aerobic exercise as any activity that uses large muscle groups, can be maintained continuously and is rhythmic in nature<sup>[10]</sup>. As the name implies, muscle groups activated by this type of exercise rely on aerobic metabolism to extract energy in the form of adenosine triphosphate (ATP) from amino acids, carbohydrates and fatty acids.

Examples of aerobic exercise include cycling, dancing, hiking, jogging/long distance running, swimming and walking. These activities can best be accessed *via* the aerobic capacity, which is defined by the ACSM as the product of the capacity of the cardiorespiratory system to supply oxygen and the capacity of the skeletal muscles to utilize oxygen<sup>[14]</sup>. The criterion measure for aerobic capacity is the peak oxygen consumption (VO<sub>2</sub>), which can be measured either through graded exercise ergometry or treadmill protocols with an oxygen consumption analyzer or *via* mathematical formulas. The value of peak VO<sub>2</sub> can be appreciated by a study performed by Vaitkevicius *et al*<sup>[15]</sup>, in which the VO<sub>2max</sub> was calculated along with other dimensions, to conclude that higher physical conditioning status was directly correlated with reduced arterial stiffness.

Various studies have been published that prove the advantages of aerobic exercise in reversing and preventing CV disease. In 2002, Wisløff *et al*<sup>[16]</sup> were the first to show the benefit of aerobic training in the myocardium after an ischemic event. Their study was performed on adult female Sprague-Dawley rats, which were placed into groups categorized based on induced myocardial infarctions (MI) with and without exercise and controls with and without exercise. Their results showed a 15% reduction in the left ventricle (LV) hypertrophy post-infarction, as well as 12% and 20% decreases in myocyte length and width, respectively, with aerobic exercise. Furthermore, a 60% improvement was noted in myocardial contractility in subjects with a MI who were assigned to the training group, suggesting enhanced myocardial Ca<sup>2+</sup> sensitivity. They were able to conclude the beneficial effects of aerobic training on cardiac remodeling and myocardial contractility<sup>[16]</sup>.

The effect of aerobic exercise were confirmed in human subjects when Wisløff *et al*<sup>[17]</sup> published another study five years later, which incorporated human subjects with post-MI heart failure. Subjects were enrolled in aerobic interval training (AIT), moderate continuous training (MCT) or a control group. The AIT group showed a 46% increase in peak VO<sub>2</sub>, which correlated with a 60% increase in the maximal rate of Ca<sup>2+</sup> reuptake in the sarcoplasmic reticulum in the skeletal muscles. Additionally, cardiac remodeling was evident in humans, much like the rat subjects in the previous study, as LV diameters declined and LV volumes increased in both the diastolic and systolic phases. Moreover, systolic function was noted to increase by 35% in the AIT group<sup>[17]</sup>, thereby further strengthening the advantages of aerobic exercise.

Furthermore, aerobic exercise has been shown to have a positive impact on other dimensions of CV health. Several studies have shown that aerobic exercise improves the lipid profile, particularly increasing the HDL-C<sup>[18]</sup>. In an Australian study, aerobic exercise led to a small but statistically significant reduction in total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG) ranging in a span of 0.08 mmol/L to 0.10 mmol/L. They also showed an increase in HDL-C with their aerobic exercise program of about 0.05 mmol/

L<sup>[19]</sup>. Similar results have been documented in children and adolescents, as well<sup>[20]</sup>. In a meta-analysis conducted by Kelley *et al*<sup>[21]</sup>, it was concluded that aerobic exercises contributed to a statistically significant 9% increase in HDL-C and an 11% decline in TG, but no statistically significant changes in TC and LDL-C.

A positive correlation between biochemical signal markers, such as endothelin-I (ET-1) and aerobic exercise was recently speculated by several studies. Vascular endothelial cells produce ET-1, which functions as a vasoconstrictor<sup>[22]</sup> and promoter of atherosclerosis<sup>[23]</sup>. Maeda *et al*<sup>[24]</sup> were able to demonstrate a statistically significant positive linear correlation of increasing age with rising levels of ET-1. They were also able to exhibit a visible reduction in ET-1 levels after a 3 mo aerobic exercise regimen<sup>[24]</sup>.

While aerobic exercise appears to have some beneficial effects, its contribution is limited on frequency and quantity. A very recent publication by a Danish group was able to represent what they called a "U shaped association" between aerobic exercise and mortality. Their research quantified 1 to 2.4 h of exercise over 2 to 3 times per week as the optimal quantity and frequency standard of aerobic exercise to promote improved health. Interestingly, they quantified any amount above that standard as being indifferent to the mortality risk, as that of sedentary individuals<sup>[25]</sup>.

## ANAEROBIC EXERCISE

Anaerobic exercise has been defined by the ACSM as intense physical activity of very short duration, fueled by the energy sources within the contracting muscles and independent of the use of inhaled oxygen as an energy source<sup>[14]</sup>. Without the use of oxygen, our cells revert to the formation of ATP *via* glycolysis and fermentation. This process produces significantly less ATP than its aerobic counterpart and leads to the build-up of lactic acid. Exercises typically thought of as anaerobic consist of fast twitch muscles and include sprinting, high-intensity interval training (HIIT), power-lifting, *etc.* Sustained anaerobic metabolism, in other words, anaerobic exercise, causes a sustained increase in lactate and metabolic acidosis and this transition point is referred to anaerobic threshold (AT)<sup>[26]</sup>. AT can be directly measured *via* frequent blood samples measuring the blood lactate level during a graded-exercise regimen. Once the blood lactate values are plotted, the point at which the curve makes a sudden sharp incline represents the AT. Other methods include portal lactate analyzers and mathematical formulas involving heart rate (HR).

Similar to aerobic exercise, anaerobic exercise may exert a potentially beneficial influence on the CV system. In a Turkish study completed by Akseki Temür *et al*<sup>[27]</sup>, the effects of anaerobic exercise were evaluated with a member of the natriuretic peptide family, known as C-type natriuretic peptide (CNP). CNP is synthesized by the endothelium and offers a protective effect through its effects on the vascular tone of blood vessels,

as well as exerting antifibrotic and antiproliferative properties. It produces a hyperpolarization effect on the smooth muscle layer of blood vessels, which causes vasodilatation<sup>[28]</sup>. CNP has also been reported to exert its nonproliferative effects on cardiac fibroblasts to help prevent cardiac aging through LV fibrosis *via* the cyclic guanosine monophosphate (cGMP) pathway<sup>[29]</sup>. In this study, twelve healthy young male subjects were divided into two groups based on their previous history of exercise. Once categorized into groups, the subjects were asked to participate in a thirty second high intensity exercise program, which encompassed the anaerobic exercise factor. Blood samples were obtained from the subjects before exercise and then one minute, five minutes and thirty minutes after exercise and were tested for the levels of aminoterminal proCNP (NT-proCNP), a biologically inactive peptide of CNP. The results showed a statistically significant increase of NT-proCNP level in the five minute mark post-exercise in the physically active group after anaerobic exercise.

Similar to aerobic exercise and their favorable effect on lipid metabolism, anaerobic exercises have been shown to have a positive influence on the lipid profile. A small European study composed of 16 obese subjects was able to show the increased benefits of an aerobic workout followed by anaerobic training, as compared to aerobic training alone. Subjects who underwent core training with both aerobic and anaerobic exercises showed a larger reduction in non-esterified fatty acids. The same group was also found to have the greatest reduction in their body mass index (BMI)<sup>[30]</sup>.

There are speculations about disadvantages of such an exercise program. One such shortcoming was brought to light by an Iranian study published by Manshouri *et al*<sup>[31]</sup>, which concluded that anaerobic training led to a significant reduction in human growth hormone (HGH). It has long been theorized that long-standing HGH deficiencies can attribute to CV morbidity and mortality through the development of premature atherosclerosis. HGH deficiency has been shown to result in higher BMI and TG, lower concentrations of HDL-C, as well as the development of hypertension (HTN)<sup>[32]</sup>. Furthermore, cardiac structure is affected in HGH deficient subjects, as manifested by reduced LV posterior wall thickness, smaller LV mass index and compromised LV ejection fraction (LVEF)<sup>[33]</sup>. The exact mechanism of action for such changes remains to be determined.

## CONCLUSION

With the high incidence of CV disease worldwide, it is an irrefutable notion that exercise helps deter CV morbidity and mortality. Both aerobic and anaerobic exercises have unique and collective positive correlations towards improved CV health. Despite all the research, further studies are still warranted to delve further into the impact that both aerobic and anaerobic exercise may have on human physiology to unequivocally determine if there is superiority of one type of exercise over

another.

## REFERENCES

- 1 **Myers J.** Cardiology patient pages. Exercise and cardiovascular health. *Circulation* 2003; **107**: e2-e5 [PMID: 12515760 DOI: 10.1161/01.CIR.0000048890.59383.8D]
- 2 **Fletcher GF,** Balady G, Blair SN, Blumenthal J, Caspersen C, Chaitman B, Epstein S, Sivarajan Froelicher ES, Froelicher VF, Pina IL, Pollock ML. Statement on exercise: benefits and recommendations for physical activity programs for all Americans. A statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. *Circulation* 1996; **94**: 857-862 [PMID: 8772712 DOI: 10.1161/01.CIR.94.4.857]
- 3 **WHO Guidelines Approved by the Guidelines Review Committee.** Global Recommendations on Physical Activity for Health. Geneva: World Health Organization, 2010 [PMID: 26180873]
- 4 **Williams PT.** High-density lipoprotein cholesterol and other risk factors for coronary heart disease in female runners. *N Engl J Med* 1996; **334**: 1298-1303 [PMID: 8609947 DOI: 10.1056/NEJM199605163342004]
- 5 **Schwartz RS,** Shuman WP, Larson V, Cain KC, Fellingham GW, Beard JC, Kahn SE, Stratton JR, Cerqueira MD, Abrass IB. The effect of intensive endurance exercise training on body fat distribution in young and older men. *Metabolism* 1991; **40**: 545-551 [PMID: 2023542 DOI: 10.1016/0026-0495(91)90239-S]
- 6 **Rosenthal M,** Haskell WL, Solomon R, Widstrom A, Reaven GM. Demonstration of a relationship between level of physical training and insulin-stimulated glucose utilization in normal humans. *Diabetes* 1983; **32**: 408-411 [PMID: 6341123 DOI: 10.2337/diab.32.5.408]
- 7 **Spirduso WW.** Physical fitness, aging, and psychomotor speed: a review. *J Gerontol* 1980; **35**: 850-865 [PMID: 7002994 DOI: 10.1093/geronj/35.6.850]
- 8 **Crews DJ,** Landers DM. A meta-analytic review of aerobic fitness and reactivity to psychosocial stressors. *Med Sci Sports Exerc* 1987; **19**: S114-S120 [PMID: 3316910 DOI: 10.1249/00005768-198710001-00004]
- 9 **Lobstein DD,** Mosbacher BJ, Ismail AH. Depression as a powerful discriminator between physically active and sedentary middle-aged men. *J Psychosom Res* 1983; **27**: 69-76 [PMID: 6834301 DOI: 10.1016/0022-3999(83)90111-3]
- 10 **Wahid A,** Manek N, Nichols M, Kelly P, Foster C, Webster P, Kaur A, Friedemann Smith C, Wilkins E, Rayner M, Roberts N, Scarborough P. Quantifying the Association Between Physical Activity and Cardiovascular Disease and Diabetes: A Systematic Review and Meta-Analysis. *J Am Heart Assoc* 2016; **5**: pii: e002495 [PMID: 27628572 DOI: 10.1161/JAHA.115.002495]
- 11 **Li J,** Siegrist J. Physical activity and risk of cardiovascular disease—a meta-analysis of prospective cohort studies. *Int J Environ Res Public Health* 2012; **9**: 391-407 [PMID: 22470299 DOI: 10.3390/ijerph9020391]
- 12 **Mampuya WM.** Cardiac rehabilitation past, present and future: an overview. *Cardiovasc Diagn Ther* 2012; **2**: 38-49 [PMID: 24282695 DOI: 10.3978/j.issn.2223-3652.2012.01.02]
- 13 **Kaminsky LA,** Jones J, Riggin K, Strath SJ. A pedometer-based physical activity intervention for patients entering a maintenance cardiac rehabilitation program: a pilot study. *Cardiovasc Diagn Ther* 2013; **3**: 73-79 [PMID: 24282749 DOI: 10.3978/j.issn.2223-3652.2013.03.03]
- 14 **American College of Sports Medicine.** ACSM's guidelines for exercise testing and prescription. USA: Lippincott Williams & Wilkins, 2013
- 15 **Vaitkevicius PV,** Fleg JL, Engel JH, O'Connor FC, Wright JG, Lakatta LE, Yin FC, Lakatta EG. Effects of age and aerobic capacity on arterial stiffness in healthy adults. *Circulation* 1993; **88**: 1456-1462 [PMID: 8403292 DOI: 10.1161/01.CIR.88.4.1456]
- 16 **Wisloff U,** Loennechen JP, Currie S, Smith GL, Ellingsen Ø. Aerobic exercise reduces cardiomyocyte hypertrophy and increases contractility, Ca<sup>2+</sup> sensitivity and SERCA-2 in rat after myocardial infarction. *Cardiovasc Res* 2002; **54**: 162-174 [PMID: 12062372 DOI: 10.1016/S0008-6363(01)00565-X]
- 17 **Wisloff U,** Støylen A, Loennechen JP, Bruvold M, Rognum Ø, Haram PM, Tjønnå AE, Helgerud J, Slørdahl SA, Lee SJ, Videm V, Bye A, Smith GL, Najjar SM, Ellingsen Ø, Skjaerpe T. Superior cardiovascular effect of aerobic interval training versus moderate continuous training in heart failure patients: a randomized study. *Circulation* 2007; **115**: 3086-3094 [PMID: 17548726 DOI: 10.1161/CIRCULATIONAHA.106.675041]
- 18 **Blumenthal JA,** Emery CF, Madden DJ, Coleman RE, Riddle MW, Schniebolck S, Cobb FR, Sullivan MJ, Higginbotham MB. Effects of exercise training on cardiorespiratory function in men and women older than 60 years of age. *Am J Cardiol* 1991; **67**: 633-639 [PMID: 2000798 DOI: 10.1161/JAHA.115.002014]
- 19 **Halbert JA,** Silagy CA, Finucane P, Withers RT, Hamdorf PA. Exercise training and blood lipids in hyperlipidemic and normolipidemic adults: a meta-analysis of randomized, controlled trials. *Eur J Clin Nutr* 1999; **53**: 514-522 [PMID: 10452405 DOI: 10.1177/0003319708324927]
- 20 **Tolfrey K,** Jones AM, Campbell IG. The effect of aerobic exercise training on the lipid-lipoprotein profile of children and adolescents. *Sports Med* 2000; **29**: 99-112 [PMID: 10701713 DOI: 10.2165/00007256-200029020-00003]
- 21 **Kelley GA,** Kelley KS, Franklin B. Aerobic exercise and lipids and lipoproteins in patients with cardiovascular disease: a meta-analysis of randomized controlled trials. *J Cardiopulm Rehabil* 2006; **26**: 131-139 [PMID: 16738448]
- 22 **Haynes WG,** Ferro CJ, O'Kane KP, Somerville D, Lomax CC, Webb DJ. Systemic endothelin receptor blockade decreases peripheral vascular resistance and blood pressure in humans. *Circulation* 1996; **93**: 1860-1870 [PMID: 8635265 DOI: 10.1161/01.CIR.93.10.1860]
- 23 **Lerman A,** Edwards BS, Hallett JW, Heublein DM, Sandberg SM, Burnett JC. Circulating and tissue endothelin immunoreactivity in advanced atherosclerosis. *N Engl J Med* 1991; **325**: 997-1001 [PMID: 1886637 DOI: 10.1056/NEJM199110033251404]
- 24 **Maeda S,** Tanabe T, Miyauchi T, Otsuki T, Sugawara J, Iemitsu M, Kuno S, Ajisaka R, Yamaguchi I, Matsuda M. Aerobic exercise training reduces plasma endothelin-1 concentration in older women. *J Appl Physiol* (1985) 2003; **95**: 336-341 [PMID: 12611765 DOI: 10.1152/jappphysiol.01016.2002]
- 25 **Schnohr P,** O'Keefe JH, Marott JL, Lange P, Jensen GB. Dose of jogging and long-term mortality: the Copenhagen City Heart Study. *J Am Coll Cardiol* 2015; **65**: 411-419 [PMID: 25660917 DOI: 10.1016/j.jacc.2014.11.023]
- 26 **Wasserman K.** The anaerobic threshold: definition, physiological significance and identification. *Adv Cardiol* 1986; **35**: 1-23 [PMID: 3551513 DOI: 10.1159/000413434]
- 27 **Akseki Temür H,** Vardar SA, Demir M, Palabıyık O, Karaca A, Guksu Z, Ortanca A, Süt N. The alteration of NTproCNP plasma levels following anaerobic exercise in physically active young men. *Anatol J Cardiol* 2015; **15**: 97-102 [PMID: 25252292 DOI: 10.5152/akd.2014.5204]
- 28 **Chauhan SD,** Nilsson H, Ahluwalia A, Hobbs AJ. Release of C-type natriuretic peptide accounts for the biological activity of endothelin-derived hyperpolarizing factor. *Proc Natl Acad Sci USA* 2003; **100**: 1426-1431 [PMID: 12552127 DOI: 10.1073/pnas.0336365100]
- 29 **Sangaralingham SJ,** Huntley BK, Martin FL, McKie PM, Bellavia D, Ichiki T, Harders GE, Chen HH, Burnett JC. The aging heart, myocardial fibrosis, and its relationship to circulating C-type natriuretic Peptide. *Hypertension* 2011; **57**: 201-207 [PMID: 21189408 DOI: 10.1161/HYPERTENSIONAHA.110.160796]
- 30 **Salvadori A,** Fanari P, Marzullo P, Codeca F, Tovaglieri I, Comacchia M, Brunani A, Luzi L, Longhini E. Short bouts of anaerobic exercise increase non-esterified fatty acids release in obesity. *Eur J Nutr* 2014; **53**: 243-249 [PMID: 23619826 DOI: 10.1007/s00394-013-0522-x]
- 31 **Manshoury M,** Ghanbari-Niaki A, Kraemer RR, Shemshaki A. Time course alterations of plasma obestatin and growth hormone levels in response to short-term anaerobic exercise training in college women. *Appl Physiol Nutr Metab* 2008; **33**: 1246-1249 [PMID: 19088784]

DOI: 10.1139/H08-098]

- 32 **Rosén T**, Edén S, Larson G, Wilhelmsen L, Bengtsson BA. Cardiovascular risk factors in adult patients with growth hormone deficiency. *Acta Endocrinol (Copenh)* 1993; **129**: 195-200 [PMID: 8212983 DOI: 10.1530/acta.0.1290195]

- 33 **Merola B**, Cittadini A, Colao A, Longobardi S, Fazio S, Sabatini D, Saccá L, Lombardi G. Cardiac structural and functional abnormalities in adult patients with growth hormone deficiency. *J Clin Endocrinol Metab* 1993; **77**: 1658-1661 [PMID: 8263155 DOI: 10.1210/jcem.77.6.8263155]

**P- Reviewer:** Cosmi E, Omboni S, Schoenhagen P **S- Editor:** Ji FF  
**L- Editor:** A **E- Editor:** Lu YJ





Published by **Baishideng Publishing Group Inc**

8226 Regency Drive, Pleasanton, CA 94588, USA

Telephone: +1-925-223-8242

Fax: +1-925-223-8243

E-mail: [bpgoffice@wjgnet.com](mailto:bpgoffice@wjgnet.com)

Help Desk: <http://www.wjgnet.com/esps/helpdesk.aspx>

<http://www.wjgnet.com>

