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Cellgevity Supplementation Improves Left Ventricular Pump Performance and Mechanical Energy Conversion Efficiency in Healthy Humans

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ABSTRACT

Background: Chemical energy in glucose is metabolized to electricaland biomechanical works done in the heart. Cellgevity(CGV) is anantioxidant supplement that improves energy recovery in myocardial tissues.

Aim: The study evaluated the changes in left ventricular pump performance and mechanical energyconversion efficiency by applying the biomechanical concepts of changes in pressure, volume, time, and oxygen consumption in healthy subjects before and after eight- (8) week CGV supplementation.

Methods: Arterial blood pressure, electrocardiogram and echocardiogram parameters were measured in twenty-four (24) age- and sex-matched subjects before and after 8-week oral (2320 mg/d) CGV supplementation. Left ventricular pump performance and mechanical energy conversion parameters were then determined before and after CGV supplementation.

Results: After 8-week CGV supplementation in the healthy subjects: There was increase in left ventricular compliance (LVC) and its consequent left ventricular stroke volume (LVSV) which translated to increases inleft ventricular ejection fraction (LVEF), cardiac output (CO), cardiac index (CI) and stroke volume-energy ratio (SVER) ($p < 0.05$ or $p < 0.001$). The reduction in rate-pressure product (RPP) translated to reduction in myocardial oxygen consumption (MVO_2) ($p < 0.01$, $p < 0.001$). The decrease in left ventricular work done in diastole (LVW_D) and the increase in left ventricular total work done (LVW_T) produced the increase in left ventricular mechanical work advantage (LVMWA) ($p < 0.001$). The increase in LVW_T translated to the increase left ventricular power (LVP) and produced an increase in left ventricular mechanical efficiency (LVMℰ) in spite of the increase in left ventricular total energy (LVTE) consumed ($p < 0.001$). The decrease in MVO_2 produced the decrease in left ventricular mechanical efficiency index (LVMℰi) ($p < 0.01$, $p < 0.05$). The decreases in MVO_2 and QRS duration with the increases in R-wave voltage, electrical diastole (ED) and RR interval were in corroboration with increases in CO, LVMWA and LVMℰ in the subjects after CGV supplementation ($p<0.05$, $p < 0.01$ or $p < 0.001$).

Conclusion: Study shows that CO, LVMWA, LVMℰ, and LVMℰi improved after CGV supplementation in healthy subjects. Thus, CGV supplementation may enhance left ventricular pump performance and mechanical energy conversion efficiency in healthy humans.

Keyword: left ventricle, pump performance, energy conversion efficiency, oxygen consumption, cellgevity

INTRODUCTION

The heart is a double-pump organ1. The left and right ventricles are connected in series by two independent systemic and pulmonary circulations in series arrangement[1]. The left ventricle pumps blood through the systemic circulation while the right ventricle pumps blood through the pulmonary circulation 1. There are right and left atria above which drain blood into the right and left ventricles below respectively[1]. The atria and the ventricles contract rhythmically and alternately. The period of contraction is termed systole and that of relaxation is diastole[2]. The atria pump blood into the ventricles via atrioventricular valves during atria systole while the ventricles are relaxing in late ventricular diastole[2]. Alternately, the ventricles pump blood into the pulmonary and systemic circulations during ventricular systole while the atria are relaxing[3]. Atria receive and store blood during atria diastole or ventricular systole while blood flows from the atria into the ventricles during ventricle diastoleor atria systole[3]. The right ventricle propels blood through the pulmonary trunk into the pulmonary circulation in the lungs, while the left ventricle propels blood through the aorta into the systemic circulation in the body tissues[2]. Before the biomechanical contraction phase (systole) of the cardiac cycle, there is generation and transmission of action potentials across the myocardial cell membrane[3]. These cardiac action potentials are responsible for the active contractile movement of the myofibril myosin filaments in between actin filaments

which uses huge energy in form of adenosine triphosphate(ATP)[4], producing ventricular contraction and consequent pumping of blood. The action potentials are generated as a result of passive influxes of Na^+, Ca^{2+}, K^+ across myocardial cell membrane. However, the active pumping of these ions across cell and sarcotubular membranes also requires metabolic energy in form of ATP[5]. The ATP molecules are produced via oxidative phosphorylation, electron transport chain and oxolysis of glucose, fatty and amino acids 5. It is known that it is impossible for the heart as biomechanical pump to convert all the chemical energy to electrical and mechanical energies during contraction at systole[1]. In which case, the energy efficiency of the heart cannot be 100%[6]. The heart is thermodynamically active during biomechanical relaxation (diastole) and thermodynamically passive during biomechanical contraction (systole), because during systole, ion movements are metabolically passive to generate cardiac action potential or electrical energy, and during diastole active transport of these ions cost extra metabolic energy in restoring the ions before next action potentials[7]. Viewing the heart as biomechanical pump, we can imagine that it has both qualitative and quantitative efficiency. The biomechanical work advantage is the ratio of the work done on the heart to that of the work done by the heart[1, 4,8]. Biomechanical efficiency is the ratio of the work output of the heart to its work input[1, 4, 8].

Biomechanical work output can be referred to as the total external work done by the heart while the work input is the addition of work output and thermodynamic heat energy loss[4, 8, 9]. Functional evaluation of the heart is indispensable for the diagnosis and therapy of diseased heart[10]. Biomechanical efficiency has been most important parameters of energy transfer system of the heart[9]. This study sought to evaluate changes in mechanical energy conversion efficiency and pump performance indices of the left ventricle before and after 8-week CGV supplementation in the healthy volunteers. Left ventricular stroke volume, cardiac output, oxygen consumption, arterial blood pressure, work done, total energy consumed, power generated, mechanical energy efficiency, ejection fraction, and mechanical work advantage were measured, derived, or estimated as the case may be before and after CGV supplementation in healthy subjects. CGV is a glutathione-rich supplement that has been reported to promote energy recovery of the heart during exercise as well as patient recovery from heart disease[11,12].

METHODOLOGY

The Healthy Subjects

Twenty-four (24) healthy subjects (age and sex-matched) students of College of Medicine of the University of Lagos, Lagos were studied. A written ethical approval was granted for this study by the Medical Research Grant and Experimentation Ethics Committee, College of Medicine of the University of Lagos, Lagos(CMUL/HREC/06/17/199).Each subject gave informed consent and was served subject information and consent, subject data and subject record forms. They were non-smokers, non-hypertensive (arterial blood pressure ≤ 140/90 mm Hg), non-alcoholic, not on medication, having no sickness or blood transfusion six (6) months prior to the course of the study. The parameters were measured before and after CGV supplementation. The subjects were then followed up daily and/or weekly via social media channels. After the measurements, each subject was administered orally 2320 mg/d CGV capsules (Max International, USA) for eight (8) weeks. At the end of the 8 weeks, all the parameters were measured made again for each subject and the effects of CGV on the parameters were analysed. Results are presented as mean ± standard error of mean (SEM). Statistical analyses were made using Graph Pad Prism 5 and Microsoft Excel 2007. Significance level was accepted at $p < 0.05$.

Measurement of Physical, Arterial Pulse Oxygen Saturation and Blood Pressureand Heart Rate Parameters of the Healthy Subjects

In the laboratory, each subject's age, height, weight (Avery Stadiometer, England), temperature (Infrared Thermometer, China) were measured. Subjects lay supine on a couch and rested for 20 min to come to physiological baseline. Arterial blood pressure (BP) and electrocardiogram (ECG) were measured in this position. Arterial BP was measured on a brachial artery using Omron Automated Sphygmomanometer (Healthcare Co. Ltd. Kyoto, Japan). The ECG waves were measured using a 12-Lead MAC 1200 ST VI.2 machine (GE Medical Systems, Freiburg, Germany). The machine was standardized at each tracing as recommended by the American Heart Association [13,14].

Measurement of Echocardiographic Parameters of the Healthy Subjects

Transthoracic echocardiogram measurement was done for each subject on a clinical bed by a Clinician. Transthoracic echocardiography measuring the 2-dimensional, M-Mode and tissue doppler imaging was performed using Mobile Digital Colour Doppler Ultrasound System (Model SS-8000, Sonomed; Rome, Italy). Echocardiogram parameters were measured in the left lateral decubitus position using standard imaging planes, according to the recommendations of the American Society of Echocardiography 15.Left ventricular end-diastolic volume and end-systolic volume were measured by the biplane Simpson disk method using 2D images from the apical 4- and 2-chamber views[16]. Each parameter obtained from chamber quantification was indexed for body surface area (BSA) when appropriate.

Data Analysis

Body mass index (BMI) was calculated as: $BMI = W \div H^2$ (Kg/m^2); Where W = weight; H = height 17. Body

surface area (BSA) was calculated as: BSA (m^2) = $0.001315 \times H^{1214}$ (cm) × W^{0262} (Kg); Where W = weight; H = height; 0.001315 = constant for Negroes BSA (18). Pulse pressure (PP) was calculated as PP = SBP – DBP (mmHg),where SBP = systolic blood pressure, DBP = diastolic blood pressure 2; mean arterial pressure (MAP) was calculated as MAP = (SBP + 2DBP) ÷ 3 (mmHg), where SBP = systolic blood pressure; DBP = diastolic blood pressure 2; heart rate (HR) was calculated as HR = 25 × 60 ÷ RR interval (b/min) 13, 17; rate-pressure product (RPP) was calculated as RPP = SBP × HR (AU),where SBP = systolic blood pressure; HR = heart rate 19. ECG voltage was calculated as volt. = 10 mm of deflection = 1 mV 13, 14;ECG durations and intervals were calculated as Int. = 25 mm = 1000 msec(13, 14);QT interval represented electrical systole (13, 14); electrical diastole (ED) was calculated as TP line + PR interval or RR interval – QT interval (msec) (13). Left ventricular diastolic area was calculated as $\pi d^2 \div 4$: where $\pi = (22 \div 7)$ (cm^2), d = end-diastolic diameter; left ventricular stroke volume, LVSV (mL) = left ventricular end-diastolic volume – left ventricular end-systolic volume; left ventricular end-diastolic volume (LVEDV) and left ventricular end-systolic volume (LVESV) were measured by the biplane Simpson disk method using 2D images from the apical 4- and 2-chamber views[15,16]; left ventricular ejection fraction, LVEF = LVSV ÷ LVEDV; cardiac output, CO = LVSV × HR where LVSV = left ventricular stroke volume and HR = heart rate; cardiac index, CI = CO ÷ BSA; left ventricular compliance, LVC = LVEDV ÷ VFP, LVFP = left ventricular filling pressure; RAP = right atrial pressure ≡ left ventricular filling pressure4, 8; myocardial oxygen consumption, MVO_2 = (RPP ÷ 30,000)× 0.14 (L/min)1; left ventricular external work done (J), LVW_E = SBP × LVEDV; left ventricular work impacted on the blood (J), LVW_i = PP × LVSV; left ventricular total work done (J), $LVW_T = LVW_E + LVW_i$; left ventricular tension energy (J), LVE_T = SBP × EDV × QT interval; stroke volume energy ratio (mL/J), SVER = LVSV ÷ LVTE; left ventricular total energy consumed (J), LVTE = $LVW_T + LVE_T$; left ventricular work done in diastole (J), LVW_D = LVFP x LVEDV; left ventricular net word done (J), LVWn = $LVW_E + LVW_D$;left ventricular mechanical work advantage, LVMWA = $LVW_T \div LVW_D$; left ventricular power generated (W), LVP = LVW_T × HR ÷ 60[4,8,9,20]; left ventricular mechanical efficiency (%), LVMƐ = $LVW_T \div$ LVTE × 100; left ventricular mechanical efficiency index, LVMƐi = LVMƐ × MVO_2[21].

RESULTS

The Biophysicalarameters of the Healthy Subjects

Table 1 shows the age and height with no change in the weight, body mass index and body temperature of the healthy subjects before and after CGV supplementation.

Effect of Cellgevity Supplementation on Arterial Blood Pressure and Heart Rate Parameters in the Healthy Subjects

Table 2 shows slight reductions in systolic blood pressure, diastolic blood pressure and mean arterial pressure in the subjects after CGV supplementation. In contrast to this, heart rate and rate-pressure product reduced after CGV supplementation in the subjects ($p < 0.05$ and $p < 0.001$ respectively).

Effect of Cellgevity Supplementation on the Electrocardiac Wave Voltage, Duration and Interval of the Ventricle in the Healthy Subjects

R-Wave voltage, electrical diastole (ED), and RR interval increased after CGV supplementation in the subjects ($p < 0.05$). On the other hand, QRS-Wave duration and QT interval reduced after supplementation in the subjects ($p < 0.05$) (Table 3).

Table 1: Showing the Biophysical Parameters of the Healthy Subjects before and after Cellgevity Supplementation

Parameters	Before Supplementation	After Supplementation
Age (years)	23.9 ± 2.5	23.9 ± 2.5$^{\#}$
Height (cm)	170.4 ± 2.3	170.4 ± 2.3$^{\#}$
Weight (Kg)	70.3 ± 3.4	72.1 ± 4.30$^{\#}$
BSA (m^2)	2.03 ± 0.01	2.04 ± 0.01$^{\#}$
Body Temperature (ºC)	36.9 ± 0.10	36.4 ± 0.10$^{\#}$

BSA = body mass index; # = not significant

Table 2: Showing the Arterial Blood Pressure and Heart Rate Parameters in Healthy Subjects before and after Cellgevity Supplementation

Parameters	Before Supplementation	After Supplementation
SBP (mm Hg)	118.0 ± 2.5	113.0 ± 3.4$^{\#}$
DBP (mm Hg)	75.0 ± 2.1	70.0 ± 2.2$^{\#}$
PP (mm Hg)	43.0 ± 1.6	43.0 ± 1.8$^{\#}$
MAP (mm Hg)	89.3 ± 3.6	84.3 ± 2.2$^{\#}$
HR (b/min)	73.8 ± 1.3	64.7 ± 2.2*
RPP (AU)	8704.4 ± 279.1	7085.1 ± 229.4***

SBP = systolic blood pressure; DBP = diastolic blood pressure; PP = pulse pressure
MAP = mean arterial pressure; HR = heart rate; RPP = rate-pressure product; AU = arbitrary unit

Table 3: Showing the Electrocardiac Wave Voltage, Duration and Interval of the Ventricle in the Healthy Subjects before and after Cellgevity

Parameters	Before Supplementation	After Supplementation
R-Wave Voltage (msec)	1.60 ± 0.03	1.80 ± 0.03*
QRS-Wave Duration (msec)	78.0 ± 2.30	66.0 ± 3.2*
QT Interval (msec)	375.0 ± 5.50	338.5 ± 6.2*
ED (msec)	445.0 ± 29.2	597.5 ± 37.8*
RR Interval (msec)	795.5 ± 34.1	949.5 ± 24.7*

ED = electrical diastole; * = $p < 0.05$

Effect of Cellgevity Supplementation on the Left Ventricular Pump Performance Indices in the Healthy Subjects

Right atrial pressure (RAP) reduced ($p < 0.05$), whereas left ventricular stroke volume (LVSV), cardiac output (CO), left ventricular compliance (LVC) and cardiac index (CI) increased in the subjects after supplementation ($p < 0.001$). Left ventricular end-systolic volume (LVESV) reduced slightly while left ventricular end-diastolic volume (LVEDV) and left ventricular ejection fraction (LVEF) increased slightly after supplementation in the subjects (Table 4).

Effect of Cellgevity Supplementation on Left Ventricular Energy Conversion Parameters in the Healthy Subjects

From Table 5, estimated myocardial oxygen consumption (MVO_2), left ventricular work done in diastole (LVW_D), left ventricular tension energy (LVE_T), left ventricular power generated (LVP) and left ventricular mechanical efficiency index (LVMƐi) reduced after supplementation in the subjects ($p < 0.01$, $p < 0.001$, $p < 0.001$, $p < 0.001$ and $p < 0.5$ respectively). However after supplementation, left ventricular external work done (LVW_E), left ventricular work imparted on the blood (LVWi), left ventricular total work done (LVW_T), left ventricular mechanical work advantage (LVMWA), left ventricular total energy (LVTE), stroke volume-energy ratio (SVER), left ventricular mechanical efficiency (LVMƐ) increased ($p < 0.001$). There was little or no increase in the net left ventricular work done (LVWn) (Table 5).

Table 4: Showing the Left VentricularPump Performance Indices in the Healthy Subjects before and after Cellgevity Supplementation

Parameters	Before Supplementation	After Supplementation
RAP (mm Hg)	11.6 ± 0.4	7.3 ± 0.5*
LVEDV (mL)	132.3 ± 7.8	145.3 ± 7.3#
LVESV (mL)	61.5 ± 3.1	55.7 ± 2.2#
Left Ventricular Stroke Volume (mL)	71.6 ± 3.4	89.6 ± 2.5***
Cardiac Output (mL/min)	5284.1 ± 29.3	5797.1 ± 31.4***
Left Ventricular Ejection Fraction (%)	54.1 ± 2.5	61.7 ± 1.9#
LVC (mL/mm Hg)	11.4 ± 1.2	19.9 ± 2.1***
Cardiac Index (mL/m²/min)	2603.1 ± 12.3	2841.7 ± 15.6***

= not significant; * = $p < 0.05$ and *** = $p < 0.001$ = significant

Table 5: Showing Left Ventricular Energy Conversion Parameters in the Healthy Subjects before and after Cellgevity Supplementation

Parameters	Before Supplementation	After Supplementation
MVO_2 (L/min)	0.041 ± 0.01	0.031 ± 0.01**
LVW_D (J)	0.21 ± 0.002	0.15 ± 0.001***
LVW_E (J)	2.15 ± 0.003	2.24 ± 0.003***
LVWn	2.36 ± 0.005	2.37 ± 0.004#
LVW_I(J)	0.41 ± 0.001	0.53 ± 0.001***
LVW_T (J)	2.56 ± 0.002	2.77 ± 0.002***
LVMWA	10.20 ± 0.71	15.50 ± 1.13***
LVE_T (J)	0.80 ± 0.00002	0.78 ± 0.00001***
LVTE (J)	3.4 ± 0.003	3.6 ± 0.003***
SVER (mL/J)	21.1 ± 1.0	24.8 ± 0.70***
LVP (W)	3.28 ± 0.004	3.03 ± 0.004***
LVMƐ (%)	75.3 ± 0.06	76.9 ± 0.06***
LVMƐi	0.96 ± 0.003	0.62 ± 0.002*

MVO = myocardial oxygen consumption; LVW_D = left ventricle work done in diastole; LVW_E = left ventricular external work; LVWn = net left ventricular work done; LVW_i = left ventricular work impacted on blood; LVW_T = left ventricular total work done; LVMWA = left ventricular mechanical work advantage; LVE_T = left ventricular tension energy; LVTE = left ventricular total energy; SVER = stroke volume ejection ratio; LVP = left ventricular power generated; LVMƐ = left ventricular mechanical efficiency; LVMƐi= left ventricular mechanical efficiency index; # = not significant; * = $p < 0.05$, ** = $p < 0.01$ and *** = $p < 0.001$ = significant

DISCUSSION

Physical Parameters of the Healthy Subjects

The physical parameters (height, weight, body mass index, body surface area and body temperature) of the healthy subjects show that the healthy subjects had normal and healthy stature. The normal body temperature shows that they were not having any feveror sickness[22].

Effect of Cellgevity Supplementation on the Left Ventricular Pump Performance Indices in the Healthy Subjects

The increased left ventricular stroke volume (LVSV), cardiac output (CO), and slight increase in left ventricular ejection fraction (LVEF) are indicators of improved pump performance of the left ventricle after CGV supplementation in the healthy subjects[4, 10].

The decrease in the right atrial pressure (RAP) is an indication that there is decrease in left ventricular filling pressure (LVFP)[2,4]. At the end of ventricular filling, the RAP is equivalent to the LVFP[2,4]. The concomitant decrease in the LVFP with the slight increase in left ventricular end diastolic volume (LVEDV) or volume preload translated to the increase in the left ventricular compliance (LVC), indicating an increase in volume preload[4,8]. The increase in the LVC after supplementation is an indication of improvement in ventricular Starling elasticity during ventricular filling with blood at late diastole[4,8]. The overbalancing increasein the LVEDV over left ventricular end-systolic volume (LVESV) translated to increase in LVSV, suggestively increasing the volume afterload for the ejection of the increased LVSV obtained at the ending of systoleafter the supplementation[4,8,10]. The increase in LVSV corroborates with the increase in LVC. The slight increase in left ventricular ejection fraction (LVEF) was consequent to the slight increase in LVEDV in spite of the increase in LVSV. The increase in LVSV in the subjects after supplementation translated to increase in CO in spite of the concomitant reduction in HR[2,3,4]. The increase in CO in turn is translated to the increase in cardiac index (CI)[4].The increase in the LVSV despite the decrease in QRS duration and QT interval (which are indicators of reduced period of ventricular depolarization and contraction) may have been possible with increase in strength of left ventricular contraction[4]. The increase in LVEDV may have facilitatedventricular stretch, sarcomere length, maximum force of contraction and potential energy needed for the increased LVSV[4]. The increase in R-wave voltage suggests possible increase in electrical energy needed for stronger left ventricular contraction4. However, the increased electrical diastole (ED) and RR interval indicate prolonged left ventricular relaxation time and cardiac cycle period (which corroborates with the reduction in HR obtained) in spite of the shortened systole or period of contraction (QT interval): faster and effective contraction and prolonged relaxation; producing improved pump performance[2,4]. The increased RR interval translated to the HR which produced the reduced rate-pressure product (RPP) together with the slightly reduced SBP[2,4]. The reductions in HR and RPP and the increases in LVSV and CO show that CGV supplementation may be beneficial for improving pump performance and preventing higher rate and pressure of heart beat. Therefore, the increased LVSV, LVEF, CO and CI suggest improvement in pump performance indices after CGV supplementation in healthy subjects[9,10]. Increased CI ultimately suggests improved blood perfusion (at reduced peripheral resistance)to peripheral tissues at reduced MAP in the subjects with CGV supplementation[3, 4, 9, 10, 28].

Effect of Cellgevity Supplementation on the Left Ventricular Energy Conversion Parameters in the Healthy Subjects

The reduction in left ventricular work done in diastole (LVW$_D$) neutralized the increase in left ventricular external work done (LVW$_E$) in systole, producing no changein net left ventricular work done per cardiac cycle (LVWn)[2,3,4], conserving and redistributing the work done in systole and diastole, improving cardiac pump performance after CGV supplementation. Thus, the reduction in LVW$_D$ implies reduction in work done by the blood on the left ventricle to cause the increased LVEDV or preload at a reduced LVFP(4). During diastole, the metabolic work done in actively pumping Na^+, K^+ across myocardial membraneand Ca^{2+} across sarcotubularmembrane by Na^+-K^+ATPases and $2Na^+$-Ca^{2+}ATPases respectively may have reduced after CGV supplementation[2,4]. The reduction in MVo_2 shows reduction in oxygen cost and energy wastage. Contractile machinery of the left ventricle may have been molecularly modeled to reduce oxygen consumption needed while improving pump performance. The reduced Mvo_2 was translated from RPP (i.e. product of HR and SBP)[1]. The reduction in QT interval or contraction time corroborates and justifies the reduction in Mvo_2. Increased R-wave voltage indicates increased ventricular depolarization and electrical energy (at reducedmyocardial oxygen consumed) impacted that may have improved left ventricle contraction in the subjects after CGV supplementation[7]. Thus, the increase in left ventricular external work (LVW$_E$), left ventricular work impacted on blood (LVWi), left ventricular total work done (LVW$_T$), left ventricular mechanical work advantage (LVWMA), left ventricular total energy (LVTE), stroke volume-energy ratio (SVER), left ventricular mechanical efficiency (LVMƐ) and the complementary reduction in left ventricle work done in diastole (LVW$_D$), left ventricular tension energy (LVT$_E$), left ventricular power generated (LVP), and left ventricular mechanical efficiency index (LVMƐi) may have been subsequent to decreased MVo_2, improving left ventricular pump performance and mechanical energy conversion efficiency at a reduced oxygen cost or efficiency index[1,9].

The increased LVW$_E$ suggests improvement in the generated pressure-volume energy loop during

systole and isovolumetric contraction on the increased LVEDV with decreased SBP and QT interval in the left ventricle.The reduced SBP may have produced reduction in temperature during isovolumetric contraction – ideal gas law[4]. The increased LVEDV might have increased the volume preload leading to more recruitment of left ventricular myofibril and stronger contraction and increased volume afterload and the subsequent LVSV[9]. The increased LVWE and LVWi translated to increased LVWT showing increased work done on the increased LVEDV or increased volume preload and afterload and theincreased in LVSV ejected[4]. The increased LVWT suggests more of themechanical energy loop may have been converted into useful mechanical work output after supplementation. This is corroborated with the reduction in LVET obtained which is an indicator of reduced tension heat energy wasted during systole[4]. The increased LVMWA shows an overbalancing increase in energy output (LVWT) during systole against the reduction work done on the ventricle by the blood (LVWD) during diastole. The increased LVTE is subsequent to increased LVWT in spite of the decreased LVET while the increased SVER is subsequent to the overbalancing increase in LVSV to that of LVTE 8, whereas the increased LVMƐ is subsequent to increasedLVWT over that of LVTE[4].

The improvements in the LVMWA and the left ventricular pump performance indices (LVSV, CO, LVEF and CI) justify the improvements in the in the LVWT, LVTE and LVMƐ (i.e. mechanical energy conversion efficiency of the left ventricle) obtained in the subjects after CGV supplementation(9, 26). The decreased LVMƐi shows improved LVMƐ at a reduced MVo_2 (i.e. indirectly oxygen and calories) cost in the left ventricular myofibrilafter supplementation in the subjects. The normal expectation would have been increased Mvo_2 for the increased LVWT and LVTE which was not obtained in this study, but rather decreased Mvo_2 was obtained for the increased LVWT and LVTE. This decrease in Mvo_2 corroborate with the improvements in the LVMWA, LVTE, LVMƐ and the complementary decrease in LVMƐi in the subjects after CGV supplementation[1,8,9]. Therefore, our study shows that increases in left ventricular pump performance and mechanical energy conversion efficiency indices were obtained at reduced Mvo_2 in the healthy subjects following CGV supplementation, instead of the normal increase in Mvo_2 for an increase in output at similar ventricular efficiency and pump performance[1,9].

Our findings suggest improvements in the: i, efficiency of electrochemical and electromechanical energy transduction in the left ventricular contractile machinery; ii, mechanical energy transfer through the increased LVSV ejected into the arterial system (since it is haemodynamically assumed that the ventricular pressure during ejection is closed to the end-systolic pressure at which the LVSV ejected traveled to the peripheral tissues); iii, volume loading than pressure loading during cardiac cycle in the subjects after CGV supplementation[6, 7, 23)].

Left ventricular mechanical work/energy conversion efficiency, pump performance, thermodynamics and work input/work output of the human heart have been something difficult to quantify, but several scholars have used several methods for estimations rather actual quantifications or measurements. These authors have employed biomechanical methods like pressure-volume area, pressure-volume loop, tension-heat-myofibril extension, blood mass-acceleration assessment, blood-density-volume-acceleration-gravity, myocardial oxygen consumption/pressure volume loop, and electrical computational model. However, our study employed left ventricular systolic and diastolic volumes, pressures and periods, and arterial pressures measured using electrocardiogram, echocardiogram, and sphygmanometer for the estimations of the left ventricular pump performance and mechanical energy conversion efficiency to investigate the changes obtained before and after CGV supplementation in the healthy subjects[1,4,8,9]. This study shows that pump performance indices and mechanical energy conversion efficiency of the left ventricles in the healthy subjects improved after CGV supplementation, suggesting the possibility of these improvements in healthy humans.

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