EXERGY DESTRUCTION AND ENTROPY GENERATION IN THE SKELETAL AND CARDIAC MUSCLES, LIFESPAN ENTROPY GENERATION BY THE MASSETER MUSCLES DURING CHEWING AND ITS USE AS AN INDICATOR OF THE LIFE EXPECTANCY

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Dedicated to my family…

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ABSTRACT

EXERGY DESTRUCTION AND ENTROPY GENERATION IN THE SKELETAL AND CARDIAC MUSCLES, LIFESPAN ENTROPY GENERATION BY THE MASSETER MUSCLES DURING CHEWING AND ITS USE AS AN INDICATOR OF THE LIFE EXPECTANCY

The lifespan entropy concept suggests that the organisms may have a limited capacity of lifespan entropy generation when they reach to that limit they die. The masseter muscles of a subject who is consuming food along the guide lines of Institute of Medicine (USA) may generate almost 1 x 10^5 J/K of entropy while chewing his food during his 76 years of expected life time. The masseter muscles of an obese person, who uptakes 10 per cent more nutrients generates the same entropy about five years earlier. The cumulative entropy generation decreases as the second law efficiency of the muscle work rises. The results of this study imply that the concept of the lifespan entropy may be correct even at the individual muscle level, and may be employed to estimate the life expectancy of a person. Experimental data from the literature are employed to carry out thermodynamic analyses of the skeletal and cardiac muscle work performance to obtain additional information beyond what the classical medical studies may offer in description of the health problems. The muscle work efficiency decreases as a result of the reduction of the metabolic energy conversion in the mitochondria with metabolic or structural failure or aging. The reduction of the mitochondrial energy generation causes further deterioration of the muscle structure, which causes in turn further decline in the muscle work efficiency. Exergy loss and entropy generation in the muscles increase as a response to the attempts to higher work production. From the thermodynamic perspective, heart can be regarded as a machine that converts part of the consumed power in external power when it pumps blood. In the course of the contraction of the heart muscle, most of the utilized chemical energy transforms into heat and a much lesser portion turns out to be work. The ratio of the work and the chemical energy expenditure is called efficiency of the cardiac contraction, or basically heart efficiency.

ÖZET

İSKELET VE KALP KASLARINDA EKSERJİ YIKIMI VE ENTROPİ ÜRETİMİ, ÇİĞNEME SIRASINDA MASSETER KASLARINDA YAŞAM SÜRESİNCE ENTROPİ ÜRETİMİ VE BUNUN YAŞAM SÜRESİ BEKLENTİSİNİN GÖSTERGESİ OLARAK KULLANIMI

Yaşam süresi entropi kavramı, organizmaların ömürleri boyunca sınırlı entropi üretimi kapasitesine sahip olduklarını ve bu sınıra eriştiklerinde öleceklerini öne sürmektedir. Gıdalarını Institute of Medicine (USA) 'in rehberliği eşliğinde tüketen bir bireyin çiğneme kasları, 76 yıllık beklenilen yaşam süresi boyunca gıdalarını çiğnerken, yaklaşık olarak 1 x 10^5 J/K entropi üretmektedir. Yüzde 10 daha fazla besin alan obez bir kişinin çiğneme kasları, aynı entropiyi yaklaşık 5 yıl daha erken üretmektedir. Kas işinin, ikinci kanun verimliliği arttıkça, kumulatif entropi üretimi azalmaktadır. Bu çalışmanın sonuçları, yaşam süresi entropi kavramının birey kas seviyesinde bile doğru olabileceğini ve kişinin ortalama yaşama süresini tahmin etmek için kullanılabileceğini göstermektedir. Literatürdeki deneysel veriler, sağlık problemlerinin tanımlanmasında, klasik tıbbi çalışmaların ötesinde ek bilgi elde etmek amacıyla, iskelet ve kalp kası iş performansının termodinamik analizlerini gerçekleştirmek için kullanılmıştır. Kas iş verimliliği, metabolik veya yapısal bozulma ya da yaşlanmayla birlikte, mitokondrideki metabolik enerji dönüşümünün azalması sonucu düşmektedir. Mitokondriyal enerji üretiminin azalması, kas yapısının daha da bozulmasına neden olur ve bu da kas iş verimliliğinin daha da azalmasına sebep olmaktadır. Kaslardaki ekserji kaybı ve entropi üretimi, daha yüksek iş üretimi girişimi karşısında artış göstermektedir. Termodinamik açıdan bakıldığında kalp, kanı pompalarken, tüketilen enerjinin bir kısmını harici bir güce dönüştüren bir makine olarak görülebilir. Kalp kasının kasılması sırasında, kullanılan kimyasal enerjinin çoğu ısıya ve çok daha az bir kısmı işe dönüşmektedir. İş ve kimyasal enerji tüketiminin oranına, kardiyak kasılma verimliliği veya esasında kalp verimliliği denmektedir.

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LIST OF SYMBOLS/ABBREVIATIONS

C_p	Heat capacity
Ex	Total exergy
ex	Specific exergy
H	Total enthalpy
h	Specific enthalpy
m	Mass
MW	Molecular weight
Q	Heat
S	Total entropy
s	Controller state vector
T	Temperature
W	Work
ΔE	Energy change
η_{II}	The second law efficiency
η_{m1}	Exergy efficiency of the metabolism
η_{m2}	Exergy efficiency of the mechanical work
μ	Chemical potential
acc	Accumulation
ch	Chemical
cl	Contralateral
exp	Experiment
gen	Generation
il	Ipsilateral
in	Inlet
oc	Oral cavity
oc_b	Oral cavity boundary
out	Outlet

1. INTRODUCTION

Ageing research follows two major paths: one of these paths focuses on the biological nature of the ageing and uses highly advanced level of experimental observation techniques to understand how and why the cells age. Another major path employs the fundamental principles of the thermodynamics to explore the nature of the same phenomena by referring to advanced level of mathematical analysis. With the advancement of the experimental techniques, it became possible to trace the consequences of the ageing-related microscopic changes in the cell morphology [1,2]. The gene level ageing occurs with the distortion of the DNA strands [3] and the cells attempt to counter this process via destroying the mutated strands [4]. The telomere-induced cellular ageing theory suggests the presence of a gene level built-in program, which triggers death to prevent the mutations to propagate to the forthcoming generations [5]. In some biological studies, fundamental rules of thermodynamics are mentioned and the terminology of thermodynamics is used without referring to mathematical tools [6]. Avoiding mathematics while trying to understand, consequently slow down, and hopefully stop such comprehensive phenomena, while using the nomenclature adopted from the science of thermodynamics is confusing [7].

There is considerable work done on the thermodynamics modelling path of the ageing studies. Ilya Prigogine was awarded the Nobel Prize in Chemistry in 1977 for his pioneering work on the dissipative structures [8,9]. The dissipative structure theory states that the open systems are capable of importing energy continuously from the environment while exporting entropy [8,10]. The concept of the dissipative structures when considered together with the other topics of non-equilibrium thermodynamics is proved to be quite useful in chemical, biological and mechanical engineering applications [11]. The dissipative systems must not only increase their entropy exporting potential but also eliminate the entropy accumulation over time to sustain their growth. Otherwise, the accumulation of positive entropy in the system will eventually bring it to thermodynamic equilibrium with its environment, a state in which the system cannot maintain its order and organisation. Prigogine's theory may lead the way to the entropy theory of ageing [1,12,13,14-17]. Annamalai and Silva [18] made significant contribution to this path of the research by quantifying the ageing stress on the individual organs. Living organisms must maintain a state of high organization [19], where entropy accumulation implies increasing disorder

[20,21]. Metabolic heat, free radicals, etc. are among the causes which result in entropy generation and change of the structure of the biological molecules during the ageing process leading to death. The entropy generation accompanies change of the energy states of biomolecules, which render them inactive or malfunction [7,22]. Also, mutual information content may serve as a resource of free energy through feedback effect and hence the entropy of system can decrease without heat dissipation in living systems by using the information correlation and functional self-organisation as a source of entropy decrease [23,24]. Genetically induced repair and replacement processes are capable of maintaining the balance in favour of the functioning molecules [7,4] , but with ageing the balance between these processes tends toward inactivation and malfunctioning [7]. The events which lead to increase of the cellular entropy during ageing are hypothesised [25] and evidenced [26,27]. The organisms have limited capacity of entropy generation when they reach to that limit they die. There are also views stating that the organisms have evolved over the years to minimise entropy generation [12,13]. This statement may imply the minimisation of the heat release and maximisation of the capability of doing work by the organisms over time. The physics behind entropy generation is described in detail by Boregowda *et al*. [28] and Annamalai and Puri (2011) [29]. In most energy conversion systems, a fraction of energy which is lost due to irreversibility is later released to the environment as heat [30]. The second law of thermodynamics states that every non-isothermal heat transfer increases the entropy of the universe, and the entropy accumulation in the universe is regarded as a kind of environmental pollution [31]. The human lifespan entropy generation was calculated by dividing the metabolic heat release to surface temperature [32,33]. The lifespan entropy generation was related with the body size [34] , heat loss from the body [35,36,16], the level of the physical activity [16] and the composition of the diet [17]. Although the number of genes which are linked in cancer induction is very large, all the cancer cells behave in identical and highly predictable fashion [37]. This behaviour involves changes in motility invasion, replication, nuclear and chromosomal fragmentation structure degradation and phenotypic fluidity. The channels which are distributing the entropy generation are organised along fractal networks. Garland [37] argues that cancer is caused by diversion of the energy needed for structural organisation into maximum entropy dissipation. The oncogenic mutations and molecular alterations in the cell redirect these channels. Oncogenic alterations create cumulative effects by permanently stabilising parts of the fractal network. Garland [37] draws attention to the need for multidisciplinary research

achieves for further understanding of the phenomena, in such a case bridging the gap between the thermodynamic modelling studies with the proposals of the medical researchers would probably be among the best attempts to be done. Garland's [37] discussion regarding the cancer cells appears to be highly applicable to the ageing cells [25].

The energy management in a living cell is called bioenergetics [38]. The useful energy is defined as exergy, which is destroyed in every irreversible process because of entropy production. In the last decade, numerous studies are published on exergy analysis in the biological systems regarding the assessment of the efficiency of the exergy utilisation in metabolism driven processes [39-44], thermal comfort [45], physical exercise [16] and non-equilibrium thermodynamics analysis as applied to cellular systems [46,38,11,10]. Masseter muscle as a skeletal muscle is capable of generating faster contractions and higher force productions [47] required specially during chewing function [48]. However, it is also reflexly controlled to maintain a constantly chewing rhythm under various food resistance conditions [49]. Swallowing and speech are the other two activities that masseter muscle participates bilateral or unilateral besides mastication function [50,51].

Therefore, this study investigates the ageing of the masseter muscles, which is a highly functional skeletal of the human body. The chewing process is analysed thermodynamically to determine the heat, work and entropy generation both in the masseter muscles and the oral cavity. The lifespan entropy generation of masseter muscles is estimated based on the type of the food chewed and calorie intake. Although the ageing of the masseter muscle is not considered as a direct cause of the death, lifespan entropy accumulation by these muscles may be used as an indicator of the life expectancy.

The second law of thermodynamics accounts for the energy dissipation via entropy production in any process, including the living organisms [19]. Organisms manage a combined entropy process in every constituent of their systems, a change in any subsystem influences the others and the entire system [52,19,53,37,54]. Entropy generation within the organisms has been the subject of numerous studies [12,55,13,36,56,16,17,57]; some of these studies focus explicitly on exercise [56,57,16,17,58].

1.1. MUSCLE WORK

The chemical energy in the muscles is converted into work by biochemical reactions. The number of moles of ATP produced as a result of the catabolism depends on the type of the nutrient and the metabolic pathway. For example, 30 to 38 moles of ATP are produced during the oxidation of one mole of glucose [43]. The production of 30 moles of ATP may be expressed with the following apparent reaction as:

$$C_6H_{12}O_{6\ muscle} + 6O_2 \xrightarrow{30\ ADP+30\ P \rightarrow 30\ ATP} 6CO_2 + 6H_2O \tag{1.1}$$

Similarly, ATP utilization for work production may be represented as:

$$ATP_{muscle} \xrightarrow{muscle\ contraction} ADP + P_i \tag{1.2}$$

The conversion ratio of the nutrients into ATP is determined by the metabolic efficiency. The conversion of the ATP into muscle work is evaluated by the muscle work efficiency. In Figure 1.1, exergy efficiency of the metabolism, η_{m1}, and exergy efficiency of the mechanical work, η_{m2} are described with the equations:

$$\eta_{m1} = \frac{ATP\ exergy}{nutrients\ exergy} \tag{1.3}$$

$$\eta_{m2} = \frac{muscle\ work}{ATP\ exergy} \tag{1.4}$$

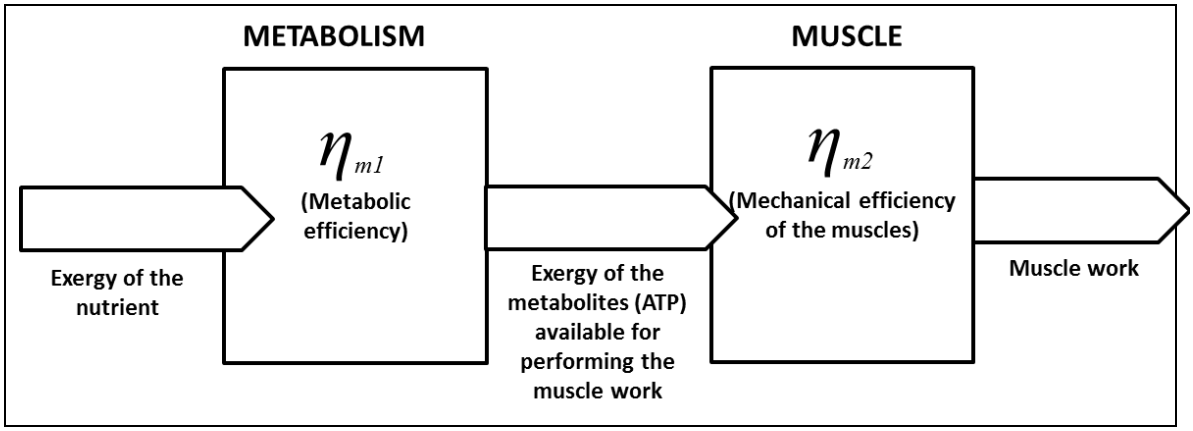


Figure 1.1. Schematic description of the metabolic and the mechanical muscle efficiencies on the conversion of the exergy of the nutrients to muscle work

The product of the metabolic and mechanical work efficiencies can be expressed with the 2nd law efficiency:

$$\eta_{II} = \eta_{m1}\ \eta_{m2} \tag{1.5}$$

The efficiency of muscle contraction measures how much external work is obtained from the input of chemical energy [40]. The first law efficiency, also called the mechanical or thermodynamic efficiency, is defined as:

$$\eta_{I} = \frac{W_{muscle}}{\Delta H_{glycolysis}} \tag{1.6}$$

The second law efficiency is defined as the ratio of the actually produced work to the maximum available work.

$$\eta_{II} = \frac{W_{muscle}}{W_{max}} = \frac{W_{muscle}}{\Delta G_{glycolysis}} \tag{1.7}$$

After substituting $\Delta G_{glycolysis} = \Delta H_{glycolysis}-\Delta S_{glycolysis}$ in equation (1.7) $\Delta S_{glycolysis}$ may be expressed as

$$\Delta S_{glycolysis} = \Delta H_{glycolysis} - \frac{W}{\eta_{\mu}} \tag{1.8}$$

Here, equation (1.8) muscle work efficiency is the factor which determines the entropy generation within the body as expressed in the literature [59-66]. Outcomes of the muscle work efficiency research are vastly valuable for diversity of disciplines; for example, rehabilitation, sport biomechanics, ergonomics, etc., have a broad application area [67]. The capability to sustain a given level of muscular activity depends on the balance between the energy requirements of the activity and the metabolic capacity of the exercising muscle to provide energy [68-70]. If the energetic requirements surpass the capacity to provide energy, the level of activity cannot be maintained and muscles fatigue is experienced [70].

From the thermodynamic perspective, heart can be regarded as a machine that converts part of the consumed power in external power when it pumps blood. In the course of the contraction of the cardiac muscle, most of the consumed chemical energy transforms into heat and a much lesser portion becomes work. The ratio of the work and the chemical energy expenditure is called efficiency of the cardiac contraction, or basically heart efficiency [71]. The myocardial efficiency is an essential factor of the efficiency of ATP supply to requirement matching. In the case that the heart efficiency of ATP utilization reduces, more ATP would be required per unit work, and thus the myocardial oxygen consumption rate would increase correspondingly. If cardiac ATP generation efficiency is diminished, myocardial oxygen consumption rate rises; however, cardiac output may still decrease owing to insufficient quantities of ATP [72].

1.2. AGING

Aging causes loss of the muscle mass and quality [73,74], and increase of the entropy of the body [7,22,19,13]. Therefore, the aged muscle cell becomes different from the one of a younger person [1]. Schematic description of the changes occurring in the muscles during aging are summarized in Figure 1.2. The loss of skeletal muscle proteins reduces the number of mitochondria [75,76]. Muscle mitochondrial dysfunction leads to metabolic disturbances [77,78]. With the decline in mitochondrial activity, rate of ATP generation slows down [79,80], the lower rate of ATP generation causes fatigability [81]. Increased energy need for pumping ion in active transportation may also cause a decrease in the muscle efficiency [82]. As a result, the elderly people usually perform low levels of exercise efficiency and slow routine in daily living activities [83-85].

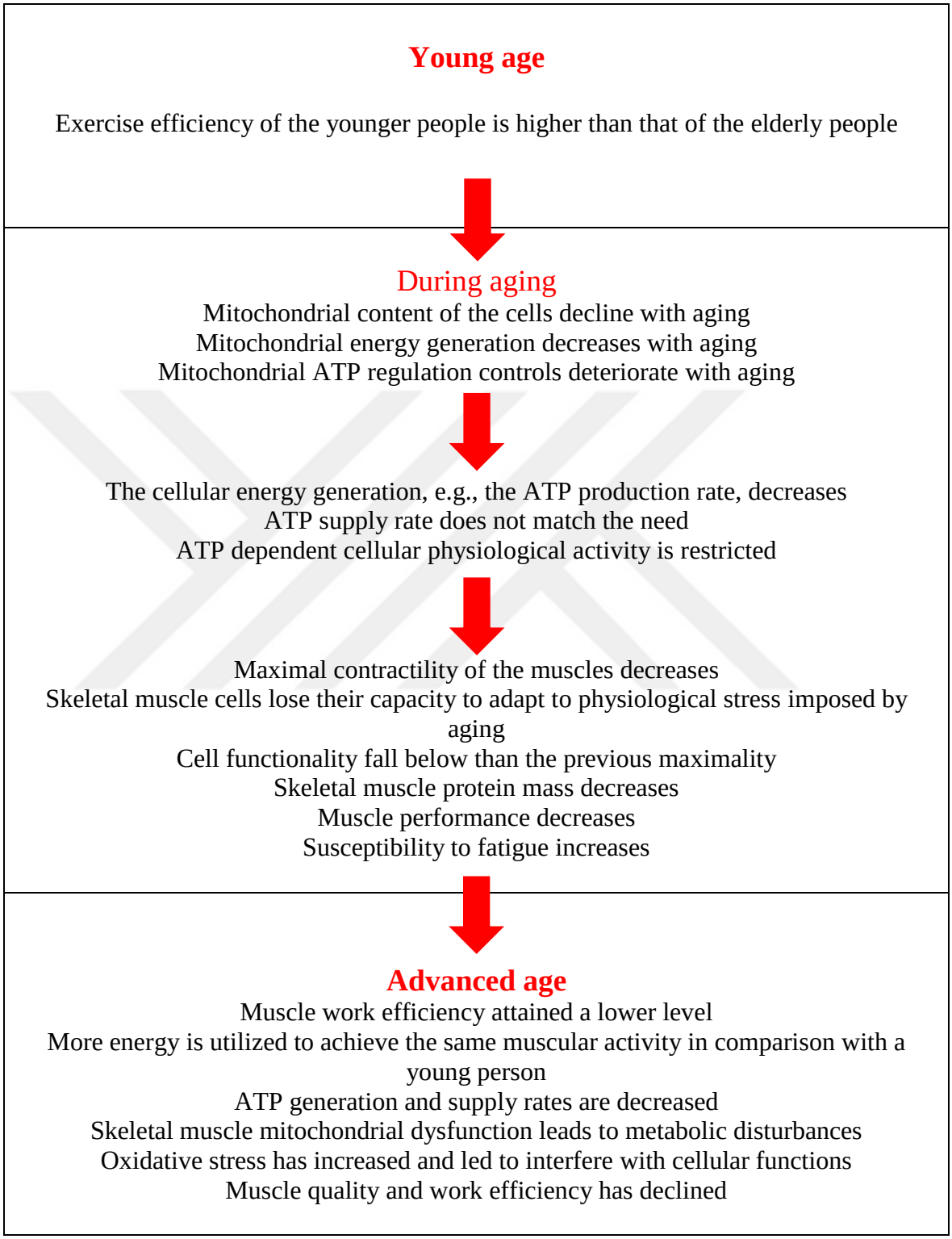


Figure 1.2. Schematic description of the changes occurring during skeletal muscle aging

The development of a heart failure is related with reduced energy metabolism, transient increase in Ca^{2+}, reduction in the ATP production capacity and subsequent reduction in the total ATP concentration and oxygen utilization in both basal and high-demand situations. Decline of these functions occurs only at high demand in healthy aging. In chronic heart failure; levels of the reactive oxygen species (ROS) increases, myocardial antioxidant reserve decreases, the PCr/ATP (phosphocreatine/adenosine triphosphate) ratio reduces, and $NADH/NAD^+$ ratio rises over the entire range of heart work.

1.3. EXERCISING

In short-term exercise blood glucose plays a vital role in the substrate supply to the exercising skeletal muscle. During the forearm and leg exercises, glucose uptake by working muscle rises 20 to 35-fold above the basal level [86,87]. At exercise intensities below 75 per cent of maximal oxygen uptake [88] blood glucose accounts for the carbohydrate consumption [89,90]. Glycogen stored in the muscle tissues and liver serves as a vital energy reserve, especially after the depletion of the glucose.

Muscle tenderness, stiffness, and ache are often related with weakness. Breathing pump muscles can also limit the physical performance in the healthy subjects and athletes [91]. Systematic exercise can increase life span [92] and sedentary men undergoing long-term maintained aerobic oxidative training have higher PCr/ATP ratio versus untrained controls [93]. Thus, a connection between the positive influences of exercise and changes in ATP supply-to-demand mechanisms may occur in aging person [94].

1.4. SOURCES OF ENERGY FOR MUSCLE CONTRACTION

Presence of approximately 4 milli molar of ATP in a muscle fiber is sufficient to maintain full contraction for only 1 to 2 seconds. The most preferred energy source to reconstitute the ATP is phosphocreatine. This high-energetic phosphate bond containing substance has a slightly higher amount of free energy than that of each ATP bond. Total amount of phosphocreatine in the muscle fiber is also limited. The combination of ATP and phosphocreatine may provide energy to cause maximal muscle contraction for only 5 to 8 seconds [95]. The second most preferred energy source is glycolysis. This rapid enzymatic breakdown of the glycogen to pyruvic acid and lactic acid liberates energy that is used to

convert ADP to ATP; the ATP can then be used directly for additional muscle contraction and also to re-form the stores of phosphocreatine. These (ATP, phosphocreatine and glycolysis) mechanisms are important energy sources under anaerobic conditions that can energize muscle contraction up to 1 to 2 minutes, depending on the anaerobic capacity of a person. The third and the least preferred energy source is oxidative metabolism. More than 95 per cent of all energy used by the muscles for long-term contraction is derived from oxidative metabolism of carbohydrates, fats and protein. By far, the greatest proportion of energy comes from fats, but for periods of 2 to 4 hours, as much as one half of the energy can come from stored carbohydrates [95].

1.5. CARDIAC CYCLE

A heart is an open dissipative system performing external work on the blood. As a result of its capability to deliver oxygen and nutrients and eliminate waste products it helps the body to retain the organization of the living structure. The cardiac cycle describes the phases of contraction and relaxation of the heart that drive blood flow throughout the body and the lungs. It refers to a complete heartbeat from its generation to the beginning of the next beat, so includes the diastole, e.g., part of the cycle when the heart refills with blood, the systole, e.g., contraction, the phase when the blood is pumped out, and the intervening pause. The frequency of the cardiac cycle is described by the heart rate. The blood chambers of the heart relax and fill with blood in repeating the cycles. Each beat of the heart involves five major stages. The first two stages, often considered together as the "ventricular filling" stage, involve the movement of blood from the atria into the ventricles. The next three stages involve the movement of blood from the ventricles to the pulmonary artery (in the case of the right ventricle) and the aorta (in the case of the left ventricle).

1.6. CARDIAC OUTPUT AND CARDIAC RESERVE

Cardiac output is the quantity of blood pumped into the aorta by the heart in a minute. The left ventricle is responsible for pumping blood throughout the body (systemic circulation) while a right ventricle sends blood to the lungs for oxygenation and elimination of carbon dioxide (pulmonary circulation). For young, healthy men, under resting conditions, cardiac output averages about 5.5 L/min [95]. However, the heart has the potential to maximize its cardiac output above normal. This is called the cardiac reserve that is about 300 to 400 per

cent in healthy sedentary individuals and can increase a little more in athletically trained persons. Cardiac reserve declines with aging. Blood circulation is directed to tissues depending on their metabolic activity. For example, inactive skeletal muscles of the body receive very low amount of blood (4 mL/min/100 g of muscle), while it can increase up to 20 times (80 mL/min/100 g of muscle) in exercise when muscle metabolic activity is increased. Therefore, a heart allows operation of different pressures in the left and right ventricles with the help of heart valves. A thermodynamic model has recently been offered by Henriques *et al*. [96] to analyze heart work in a simple way .

Cardiac cycle, where energy supplied to the heart muscles is converted in to work operates like a thermal machine [71]. In this process the randomness of the tissue increases due to entropy accumulation, which eventually may lead to cardiac damage [97]. Reduced expression of mitochondrial transcription factors and mitochondrial proteins are involved in the mechanisms leading to mishandling of energy in heart failure [98]. Getting energy to where it is needed is the problem in an enlarged heart [62]. During exercise, circulation of the cardiac output to specific tissues is adjusted according to the metabolic activity of the tissue and most of the cardiac output is devoted to the working skeletal muscles [99]. Annamalai and Silva, analyzed entropy stress on the organs over the life span and ranked them in the order of entropy generation, where heart appeared to be the highest entropy generating and the most entropy-stressed organ [18]. Weakening in the peripheral skeletal muscle function may be a factor in the pathophysiology of heart failure [100].

2. METHODOLOGY

2.1. LIFESPAN ENTROPY GENERATED BY THE MASSETER MUSCLES DURING CHEWING: AN INDICATOR OF THE LIFE EXPECTANCY?

Chewing, which is performed bilateral or unilateral is the first step of the digestion. In our model, mouth is regarded as composed of two subsystems: the oral cavity and the masseter muscles (Figure 2.1). The masseter muscles move the mandibles towards the maxilla and provide force for chewing and biting the food [101-104]. If chewing is unilateral, the contractile activities of masseter muscles on the chewing side are in the ipsilateral side and non-chewing side are in the contralateral side. However, regardless whether they contract bilateral or unilaterally, masseter muscles mainly contract concentric during the phase of jaw closing and isometric during the phase of dental occlusion [50]. The neuromuscular control of chewing is required to maintain a constant chewing rhythm under varying food resistance conditions in which the jaw muscle activity, especially the masseter muscle, needed to break solid food [105,49].

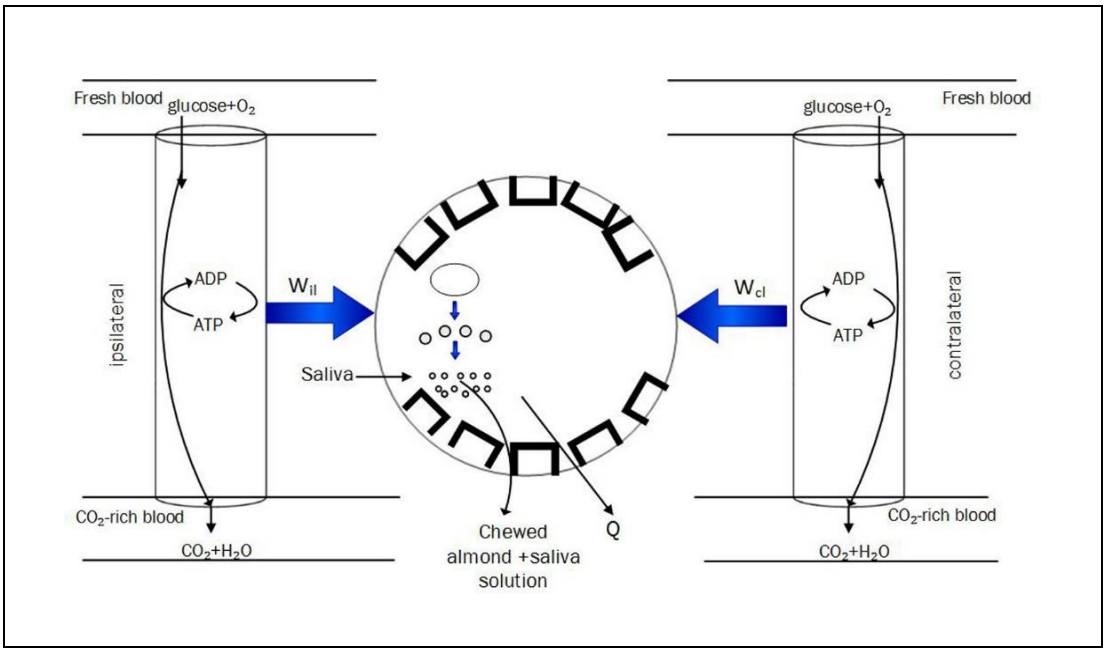


Figure 2.1. The schematic description of the systems involved in the chewing process within the context of this study, the ipsilateral muscle is on the right side of the jaw of the subject, while the contralateral muscle is on the left

In the following analysis, the powerful chewing process begins as a piece of almond is placed into the mouth. Then the masseter muscles move the mandibles 25 times up and down, until the almond is grinded into very small pieces, mixed with the saliva and send to the oesophagus as bolus. Duration of the chewing process is 20 s. Mass, energy and energy analyses are performed for both of the subsystems, i.e., the oral cavity and the muscles. The total entropy generation is calculated. These data are extrapolated to estimate the lifespan entropy of the masseter muscles.

2.2. MUSCLE WORK PERFORMANCE BY THE LEFT AND RIGHT HEART IN ONE BEAT

Mass, energy, exergy and entropy balances are performed around the muscles to calculate the glucose consumption, exergy destruction and entropy generation. A human heart is modeled as the left and the right ventricle muscle subsystems in the present study.

The heartbeat data were adapted from [95]. Heartbeat rate was 75 beats / min, duration of each cycle was 0.8 s and the coronary artery and the left main coronary artery resting blood flows were 0.25 L/min. Energy balance around the muscle system (Figure 2.2.) requires:

$$Q \; -W \; + \sum_{i}^{4} (mh)_{in} - \sum_{i}^{4} (mh)_{out} = \Delta E = 0 \tag{2.1}$$

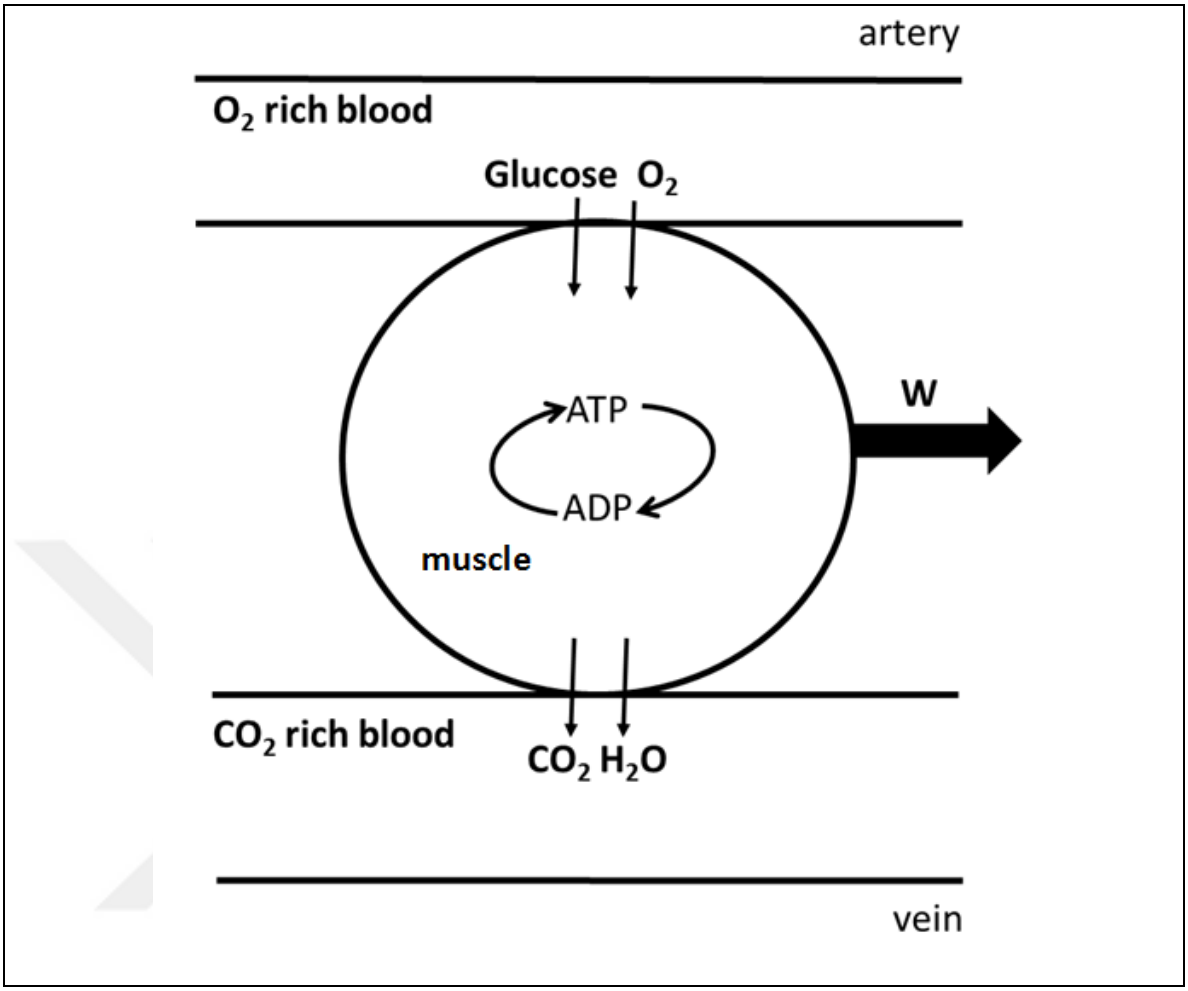


Figure 2.2. The schematic description of the muscle contraction process

Where i=1, 2, 3 and 4 refer to glucose, oxygen, carbon dioxide and water, respectively. Heartbeat works of the left and right ventricles were calculated from the Wiggers diagrams, standard diagrams used in physiology for a cardiac cycle. During contraction under the steady state conditions $\Delta E = 0$. Heat released during work performance from the muscles was calculated from equation 2.1 with the data presented in Table 2.1.

Table 2.1. Chemical composition, mass, heat capacity, enthalpy of formation, absolute entropy and chemical exergy of the constituents entering and leaving through the system boundaries.

Chemical species	**Mass input to System I (g)**	**Mass Output from System I (g)**	**Δh_f^0 (kJ/g)**	**C_p (kJ/g K)**	**Δh_{f310K}^{**} (kJ/g)**	**s^0 (kJ/g K)**	**ex^0 (kJ/g)**
System - Muscle cycle							
Glucose	2.1×10^{-4}*		-7.05 [43]	1.25×10^{-3} [106]	-7.04	1.16×10^{-3}	11.47 [43]
Oxygen	2.2×10^{-4}*		-3.8×10^{-1} [43]	9.20×10^{-4} [106]	-3.7×10^{-1}	6.41×10^{-3}	2.7×10^{-1} [43]
Carbon dioxide		3×10^{-4} *	-15.93 [43]	8.40×10^{-4} [106]	-15.92	4.89×10^{-3}	-3.11 [43]
Water		1.2×10^{-4} *	-15.97 [43]	4.18×10^{-3} [106]	-15.92	5.05×10^{-4}	-9.24

* The stoichiometry of the reaction $C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_2O$, the molar concentrations are converted into mass concentrations

**Calculated by $\Delta H^0_{f310K} = \Delta H_f^0 + CpdT$

Exergy destroyed in the blood stream was calculated as:

$$Ex_{destroyed} = Q\left(1 - \frac{T_0}{T}\right) - W + (m\ ex)_{in} - (m\ ex)_{out} \tag{2.2}$$

where T is the temperature at the boundary of the muscle and T_0 is the reference temperature. The specific exergy of the species is calculated from their chemical composition and thermophysical state:

$$ex = ex_{ch} + h - T_0 s - \sum x_i \mu_i^0 \tag{2.3}$$

where, μ_i^0 is the chemical potential of the pure species and $Ex_{destroyed}$ may be regarded as the exergy loss in the blood stream, and equals to multiplication of entropy accumulation in the blood stream and the body temperature as provided in equation 2.4. Here $T = T_{in}$ is the temperature at the boundary of the muscle system, and $T_0 = T_{out}$ is the reference temperature. Exergy of formation of each chemical is listed in Table 2.1, when $T_0 = T_{out}$ = 298 K, and $T=T_{in}$= 310 K. Muscle work performance is achieved with the consumption of ATP; heat generation is an inevitable part of the process and equals to about 2/3 of the enthalpy change of the glucose to metabolic end-product conversion reactions. After assuming that heat transfer from blood to the muscle occurs at 310 K and from the muscle to air at 298 K, we calculate S_{gen} (J/K) for the muscle as:

$$S_{gen} = \frac{Ex_{destroyed,}}{T_o} \tag{2.4}$$

3. RESULTS AND DISCUSSION

3.1. THE ORAL CAVITY

The oral cavity system contains the teeth, upper and lower jaw and tongue. At the initial state, system boundaries enclose the empty oral cavity. One piece of almond, which is in thermal equilibrium with the oral cavity (i.e., at 37°C), is placed on the tongue. Properties of the almond are adapted from Paphangkorakit *et al*. [107]. Then the chewing process begins. During the chewing process, salivary glands release saliva in a range of 0.53–8.64 mL/min [108]; saliva secretion rate is assumed to be constant at 5 mL/min, 99.5 per cent of the saliva is water, and the rest is a mixture of enzymes. Saliva is assumed to be in thermal equilibrium with the oral cavity. Thermodynamic data of saliva are assumed to be the same as pure water, since more than 99.5 per cent of the saliva is constituted of water [102]. Therefore, the other constituents, e.g., amylase and lysosomes are expected to have negligible effect on the calculations of enthalpy, entropy and exergy of the formation.

Almond contains lipid, protein, carbohydrate, fibre, some minerals and water are modelled as a mixture of the chemicals given by the Almond Board of California [109]. All the lipid in almond is taken as oleic acid, all the protein as glutamic acid, all the carbohydrates as sucrose, all fibre as cellulose and all minerals as potassium. The exact chemical composition of the almond is given in Table 3.1.

Table 3.1. Chemical composition, mass, heat capacity, enthalpy of formation, absolute entropy and chemical exergy of the constituents entering and leaving through the system boundaries.

Mass Transfer	Chemical composi-tion	mass (g)	mass (g)	ΔH_f^0 (kJ/g)	C_p (kJ/g K)	ΔH_{f310K}** (kJ/g)	s^0 (kJ/g K)	ex^0 (kJ/g)
	System I - Oral cavity (almond input: 1 g, saliva input: 1.66 g)							
		Input to System I	**Output from System I**					
Almond	Oleic acid	$4.94x10^{-1}$	$4.94x10^{-1}$ [109]	-2.73 [110]	$1.37x10^{-3}$ [111]	-2.72	$2.76x10^{-3}$ [111]	39.54 [112]
	Glutamic acid	$2.12x10^{-1}$	$2.12x10^{-1}$ [109]	-6.82 [113]	$1.19x10^{-3}$ [113]	-6.80	$1.28x10^{-3}$ [113]	16.27 [113]
	Sucrose	$3.89x10^{-2}$	$3.89x10^{-2}$ [109]	-6.49 [114]	$1.24x10^{-3}$ [114]	-6.47	$1.15x10^{-3}$ [114]	17.52 [115]
	Cellulose	$1.22x10^{-1}$	$1.22x10^{-1}$ [109]	-5.67 [116]	$1.07x10^{-3}$ [116]	-5.65	$1.17x10^{-3}$ [117]	18.86 [116]
	Potassium	$1.73x10^{-2}$	$1.73x10^{-2}$ [109]	0 [118]	$7.60x10^{-4}$ [119]	0	$1.64x10^{-3}$ [118]	9.38 [115]
	Water in almond	$4.7x10^{-2}$	-	-15.87 [113]	$4.18x10^{-3}$ [113]	-15.82	$5.05x10^{-4}$ [113]	$5.00x10^{-2}$ [113]
Saliva	Water in saliva	1.66	1.72	-15.87 [113]	$4.18x10^{-3}$ [113]	-15.82	$5.05x10^{-4}$ [113]	$5.00x10^{-2}$ [113]
	System II - Masseter muscle cycle							
		Input to System II	**Output from System II**					
O_2- rich blood	Glucose	$2.1x10^{-4}$*		-7.05 [43]	$1.25x10^{-3}$ [113]	-7.04	$1.16x10^{-3}$	11.47 [43]
	Oxygen	$2.2x10^{-4}$*		$-3.8 x10^{-1}$ [43]	$9.20x10^{-4}$ [113]	$-3.7x10^{-1}$	$6.41x10^{-3}$	$2.7x10^{-1}$ [43]
CO_2- rich blood	Carbon dioxide		$3x10^{-4}$ *	-15.93 [43]	$8.40x10^{-4}$ [113]	-15.92	$4.89x10^{-3}$	-3.11 [43]
	Water		$1.2x10^{-4}$ *	-15.97 [43]	$4.18x10^{-3}$ [113]	-15.92	$5.05x10^{-4}$	-9.24

* Based on the previous calculation of $2.3678x10^{-6}$ mole glucose, and the stoichiometry of the reaction $C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_2O$, the molar concentrations are converted into mass concentrations

**Calculated by $\Delta H^0_{f310K} = \Delta H_f^0 + CpdT$

During the chewing process, masseter muscles on both sides perform forceful movements and provide the work required to grind the almond. The work done by the masseter muscles is calculated based on the force and elongation data given by Hannam *et al*. [120] as detailed in the next section. A fraction of the produced muscle work is used to overcome the friction occurring at the jaw intersection points and between the upper and lower teeth when balancing work happens. Kapur *et al*. [121] defines a masticatory efficiency (η_{mech}) as the ratio of the work used for chewing to the work produced by the muscles. The masticatory efficiency is a mechanical efficiency and it is estimated to be 0.69 [121]. Work input to the oral cavity is then calculated as:

$$W_{oc} = \eta_{mech} W_{muscle} \tag{3.1}$$

During the actual chewing process, the tongue pushes the food to left and right sides of the mouth, so that the masseter muscles on both sides share the work load. In our simplified process, the tongue does not move and the almond remains on the right side of the mouth throughout the whole process. At the end of the chewing process, the grinded almond particles and saliva form a solution and head the way to the oesophagus. At the final state, system boundaries enclose the empty oral cavity. Since the oral cavity is empty both in the initial and the final states, at the end of the chewing process, the first system comes to its initial thermodynamic state. Mass, energy and exergy balances for the oral cavity can be written as:

$$m_{almond} + m_{saliva} - m_{solution} = 0 \tag{3.2}$$

$$Q_{oc} - W_{oc} + (mh)_{almond} + (mh)_{saliva} - (mh)_{solution} = 0 \tag{3.3}$$

$$Ex_{destroyed,oc} = Q_{oc}\left(1 - \frac{T_0}{T_{oc}}\right) - W_{oc} + (m\,ex)_{almond} + (m\,ex)_{saliva} - (m\,ex)_{solution} \tag{3.4}$$

Here T_{oc} is the temperature of the boundary of the oral cavity, and T_0 is the reference temperature. The specific exergy of the species is calculated based on their chemical composition and thermo physical state ($ex = ex_{ch} + h - T_0 s - \sum x_i \mu_i^0$). In this analysis, reference temperature is set as equal to the oral cavity ($T_0 = T_{oc} = 37^o C$). Assuming that the solution heading to esophagus is ideal, the enthalpy difference in Equation (3.3) is zero. Then, the heat released can be calculated by simplifying Equation (3.3) as:

$$Q_{oc} - W_{oc} = 0 \tag{3.5}$$

The entropy generated is calculated as:

$$S_{gen,oc} = \frac{Ex_{destroyed,oc}}{T_o} \tag{3.6}$$

Table 3.1 lists the chemical composition of the almond and the thermodynamic data of each species.

In the oral cavity 9.555×10^{-2} kJ/K of entropy is generated while chewing one gram of almond. This entropy generation is due to the disintegration of the almonds physical structure and the friction between the teeth and the almond.

3.2. THE MASSETER MUSCLES

Work done by the masseter muscles is calculated as:

$$W_{muscle} = \oint_{il} Fdl + \oint_{cl} Fdl \tag{3.7}$$

The work done by the masseter muscle to move the lower jaw in one loop is calculated from the force versus elongation data [120] as 1.56 J for the contralateral muscle and 2.83×10^{-2} J for the ipsilateral masseter muscle (Figure 3.1). Throughout one chewing cycle, the

ipsilateral muscles create forces as large as 40 N, whereas the maximum force created by the contralateral muscles reaches only up to 25 N. However, contraction of the ipsilateral muscles stops during the high force period. Therefore, the total work done by the contralateral muscles is about 55 times larger than the ipsilateral muscles.

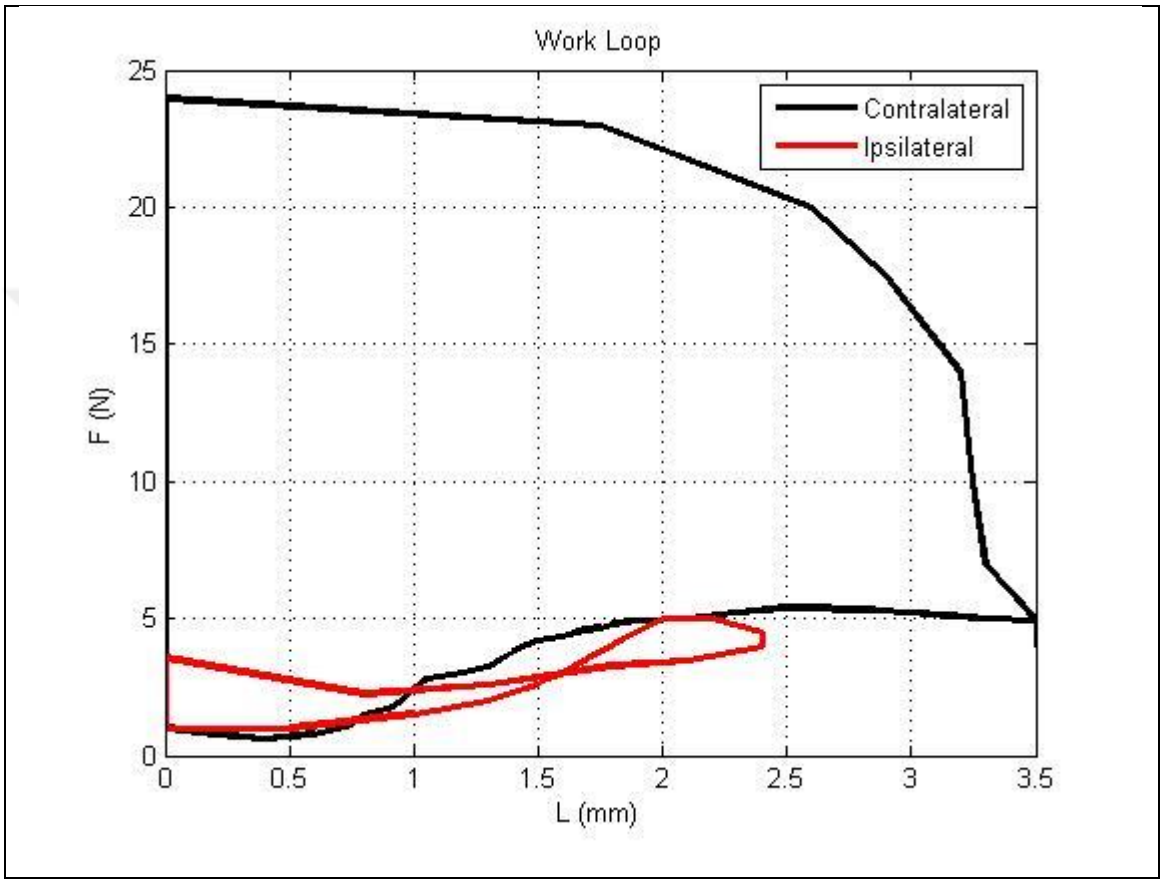


Figure 3.1. The contralateral (top) and ipsilateral (bottom) jaw movement work loops

The muscle cells produce work by consuming chemical potential of the nutrients. The muscle cells are fed with carbohydrate and O_2 rich blood. Carbohydrate, mostly in form of glucose, is metabolized in the muscle cells and adenosine triphosphate (ATP) is produced. Then ATP is dissociated into adenosine diphosphate (ADP) to reposition the myosin head, which causes a contraction in the muscle fibers [122].

In the muscles, chemical energy is converted into mechanical energy, via biochemical reactions, which have high efficiencies [43]. The number of moles of ATP produced as a result of the catabolism depends on the type of the nutrient and on the metabolic pathway. For example 30 to 38 moles of ATP is produced during the oxidation of glucose. Assuming

that only glucose is catabolized by the muscle cells and 30 moles of ATP is formed, then the complicated energy metabolism can be simplified as one chemical reaction, i.e., glycolysis:

$$C_6H_{12}O_6 \xrightarrow{30\,ADP+30\,P\rightarrow 30\,ATP} 6CO_2+6H_2O \tag{3.8}$$

Similarly, work production can be represented as:

$$ATP \xrightarrow{muscle\ contraction} ADP + P_i \tag{3.9}$$

Pioneering work in the field of muscle work was performed by Hill [123], who experimentally showed that both force and the heat released during the contraction of a muscle can be written as a function of the contraction velocity.

The muscle work efficiency is defined based on the first or the second law of thermodynamics. The first law efficiency, which is also called the mechanical or thermodynamic efficiency, is defined as:

$$\eta_{\imath} = \frac{W}{\Delta H_{glycolysis}} \tag{3.10}$$

Recent studies have shown that the heat released during glycolysis and ATP hydrolysis may vary depending on metabolic conditions such as Ca^{2+} concentration. The measured heat release varies between 879 to 4017 kJ for each mole of consumed glucose.

The second law efficiency is defined as the ratio of the actually produced work to the maximum available work.

$$\eta_{\imath\imath} = \frac{W}{W_{max}} = \frac{W}{\Delta G_{glycolysis}} \tag{3.11}$$

Experimental results on the first law efficiencies are reported between 0.14 and 0.35, and the second law efficiencies are reported between 0.17 and 0.42 [124]. These data are based on the experiments done with small animals, like a mouse or a frog. Since data specific to masseter muscles are unavailable, the above mentioned efficiencies are assumed to be also valid for the masseter muscles and used in the further calculations. Equation (3.12) can be used to calculate the mass of the consumed glucose, assuming that the Gibbs free energy of the glycolysis reaction is 3868 kJ/mol of glucose:

$$m_{glucose} = \frac{W}{\eta_{II}} \frac{180.16 \frac{kg}{kmol}}{3.86 \frac{kJ}{kmol}} \tag{3.12}$$

Table 3.2 provides the numerical values of the work performed, exergy destroyed and entropy generated when glucose consumption in association with the almond uptake is 5.95 $\times 10^{-3}$ mmol during 25 cycles of the chewing process. The cumulative entropy generation by the individuals who are consuming 50 per cent and 80 per cent of their food in solid form through the whole lifespan with the second law muscle work efficiency of 0.3 is 96.766 J/K and 155.116 J/K, respectively (Table 3.2). Table 3.3 lists the mass of the consumed glucose, its concentration, entropy generation and exergy destruction as a function of the second law efficiency. As the efficiency increases, a larger fraction of the chemical exergy of glucose is converted into work. Therefore, the amount of glucose needed to achieve a certain amount of muscle work decreases as the efficiency increases.

For a healthy male, typical values for the masseter artery diameter and the flow velocity are given as 1.9 mm and 254 mm/s [125], which results in a volumetric blood flow rate of 4.32 x 10^{-2} L/min. Thus, assuming that η_{II}=0.3 the glucose concentration in the blood decreases by 0.138 mmol/L due to the chewing process. The glucose consumed in the blood concentration varies with the second law efficiency. Glucose consumption declines marginally as the second law efficiency rises to 0.3. However between 0.3 and 0.42, the drop in the consumption is slight. There is a certain and expected relation between entropy generation as well as exergy destruction as it was in the consumption and the second law efficiency (Table 3.3).

Table 3.2. The lifespan entropy generation in the masseter muscles and the oral cavity of a person while taking 50 per cent, and 80 per cent of his calorific need in the form of solid foods.

	Contralateral & Ipsilateral Masseter Muscles (η_{II}=0.3)		**Contralateral & Ipsilateral Masseter Muscles (η_{II}=0.42)**	
	Masseter Muscle	**Oral Cavity**	**Masseter Muscle**	**Oral Cavity**
Glucose consumed (mmol/min)	$5.95x10^{-3}$	----	$4.25x10^{-3}$	----
Work (J)	2.30	1.59	2.30	1.59
Exergy destroyed (25 cycle) (J/K)	5.77	28.5	3.438	28.5
Entropy generation (25 cycle) (J/K)	$1.94x10^{-2}$	$9.55x10^{-2}$	$1.15x10^{-2}$	$9.55x10^{-2}$
Lifespan Entropy Generation (J/K) while taking 50 per cent of the calorific need in form of solid foods	96.766	----	57.657	----
Lifespan Entropy Generation (J/K) while taking 80 per cent of the calorific need in form of solid foods	155.116	----	92.425	----

Table 3.3. Numerical values of the glucose consumption rate, exergy destroyed and the entropy generated in the control volumes of masseter masseter muscles due to 2nd law efficiency range.

η_{II}	$m_{glucose}$ (mmol/min)	Glucose Concentration in Blood (mmol/L)	$Ex_{destroyed,muscle}$ (kJ)	$S_{gen,muscle}$ (kJ/K)
0.17	$10.50x10^{-3}$	0.243	$12x10^{-3}$	$4.03x10^{-5}$
0.22	$8.12x10^{-3}$	0.188	$8.7x10^{-3}$	$2.93x10^{-5}$
0.26	$6.87x10^{-3}$	0.159	$7x10^{-3}$	$2.35x10^{-5}$
0.3	$5.95x10^{-3}$	0.138	$5.7x10^{-3}$	$1.93x10^{-5}$
0.34	$5.25x10^{-3}$	0.121	$4.8x10^{-3}$	$1.6x10^{-5}$
0.4	$4.46x10^{-3}$	0.103	$3.7x10^{-3}$	$1.25x10^{-5}$
0.42	$4.25x10^{-3}$	0.098	$3.4x10^{-3}$	$1.15x10^{-5}$

Exergy loss during the chewing process is depicted in Figure 3.2; Figure 3.3 simulates the entropy generation as a function of the efficiency. Entropy generated in the masseter muscle and the oral cavity is different from each other. The latter is five times larger than the former due to the masticatory efficiency and the second law efficiency.

3.3. LIFE EXPECTANCY

Our calculations show that the masseter muscles generate 4.03 x 10^{-5} to 1.15 x 10^{-5} kJ/K of entropy while chewing one gram of almond. Assuming that the work required to chew any solid food is the same as to chew an almond, then the total entropy generation in the masseter muscles within the lifespan of a person can be estimated. The recommended daily calorie intake for healthy living is adapted from the Institute of Medicine (USA) [126] data in Figure 3.4. Assuming that 50 per cent of the calorific need is taken from solid foods, then a 26 year old healthy male would need to chew solid foods with a calorific value of 1250 kcal per day. Assuming that 1 gram of solid food provides 5.85 kcal (24.25 kJ) leads the calculation of

$2.5x10^{-5}$ kJ/K of entropy generation per day. The cumulative entropy generation in the masseter muscles is shown as a function of age in Figures 3.5 and 3.6.

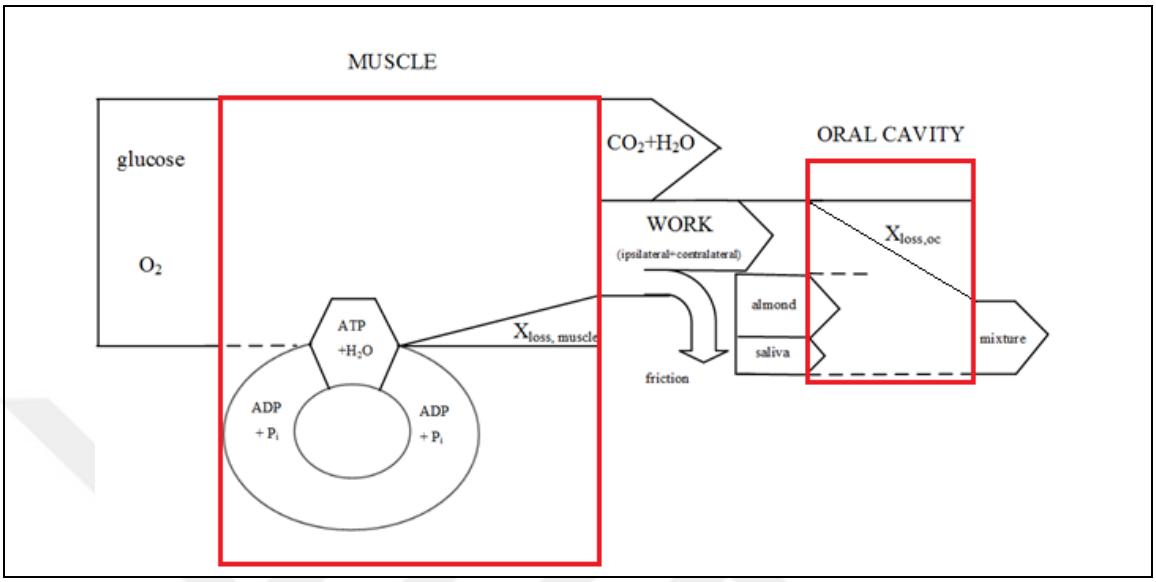


Figure 3.2. Schematic diagram of the exergy balance for the masseter muscle

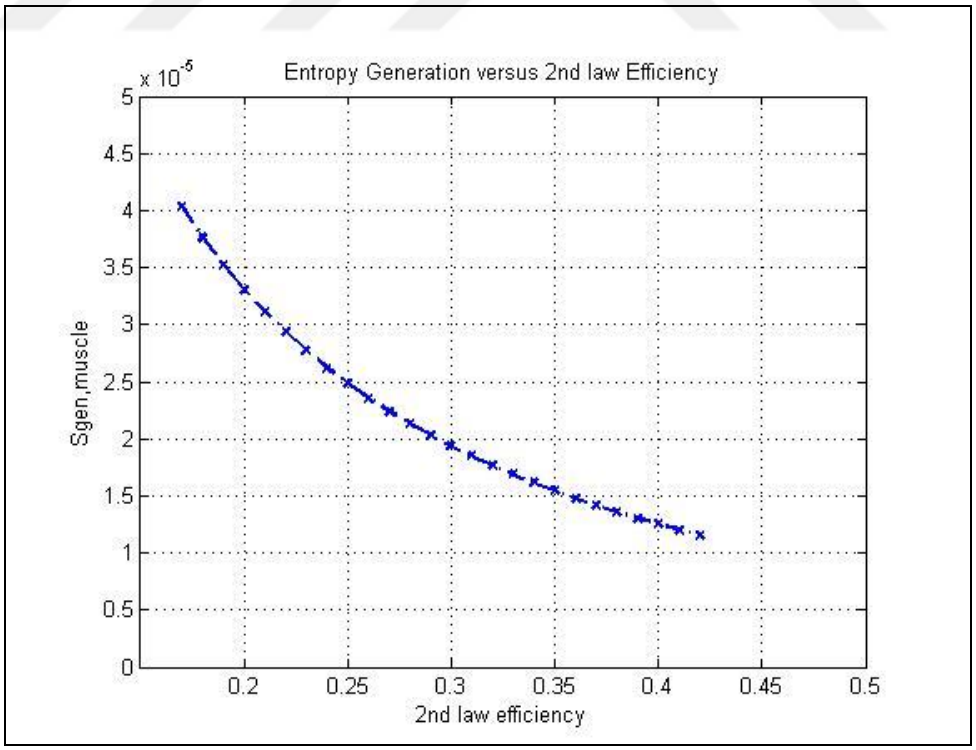


Figure 3.3. Entropy generation of muscle versus 2nd law efficiency

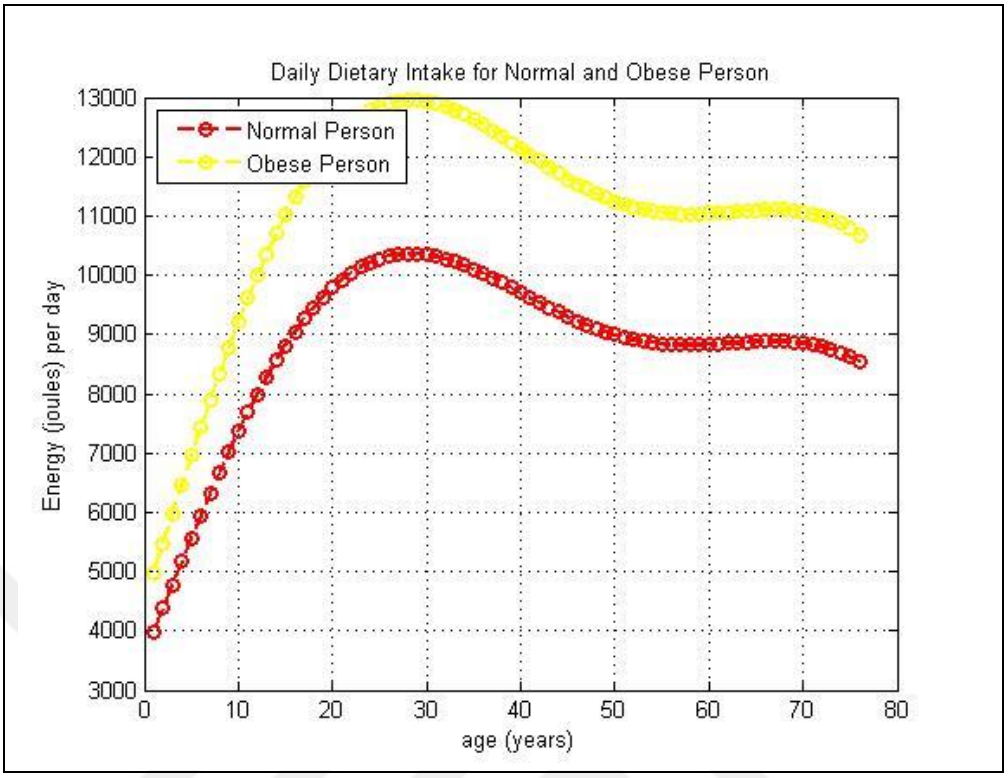


Figure 3.4. Energy intake data (adapted from the Institute of Medicine). Level of obese person is assumed to consume 10 per cent more intake

Figure 3.5 indicates that a healthy individual who consumes half of his calorific need from solid foods and the remaining part from liquid foods, all his life and has masseter muscles with a second law efficiency of 0.3, then he generates almost $1x10^5$ J/K of entropy in his masseter muscles until his age reaches to 76, while chewing his food. This amount does not include the entropy generated by this muscle for the other activities, such as speaking. Figure 3.5 also shows that an obese person is expected to generate the same amount of the entropy at the age of 71 years. However, according to the Figure 3.6, a normal individual who consumes 80 per cent of his food in solid form through his lifespan and has masseter muscles with the second law efficiency of 0.3, generates the same amount of entropy at the age of 51 years, an obese generates it when he is 46 years old. Furthermore; the normal individual can generate $1x10^5$ J/K of entropy when he is 76 years old if the glycolysis reaction occurs more efficiently such as 0.42. Thus, it is clear that an individual generates less entropy if he consumes more liquid with an efficient glycolysis reaction in regard to Figure 3.5 It is not possible to assess the results of this further, at the moment, because there are no clear cut tables available yet to relate the life expectancy with the percentage of the food consumed

over the daily intake recommended by the dietitians. The concept developed by Silva and Annamalai [17], and as supported by the findings of their paper, maybe instrumental to collect such data in the future. The chewing data employed in this study are personal. The work produced by the masseter muscles is different for every individual, depending on his jaw structure and the chewing behavior.

Ageing may be accelerated by many factors, including free radicals, ionization-radiation, ultraviolet light and lack of correlation between mutual information [127-129,23,24]. Comparison of Figures 3.5 and 3.6 show that the masseter muscles of a normal person generates more entropy when he consumes food with larger solids content.

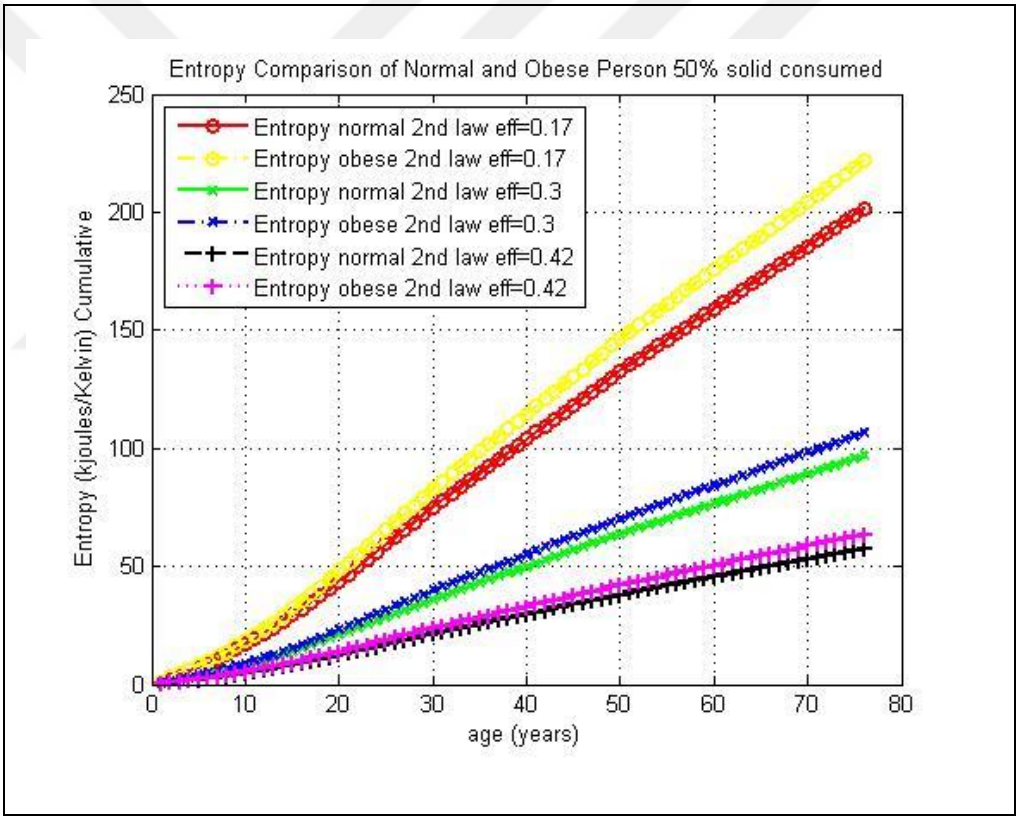


Figure 3.5. Simulations with 1.27 kJ/K (η_{II}=0.3) of entropy generation rate per year with the effect of more solid (50 per cent) consumption and the other factors, such as ultraviolet light and ions, in the masseter muscles- this figure shows that the cumulative entropy generation drops as the 2nd law efficiency rises and the same amount of entropy generated for an obese person five years earlier than for the normal individual

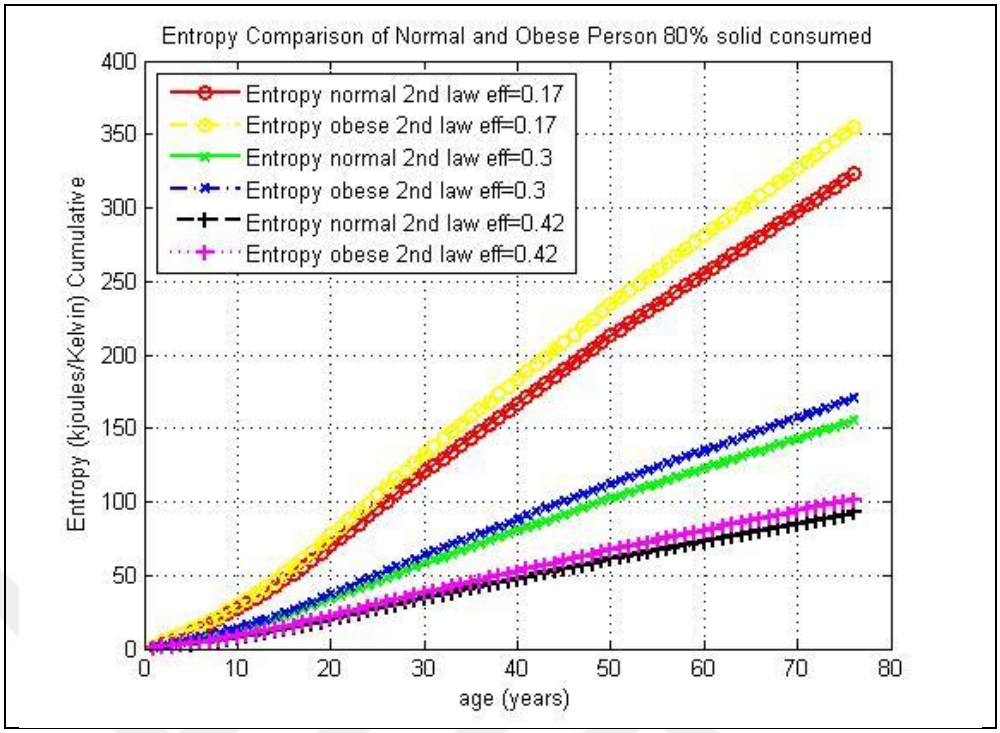


Figure 3.6. Simulations with 2.03 kJ/K (η_{II}=0.3) of entropy generation rate per year with the effect of more solid (80 per cent) consumption and the other factors, such as ultraviolet light and ions, in the masseter muscles- this figure shows that the cumulative entropy generation drops as the 2nd law efficiency rises and the same amount of entropy generated for an obese person five years earlier than for the normal individual

The data employed in this study were obtained with a model food – almond. The work loop (Figure 3.1) and the all the numerical values given in Table 3.1, therefore the lifespan entropy calculated are expected to change, if the study should be repeated with a different food. It is assumed that consuming 10 per cent (Figure 3.3) more food by the obese person does not affect the chewing behavior, and the entropy generation per unit mass of the food.

Since the mass of the food chewed by the obese person is higher (entropy generation per mass of the food) x (the mass of food) than that of a normal person. The jaw muscles are the motors of the masticatory apparatus. The masticatory efficiency, given as 69 per cent [121], plays an essential role on the entropy generation for chewing. The available energy for work that can be utilized by jaw movement creating the chewing action with participation of saliva which creates solution with almond particles. The concentration difference at this solution

as well as dissipation at jaw intersections result in exergy destruction as it is illustrated by Figure 3.2. Main process in the oral cavity is grinding, which involves friction, increasing disorder mainly affected by masticatory efficiency. The masticatory efficiency is only assumed to be 69 per cent because the work loop is obtained from jaw-displacement data provided by Hannam *et al*. [120]. All these processes increase the exergy destruction together with entropy generation. (See Figure 3.2) Given their role in producing forces and doing work during chewing, the musculoskeletal morphology of the masticatory apparatus plays a fundamental role in jaw-muscle and masticatory performance. In most primates usually one side of the jaw does the chewing work, while the other does the balancing work [130-132]. The model is built on the scenario that the subject chews the standard food, but in real life the entropy generation will increase as the work performed by the muscles increase, leading to faster destruction of the muscle.

There are studies in the literature reporting animal masseter muscle masses [133-135], but the masseter muscle mass of the people are not reported. The difficulty of making such a measurement with the live subjects might be the reason. In the literature entropy generation is reported per body mass (J/kg/K), whereas our analyses are based on the entropy generation by the total muscle in J/K, since the mass of the human masseter muscles are highly changeable and not reported in the literature.

The subject of the experiments apparently preferred chewing the almond on the right side of his jaw, this is the location referred to as ipsilateral, whereas the other, e.g., symmetrical, side of the jaw is referred to as contralateral. The thermodynamic analysis indicates that the work done by the contralateral muscles is more than the work done the ipsilateral muscle (Figure 3.1). The jaw elevator muscles contract on both sides of the jaw to contribute the chewing action by generating an unsymmetrical and complex pattern of movement to produce the bite force. The two phased work loop of Figure 3.1 shows that the ipsilateral muscle experiences contraction and stop in the cycle then continue the chewing action, while the contralateral muscles indicate a totally different chewing behavior. It is clearly indicated in the literature that the contralateral muscles, while contributing both to the generation of the bite force and trying to maintain the balance do more work [130-132].

In our model we use time average thermodynamic properties of the muscle. It is assumed in the entropy balance around the masseter muscle that all the entropy generated is flushed out

for steady system, making $\Delta s=0$ in Equations (3.8) and (3.12). In the muscle, generation of the metabolic heat results in entropy generation. Ageing is related with the increase of entropy in the body [7,22]. Although accumulation of the entropy within the muscle may be neglected for a single cycle of chewing the same assumption is not true for the lifelong repeated cycles. Therefore as time passes by, the agreed structure of the muscle cell becomes different from that of a younger person. The metabolic heat generation may be regarded among the major causes of this structural change. In such a case ageing may be related with the accumulation of the fraction of the entropy generated through the lifespan as ΔS within the muscle structure. While describing the ageing of the fibroblast stem cell system, Toussaint *et al*. [1] describes the evolution of seven morphotypes, each characterized with a stable level of entropy accumulation. Toussaint *et al.* [1] also related with the entropy accumulation in the cell with the structural changes accumulating in the DNA and other cellular components.

3.4. THERMODYNAMIC ANALYSIS OF THE SKELETAL MUSCLE SYSTEM

Mass of glucose consumed in the muscle was calculated after substituting 3.868 kJ/kmol for the Gibbs free energy of the apparent glycolysis reactions and 180.16 for the molar mass of glucose in equation (3.12) [106].

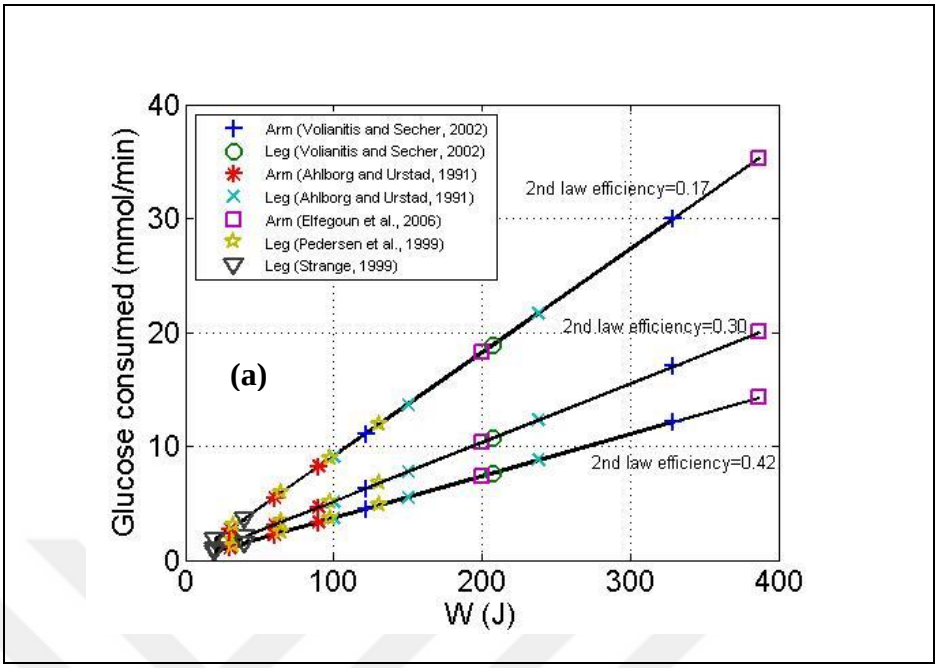
In the literature the first law efficiencies based on the experiments carried out with animals like frog and mouse are between 0.14 and 0.35 and the second law efficiencies are between 0.17 and 0.42 [65]. Work performance data are adapted from the literature (Table 3.4).

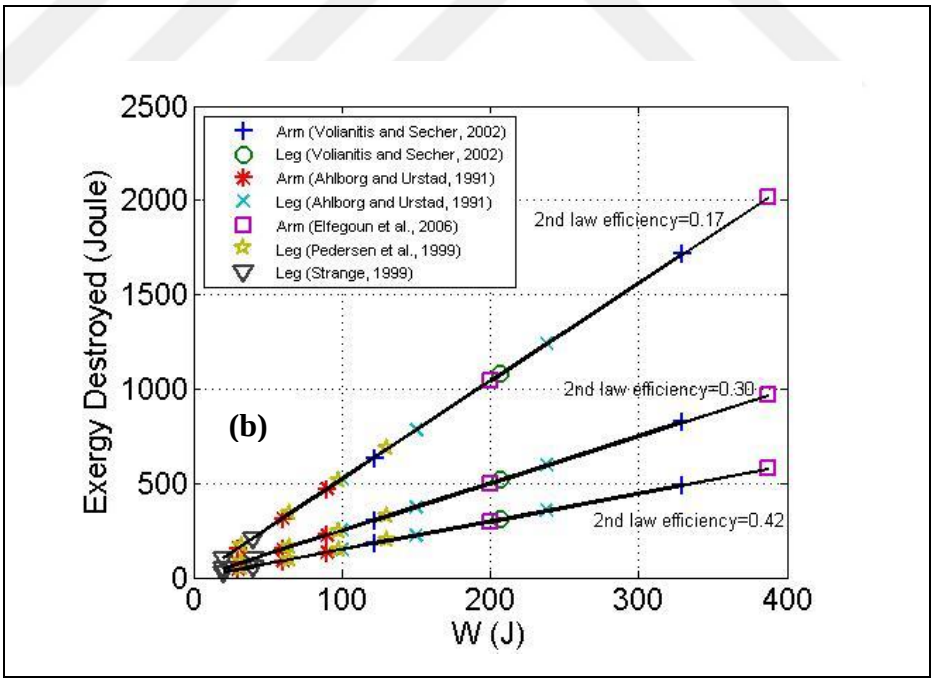
Table 3.4. Numerical values of the glucose consumption rate, exergy destroyed and the entropy generation in the muscles with the work performance efficiencies η_{II}=0.17, η_{II}=0.3 and η_{II}=0.42.

Work (J)	**η_{II}**	**Glucose consumed (mmol/min)**	**Exergy destroyed (J)**	**Entropy generation (J/K)**	**References**
122	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	11.13 6.308 4.505	636 305.5 182	2.134 1.025 0.610	Arm [136]
329	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	30.02 17.01 12.15	1715 823.8 490.8	5.755 2.764 1.647	Arm combined leg [136]
122	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	11.13 6.308 4.505	636 305.5 182	2.134 1.025 0.610	Arm cardiac output [136]
207	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	18.88 10.70 7.645	1079 518.3 308.8	3.621 1.739 1.036	Leg [136]
30	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	2.737 1.551 1.107	156.4 75.12 44.75	0.524 0.252 0.150	Arm exp1 [137]
60	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	5.474 3.102 2.215	312.8 150.2 89.51	1.049 0.504 0.300	Arm exp2 [137]
90	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	8.212 4.653 3.323	469.2 225.3 134.2	1.574 0.756 0.450	Arm exp3 [137]
100	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	9.124 5.170 3.693	521.3 250.4 149.1	1.749 0.840 0.500	Leg exp1 [137]
150	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	13.68 7.755 5.539	782 375.6 223.7	2.624 1.260 0.750	Leg exp2 [137]
238	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	21.71 12.30 8.790	1240 595.9 355	4.163 1.999 1.191	Leg exp3 [137]
387	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	35.31 20.01 14.29	2017 969.1 577.3	6.770 3.252 1.937	Arm combined leg cardiac output maximal [138]
200	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	18.24 10.34 7.386	1042 500.8 298.3	3.498 1.680 1.001	Arm combined leg cardiac output submaximal [138]

33	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	3.011 1.706 1.218	172 82.63 49.23	0.577 0.277 0.165	Leg exp1 [139]
65	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	5.931 3.360 2.400	338.8 162.7 96.97	1.137 0.546 0.325	Leg exp2 [139]
98	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	8.942 5.067 3.619	510.9 245.4 146.2	1.714 0.823 0.490	Leg exp3 [139]
131	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	11.95 6.773 4.838	682.9 328 195.4	2.291 1.100 0.655	Leg exp4 [139]
20	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	1.824 1.034 0.738	104.2 50.08 29.83	0.349 0.168 0.100	Leg cardiac output exp1 [140]
40	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	3.649 2.068 1.477	208.5 100.1 59.67	0.699 0.336 0.200	Leg cardiac output exp2 [140]
20	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	1.824 1.034 0.738	104.2 50.08 29.83	0.349 0.168 0.100	Leg exp1 [140]
40	η_{II}=0.17 η_{II}=0.30 η_{II}=0.42	3.649 2.068 1.477	208.5 100.1 59.67	0.699 0.336 0.200	Leg exp2 [140]

Numerical values of the work performed, glucose consumed, exergy destroyed and entropy generated are calculated for the second law efficiency range given in the literature as η_{II}=0.17, η_{II}=0.3 and 0.42 (Table 3.4). While doing the same amount of muscle work, decreases are observed in glucose consumption, exergy destruction and entropy generation with the increasing 2nd law efficiency (Figure 3.7) implying that as the efficiency increases, a larger fraction of the chemical exergy of glucose is converted into work. In Figure 3.7(a) glucose consumption at W=0 describes its use either for the basal metabolism or the depletion of the cellular reserve, with muscle fat consumption. The slope of this line is the inverse second law efficiency, e.g. $slope = 1/\eta_{II}$. The plots of Figure 3.7 are linear, since the data were obtained with healthy young subjects only. If we attempt to utilize the data obtained with the elderly or unhealthy people we would not be able to obtain linear relations. Figures 3.7(b) and 3.7(c) show that the exergy loss and entropy generation in the muscles increase as a response to the attempts to higher work production in the aging muscle systems.


Arm (Volianitis and Secher, 2002)
Leg (Volianitis and Secher, 2002)
Arm (Ahlborg and Urstad, 1991)
Leg (Ahlborg and Urstad, 1991)
Arm (Elfegoun et al., 2006)
Leg (Pedersen et al., 1999)
Leg (Strange, 1999)
2nd law efficiency=0.17
2nd law efficiency=0.30
2nd law efficiency=0.42
(a)
Glucose consumed (mmol/min)
W (J)
0
10
20
30
40
0
100
200
300
400


Arm (Volianitis and Secher, 2002)
Leg (Volianitis and Secher, 2002)
Arm (Ahlborg and Urstad, 1991)
Leg (Ahlborg and Urstad, 1991)
Arm (Elfegoun et al., 2006)
Leg (Pedersen et al., 1999)
Leg (Strange, 1999)
2nd law efficiency=0.17
2nd law efficiency=0.30
2nd law efficiency=0.42
(b)
Exergy Destroyed (Joule)
W (J)
0
500
1000
1500
2000
2500
0
100
200
300
400

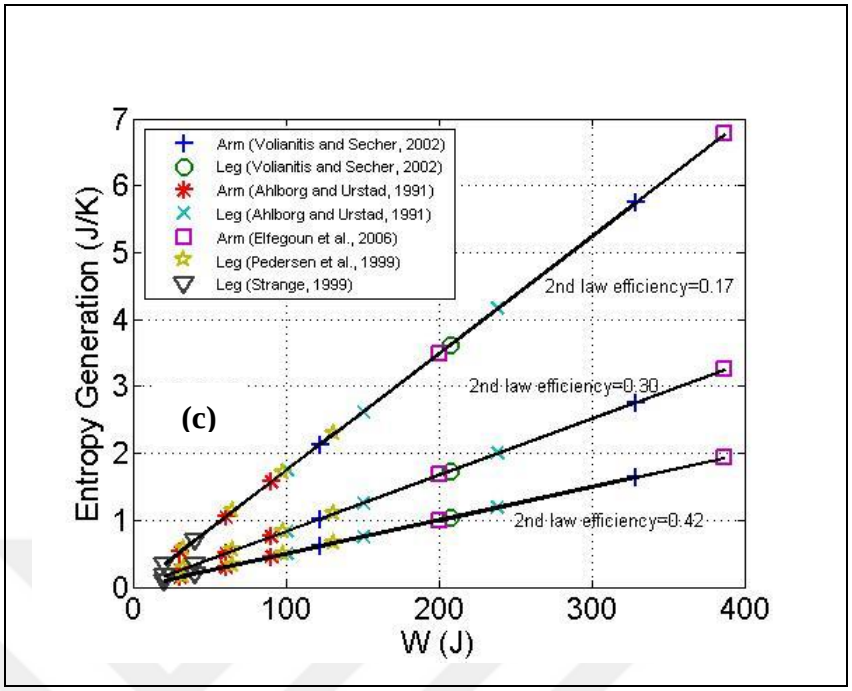
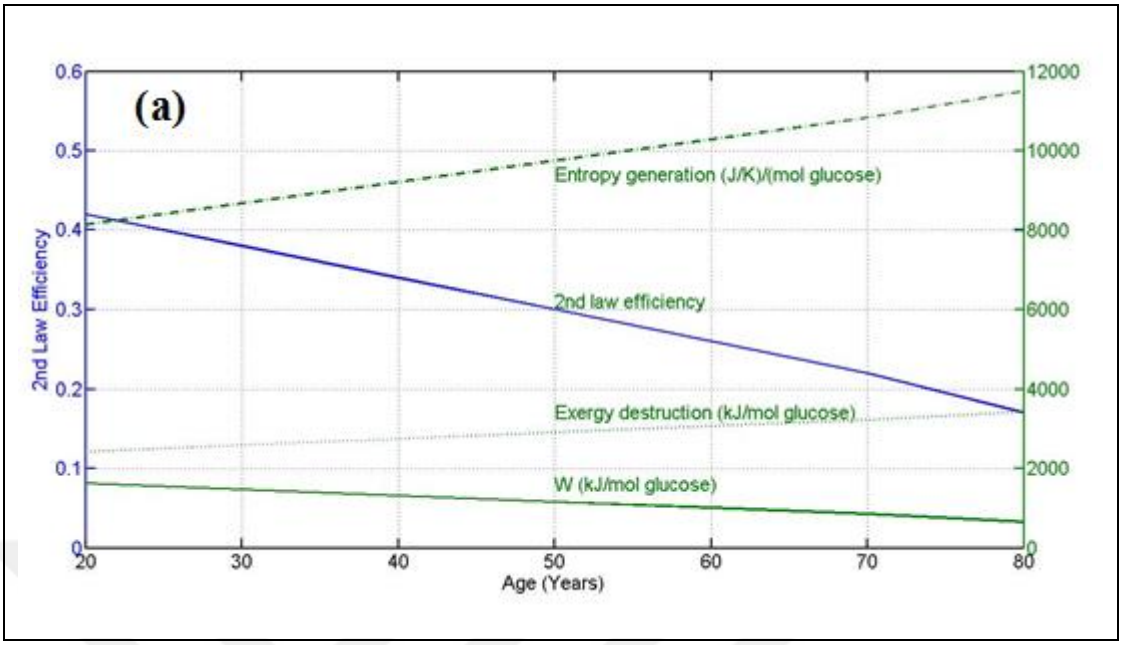
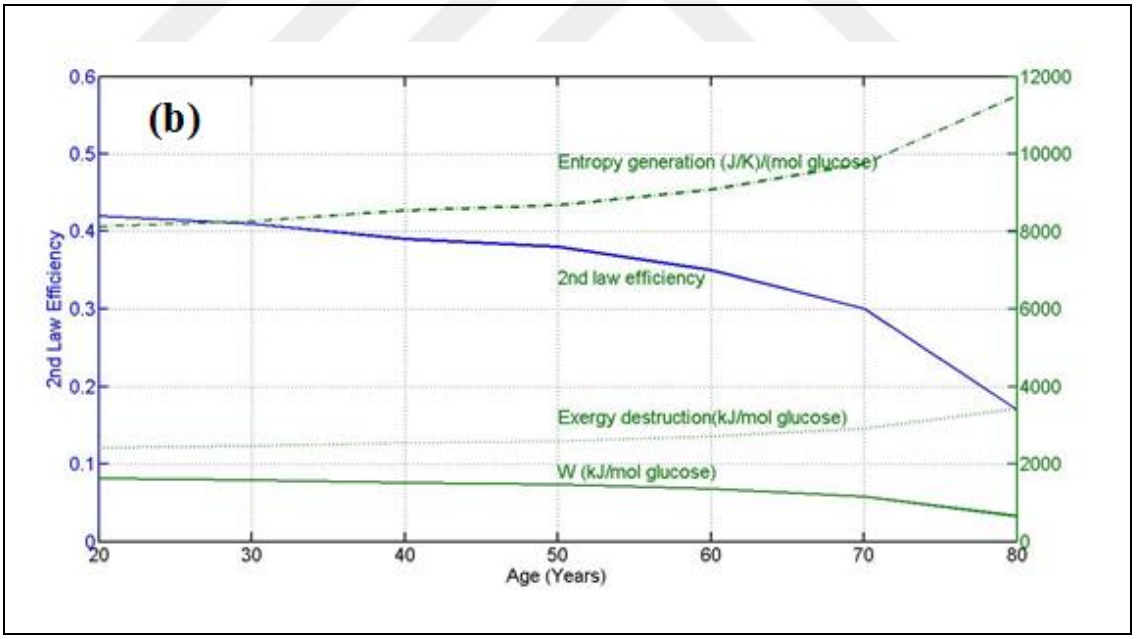


Figure 3.7. (a) Glucose consumption, (b) Exergy destruction and (c) Entropy generation rates during muscle work performance when the 2nd law efficiencies are η_{II}=0.17, η_{II}=0.3 and η_{II}=0.42

Recent studies show that the elder adults utilize more energy than their younger counter parts to do the same activity due to their lower muscle work performance efficiency [83,141-143,77]. Figures 3.7(b) and 3.7(c) show that this observation may be caused by higher exergy loss and entropy generation at lower second law muscle work performance efficiencies [144]. The results given in Figures 3.7(b) and 3.7(c) may imply that the young individuals who improve their muscle work efficiency by exercise may experience less exergy loss and less entropy generation while performing muscle work.

The aging process of two different people following path 1 and 2 are assessed in Figures 3.8(a) and 3.8(b). In the examples given in Figure 3.8 thermodynamic analysis starts at the age of 20, when the 2nd law efficiency is 0.42 and at the age of 80 the 2nd law efficiency becomes 0.17; Figure 3.8(c) visualizes the allocation of the exergy driven from one mole of glucose for each path of aging described here. At the age of 60, a person who is following to path 2 of aging do more work compared to a person who is aging by following path 1. His muscles have been subject to smaller amounts of exergy destruction and generated less entropy, in comparison with the person who is following path 2.


(a)
Entropy generation (J/K)/(mol glucose)
2nd law efficiency
Exergy destruction (kJ/mol glucose)
W (kJ/mol glucose)
2nd Law Efficiency
Age (Years)
0.6
0.5
0.4
0.3
0.2
0.1
0
12000
10000
8000
6000
4000
2000
0
20
30
40
50
60
70
80


(b)
Entropy generation (J/K)/(mol glucose)
2nd law efficiency
Exergy destruction(kJ/mol glucose)
W (kJ/mol glucose)
2nd Law Efficiency
Age (Years)
0.6
0.5
0.4
0.3
0.2
0.1
0
12000
10000
8000
6000
4000
2000
0
20
30
40
50
60
70
80

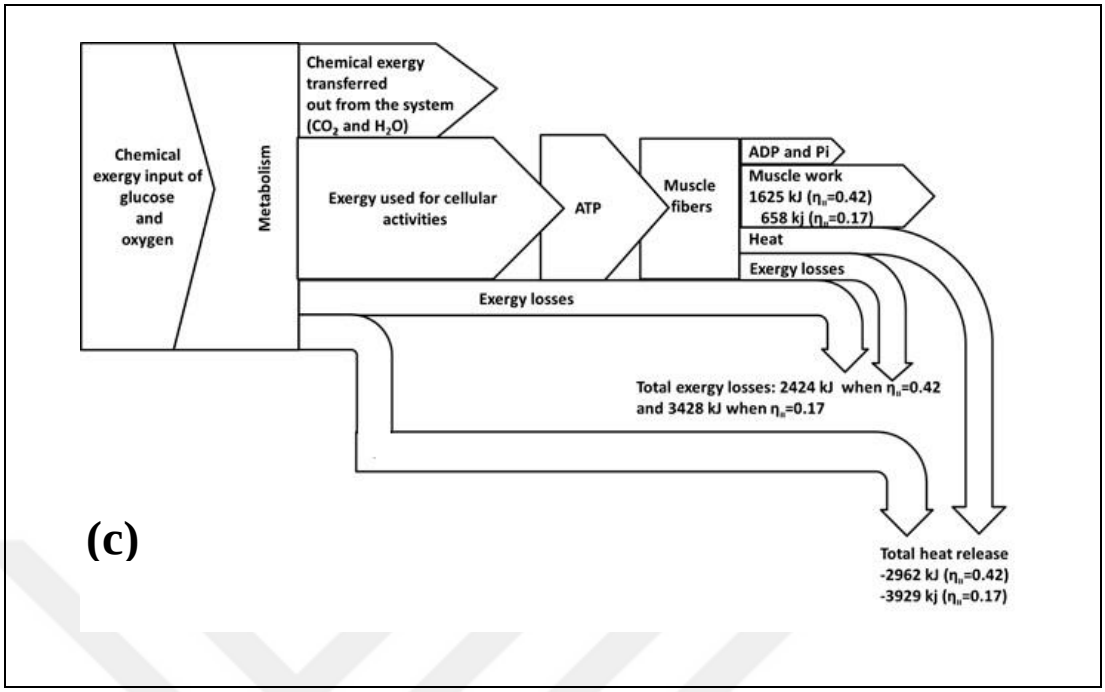


Figure 3.8. Work performance, exergy destruction and entropy generation associated with one mole of glucose consumption in the cases when the second law muscle work efficiency decreases (a) Linearly and (b) Gradually until the age of 70 and then faster. (c) Muscle work production and exergy destruction in exercise during utilization of one mole of glucose when the second law efficiencies are 0.42 and 0.17

Decline of either the metabolic exergy efficiency, η_{m1}, or the mechanical work performance efficiency, η_{m2}, causes a drop in the second law muscle work performance efficiency. Kiriazis and Gibbs, while investigating the effect of aging on the work output and efficiency of rat papillary muscle reported that the mechanical work performance efficiency, η_{m2}, was not influenced by aging, it was actually the metabolic exergy efficiency, η_{m1}, what decreased by aging [145]. Figueiredo *et al.* [146] argue that the age-related increase in the levels of oxidative stress and damage on the mitochondrial biomolecules becomes progressively more apparent with aging . When the cellular capacity for the repair is not sufficient, this increased damage leads to accumulation of the dysfunctional proteins, impaired membrane integrity and increased levels of the mutant mitochondrial DNA. These damaged cellular compounds proliferate irreversibly through mitochondrial and cellular divisions and causes reduction of the intact mitochondria and the maximal mitochondrial function, and the maximal energetic

capacity of skeletal muscle fibers. This progressive loss of mitochondrial redundancy may not limit its capacity to supply the cellular energetic demands at basal metabolic conditions, but might limit the functionality of myofibrils during situations with higher energetic requirements. Observations of Figueiredo *et al*. [146]imply that the aging damage occurring to the metabolic pathways may not be confined to metabolism, but eventually propagate to the mechanical work performing sites of the muscle . It has been suggested that the muscle changes in persons start in the fourth decade of life and cause weakness and disabilities [147]. Associated changes in body composition establishes the basis of many metabolic disorders. Decreases in the synthesis rates of several muscle proteins, specifically of myosin heavy chain and mitochondrial proteins, occur with age. Reduction in mitochondrial biogenesis and ATP production leads to decreases in mitochondrial DNA and messenger RNA. Reduced ATP production could be the basis of reduced muscle protein turnover, which requires energy [147]. Kiriazis and Gibbs, observed that the efficiency loss was confined to the metabolic pathways only, probably because of the short lifespan of the rats, which did not have the opportunity of spreading the damage to the mechanical work performing sites of the muscle. Exercise enhances muscle protein synthesis and mitochondrial biogenesis [145,147].

3.5. THERMODYNAMIC ANALYSIS OF THE CARDIAC HEART MUSCLE SYSTEM

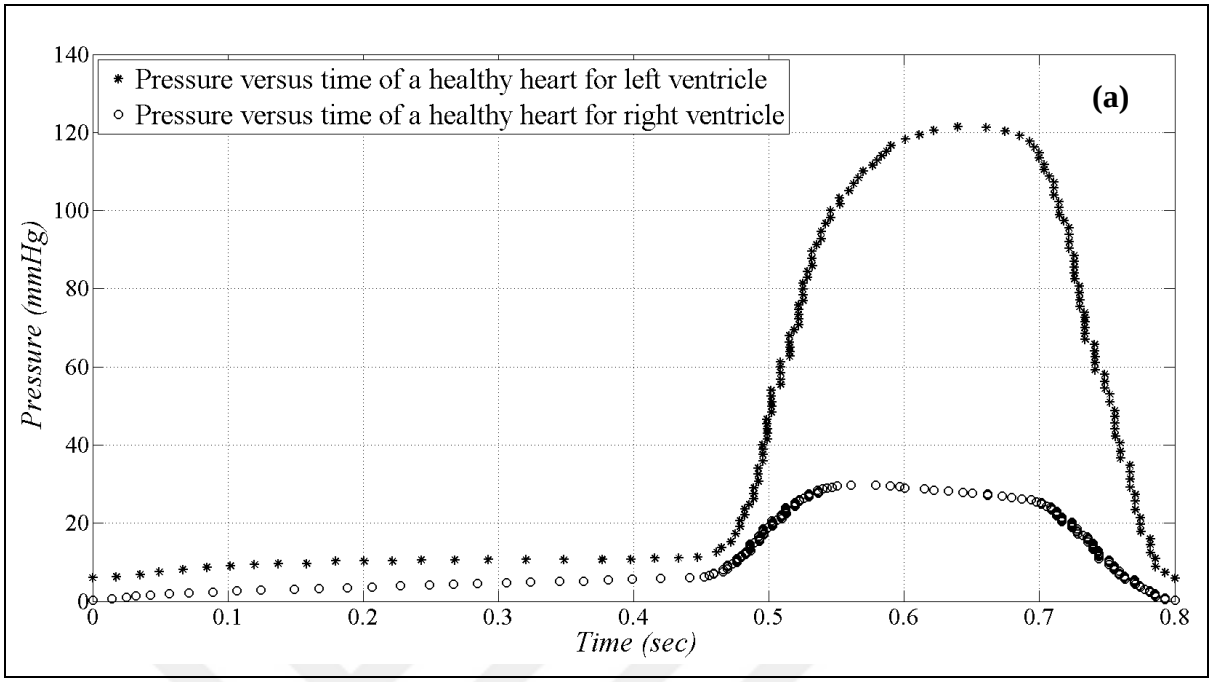
A heart is an open system with moving boundaries, pressure and the volume of left and right ventricles change throughout the heartbeat. Left ventricle pressure remains at 10 mmHg for the first part (i.e., 0 to 0.4 sec.) of the cardiac cycle (Figure 3.9(a)) while the blood flows into the left ventricle doubles its size (Figure 3.9(b)). When heart muscles contract, left ventricle pressure drastically rises to above 120 mmHg to force the blood from left ventricle chamber into the aorta after opening the aortic valve (Figure 3.9(a)). Meanwhile, the left ventricle volume exhibits a sharp decrease, because nearly half of the blood in the left ventricle is sent to the person body through the aorta (Figure 3.9(b)). In physiology Cardiac output (CO) is used to evaluate the performance of the heart:

$$CO = HRxSV \tag{3.13}$$

where, HR is the heartbeat rate and SV is the stroke volume. In the present study, CO of the healthy heart was calculated from the Wiggers diagram as 5.15 L/min when HR and SV were 75 beat/min and 73.6 mL/min, respectively. The mean arterial pressure (MAP) is the weighted average arterial pressure calculated as over one cardiac cycle and used together with CO to evaluate the efficiency of the pumping pressure of the heart:

$$MAP = \frac{1}{3}\left(P_{sys} + 2P_{dias}\right) \tag{3.14}$$

where, P_{sys} and P_{dias} are the systole and diastole pressures, respectively. For the present case P_{sys} and P_{dias} and MAP were 121.5, 76.88 and 91.75 mmHg, respectively for the healthy heart. The magnitude of the MAP indicates how well the aortic valve function when pressure reaches to 80 mmHg. Once pressure and volume variation in left and right ventricles were obtained, pressure can be plotted against volume to examine the relation between pressure and volume for a single cardiac cycle as illustrated in Figure 3.11(a). Variation of pressure along with volume implies boundary work; therefore, the enclosed areas in Figure 3.11(a) were the work done by the left and right ventricle walls of the heart - work done by the left and right ventricles were calculated as 0.9607 and 0.1969 J, respectively. This indicates that the heart does approximately 5 times more work to move blood from left ventricle to the person body, compared to that of the work done by the right ventricle, which sends CO_2 rich blood to the pulmonary circulation.



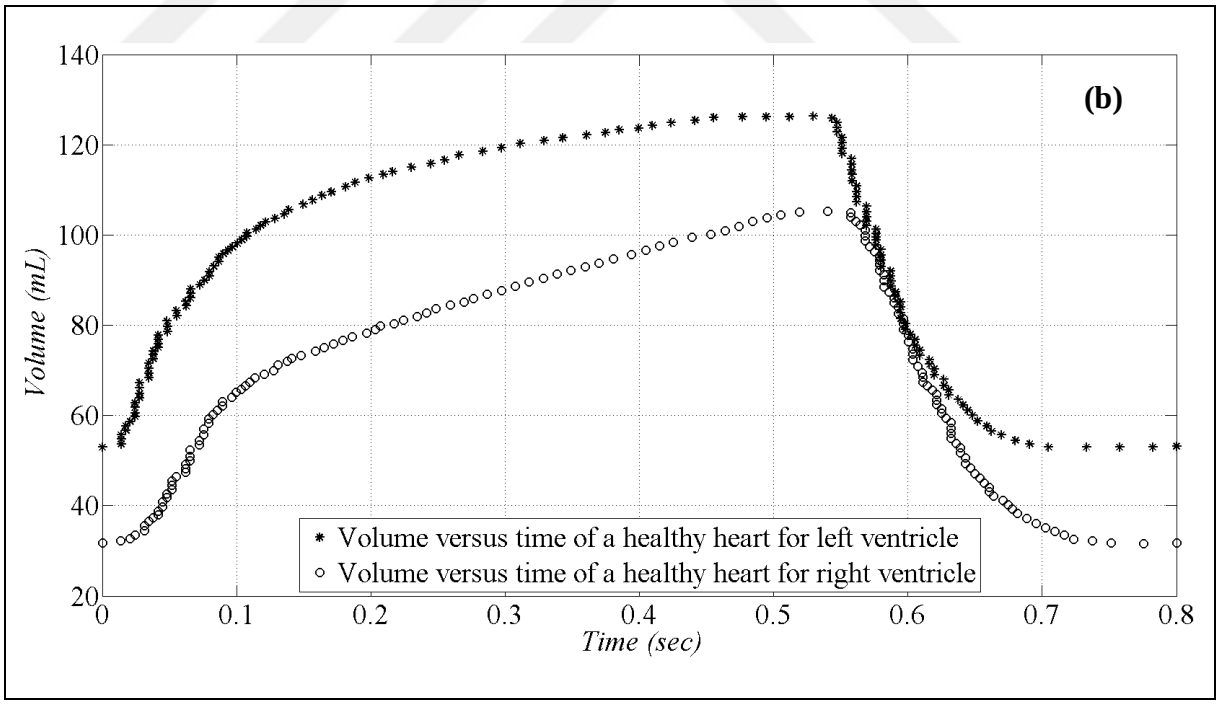
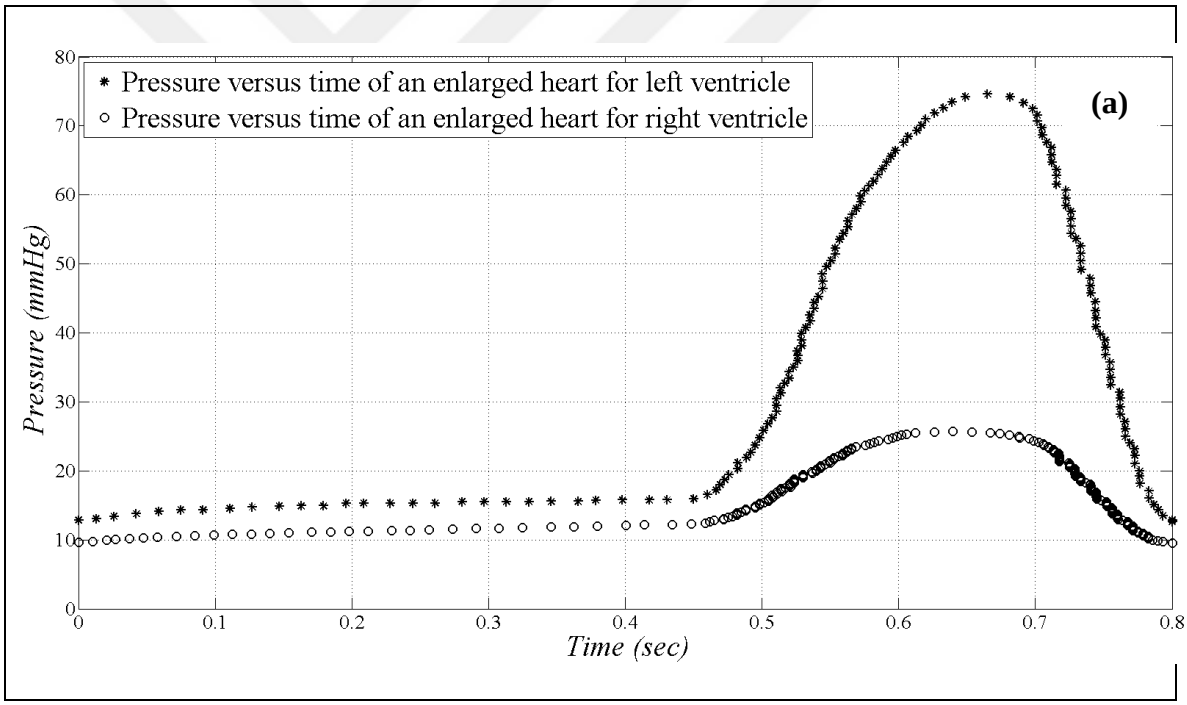


Figure 3.9. (a) Pressure variation of the healthy heart in the left and right ventricles based on Wiggers diagram and (b) Volume variation of a healthy heart in the left and right ventricles based on Wiggers diagram

Pressure and volume changes in the left and right ventricles of the enlarged heart are plotted in Figs 3.10a and 3.10b, respectively. Pressure in the left ventricle of an enlarged heart was approximately half of the pressure in the left ventricle of the healthy heart; implying that an enlarged heart cannot build as high pressure in the left ventricle as the healthy heart. The lower pressure in the left ventricle disturb the operation of the aortic valve and change the magnitudes of CO and MAP. However, the right ventricle pressure was 15 per cent less in the enlarged heart. Effect of decrease in the pressure of the left and the right ventricles was accompanied with increase in the left and right ventricle volumes, e.g., the left and right ventricle volumes increased nearly 50 per cent and 15 per cent compared to those of the healthy heart, respectively.



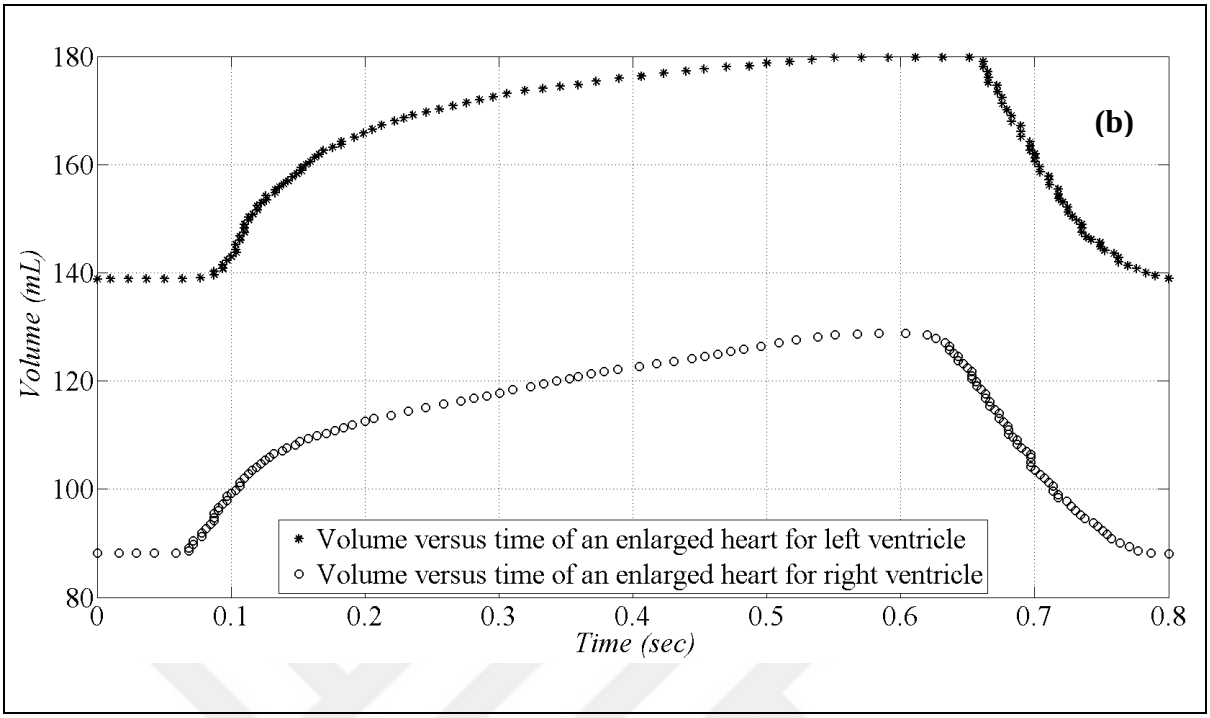


Figure 3.10. (a) Pressure variation of the enlarged heart in the left and right ventricles and (b) Volume variation of the enlarged heart in the left and right ventricles

Change of pressure along with volume in the enlarged heart illustrated quite different curves than those of the healthy heart as shown in Figure 3.11. Boundary works for left and right ventricles were calculated by taking the numerical integral of the enclosed areas as 0.2812 and 0.0723 J, respectively. Furthermore, nearly 50 per cent decrease in CO indicate that the internal organs and tissues would not receive sufficient amount of blood in the body of the person of the enlarged heart; 30 per cent drop in MAP of the same person may imply an operation problem in the aortic valve since this valve is activated only when a certain pressure is applied on upstream side.

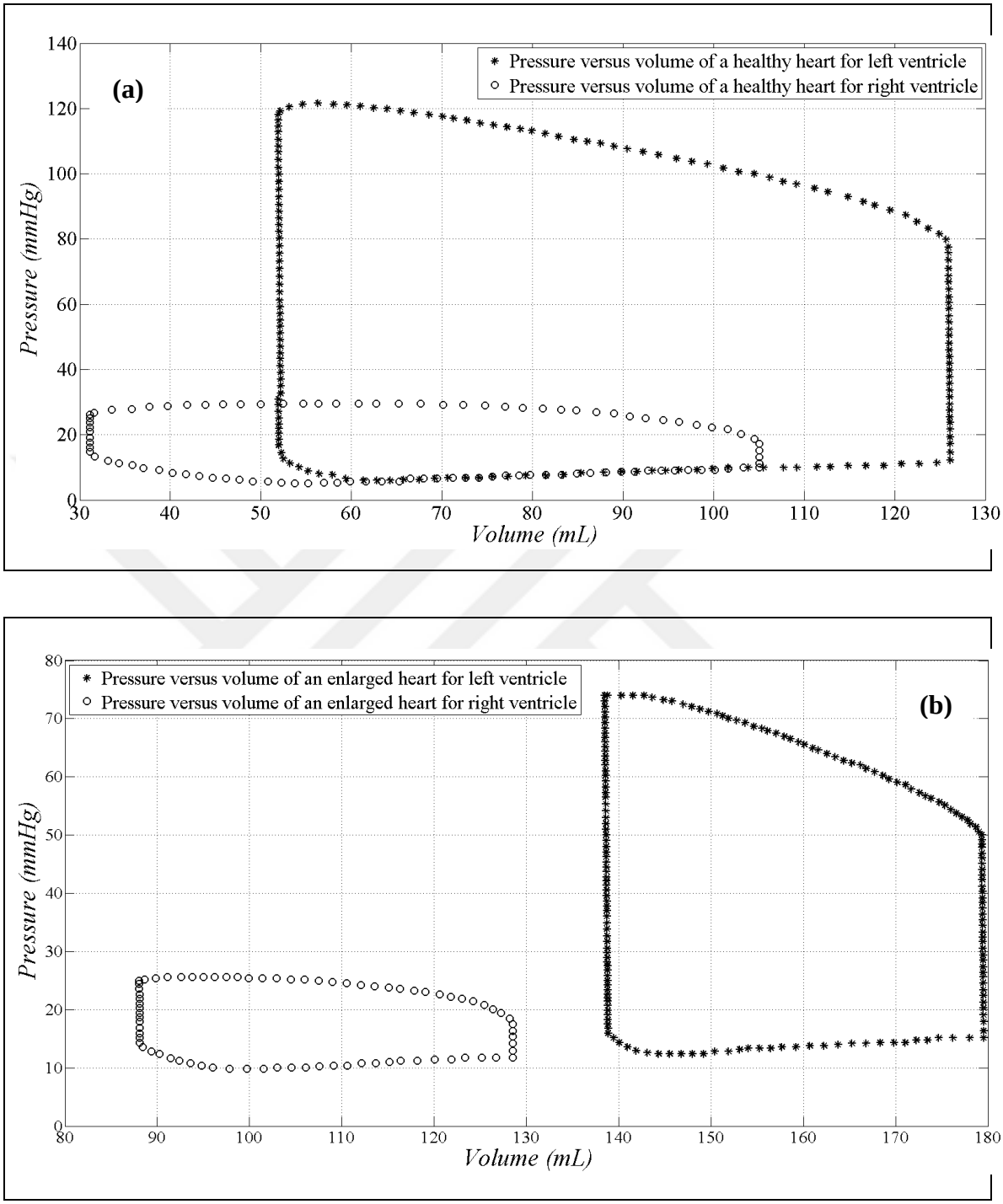


Figure 3.11. (a) Wigger diagrams for the healthy and (b) Enlarged hearts

Table 3.5 shows the variation of the glucose consumption rate, exergy destruction rate and the entropy generation rate with the second law efficiency in a single beat in the cardiac muscles of the health person at the heartbeat rate of 75 beat/min (one beat = 0.8 s, left ventricular beat work = 9.607 x 10^{-4} kJ). Tables 3.6 and 3.7 show the glucose consumption

and the exergy destroyed and the entropy generated in the same heart. Similar analysis is presented for the enlarged heart in Tables 3.8 and 3.9 when the left ventricular beat work was 2.812 x 10^{-4} kJ/beat and that of the right ventricle heart muscles was 7.23 x 10^{-5} kJ/beat. Tables 3.5-3.9 and Figure 3.12 show that in the healthy and enlarged heart as the efficiency increases, a larger fraction of the chemical exergy of glucose is converted into work. Therefore, the amount of the glucose required to achieve the same work decreases as the efficiency increases. When the heart rate increases from 60 to 140 beat/min, cycle time decreases from 1 s to 0.43 s, as a result, glucose consumption and absorption from the blood increase (Figure 3.12) and heart can do more work in a minute.

Table 3.5. Variation of the glucose consumption, exergy destruction and the entropy generation rates with the second law efficiency in the left ventricle muscles of the person with the healthy heart at heartbeat rate of 75 beats/min (one beat 0.8 second and healthy rest subject left ventricular one beat work= 9.607 x 10^{-4} kJ).

η_{II}	$m_{glucose}$ (mol/beat)	$m_{glucose}$ (mmol/min)	Glucose concentration in the blood (mmol/L)	$Ex_{destroyed,\ muscle}$ (kJ/beat)	$S_{gen,muscle}$ (kJ/K)/beat
0.17	$1.46x10^{-6}$	$1.09x10^{-1}$	$4.38x10^{-1}$	$5.01x10^{-3}$	$1.68x10^{-5}$
0.22	$1.13x10^{-6}$	$8.46x10^{-2}$	$3.38x10^{-1}$	$3.64x10^{-3}$	$1.22x10^{-5}$
0.26	$9.55x10^{-7}$	$7.16x10^{-2}$	$2.86x10^{-1}$	$2.92x10^{-3}$	$9.83x10^{-6}$
0.3	$8.27x10^{-7}$	$6.21x10^{-2}$	$2.48x10^{-1}$	$2.40x10^{-3}$	$8.07x10^{-6}$
0.34	$7.30x10^{-7}$	$5.47x10^{-2}$	$2.19x10^{-1}$	$2.01x10^{-3}$	$6.73x10^{-6}$
0.4	$6.21x10^{-7}$	$4.65x10^{-2}$	$1.77x10^{-1}$	$1.55x10^{-3}$	$5.21x10^{-6}$
0.42	$5.91x10^{-7}$	$4.43x10^{-2}$	$1.29x10^{-1}$	$1.43x10^{-3}$	$4.81x10^{-6}$

Table 3.6. Variation of the glucose consumption rate in the person heart muscles with the different second law efficiencies at a heart rate of 60 and 140 beats per min in the left ventricle (healthy rest subject left ventricular one beat work= 9.607 x 10^{-4} kJ).

η_{II}	**Heart rate (beat/min)**	**Cycle time (sec)**	**Glucose consumed (mmol/min)**	**Glucose absorbed from the blood flow (mmol/liter)**
0.17	60	1	$8.76x10^{-2}$	0.3506
0.17	140	0.43	$2.04x10^{-1}$	0.8181
0.42	60	1	$3.55x10^{-2}$	0.1419
0.42	140	0.43	$8.27x10^{-2}$	0.3311

Table 3.7. Variation of the glucose consumption, exergy destruction and the entropy generation rates with the second law efficiency in the right ventricle muscles of the person with the healthy heart at heart rate of 75 beats/min (one beat 0.8 second and healthy rest subject right ventricular one beat work = 1.969 x 10^{-4} kJ).

η_{II}	$m_{glucose}$ **(mol/beat)**	$m_{glucose}$ **(mmol/ min)**	**Glucose concentration in the blood (mmol/L)**	$Ex_{destroyed,muscle}$ **(kJ/beat)**	$S_{gen,muscle}$ **(kJ/K)/beat**
0.17	$2.99x10^{-7}$	$2.24x10^{-2}$	$8.98x10^{-2}$	$1.02x10^{-3}$	$3.44x10^{-6}$
0.22	$2.31x10^{-7}$	$1.73x10^{-2}$	$6.94x10^{-2}$	$7.46x10^{-4}$	$2.50x10^{-6}$
0.26	$1.96x10^{-7}$	$1.46x10^{-2}$	$5.87x10^{-2}$	$6.00x10^{-4}$	$2.01x10^{-6}$
0.3	$1.69x10^{-7}$	$1.27x10^{-2}$	$5.09x10^{-2}$	$4.93x10^{-4}$	$1.65x10^{-6}$
0.34	$1.49x10^{-7}$	$1.12x10^{-2}$	$4.49x10^{-2}$	$4.10x10^{-4}$	$1.38x10^{-6}$
0.4	$1.27x10^{-7}$	$9.54x10^{-3}$	$3.81x10^{-2}$	$3.18x10^{-4}$	$1.07x10^{-6}$
0.42	$1.21x10^{-7}$	$9.09x10^{-3}$	$3.63x10^{-2}$	$2.93x10^{-4}$	$9.85x10^{-7}$

Table 3.8. Variation of the glucose consumption, exergy destruction and the entropy generation rates with the second law efficiency in the left ventricle muscles of the person with the enlarged heart at heart rate of 75 beats/min (one beat 0.8 second and enlarged rest subject left ventricular one beat work = 2.812 x 10^{-4} kJ).

η_{II}	$m_{glucose}$ (mol/beat)	$m_{glucose}$ (mmol/min)	Glucose concentration in the blood (mmol/L)	$Ex_{destroyed,muscle}$ (kJ/beat)	$S_{gen,muscle}$ (kJ/K)/beat
0.17	$4.27x10^{-7}$	$3.20x10^{-2}$	$1.28x10^{-1}$	$1.46x10^{-3}$	$4.92x10^{-6}$
0.22	$3.30x10^{-7}$	$2.47x10^{-2}$	$9.91x10^{-2}$	$1.06x10^{-3}$	$3.57x10^{-6}$
0.26	$2.79x10^{-7}$	$2.09x10^{-2}$	$8.38x10^{-2}$	$8.57x10^{-4}$	$2.87x10^{-6}$
0.3	$2.42x10^{-7}$	$1.81x10^{-2}$	$7.26x10^{-2}$	$7.04x10^{-4}$	$2.36x10^{-6}$
0.34	$2.13x10^{-7}$	$1.60x10^{-2}$	$6.41x10^{-2}$	$5.86x10^{-4}$	$1.96x10^{-6}$
0.4	$1.81x10^{-7}$	$1.36x10^{-2}$	$5.45x10^{-2}$	$4.55x10^{-4}$	$1.52x10^{-6}$
0.42	$1.73x10^{-7}$	$1.29x10^{-2}$	$5.19x10^{-2}$	$4.19x10^{-4}$	$1.40x10^{-6}$

Table 3.9. Variation of the glucose consumption, exergy destruction and the entropy generation rates with the second law efficiency in the right ventricle muscles of the person with the enlarged heart at heart rate of 75 beats/min (cycle time = 0.8 s, left ventricular resting beat work/cycle = 7.23 x 10^{-5} kJ).

η_{II}	$m_{glucose}$ (mol/beat)	$m_{glucose}$ (mmol/min)	Glucose concentration in the blood (mmol/L)	$Ex_{destroyed,muscle}$ (kJ/beat)	$S_{gen,muscle}$ (kJ/K)/beat
0.17	$1.09x10^{-7}$	$8.24x10^{-3}$	$3.29x10^{-2}$	$3.76x10^{-4}$	$1.26x10^{-6}$
0.22	$8.49x10^{-8}$	$6.37x10^{-3}$	$2.54x10^{-2}$	$2.74x10^{-4}$	$9.20x10^{-7}$
0.26	$7.18x10^{-8}$	$5.39x10^{-3}$	$2.15x10^{-2}$	$2.20x10^{-4}$	$7.39x10^{-7}$
0.3	$6.23x10^{-8}$	$4.67x10^{-3}$	$1.86x10^{-2}$	$1.81x10^{-4}$	$6.07x10^{-7}$
0.34	$5.49x10^{-8}$	$4.12x10^{-3}$	$1.64x10^{-2}$	$1.51x10^{-4}$	$5.06x10^{-7}$
0.4	$4.67x10^{-8}$	$3.50x10^{-3}$	$1.40x10^{-2}$	$1.17x10^{-4}$	$3.92x10^{-7}$
0.42	$4.45x10^{-8}$	$3.33x10^{-3}$	$1.33x10^{-2}$	$1.07x10^{-4}$	$3.62x10^{-7}$

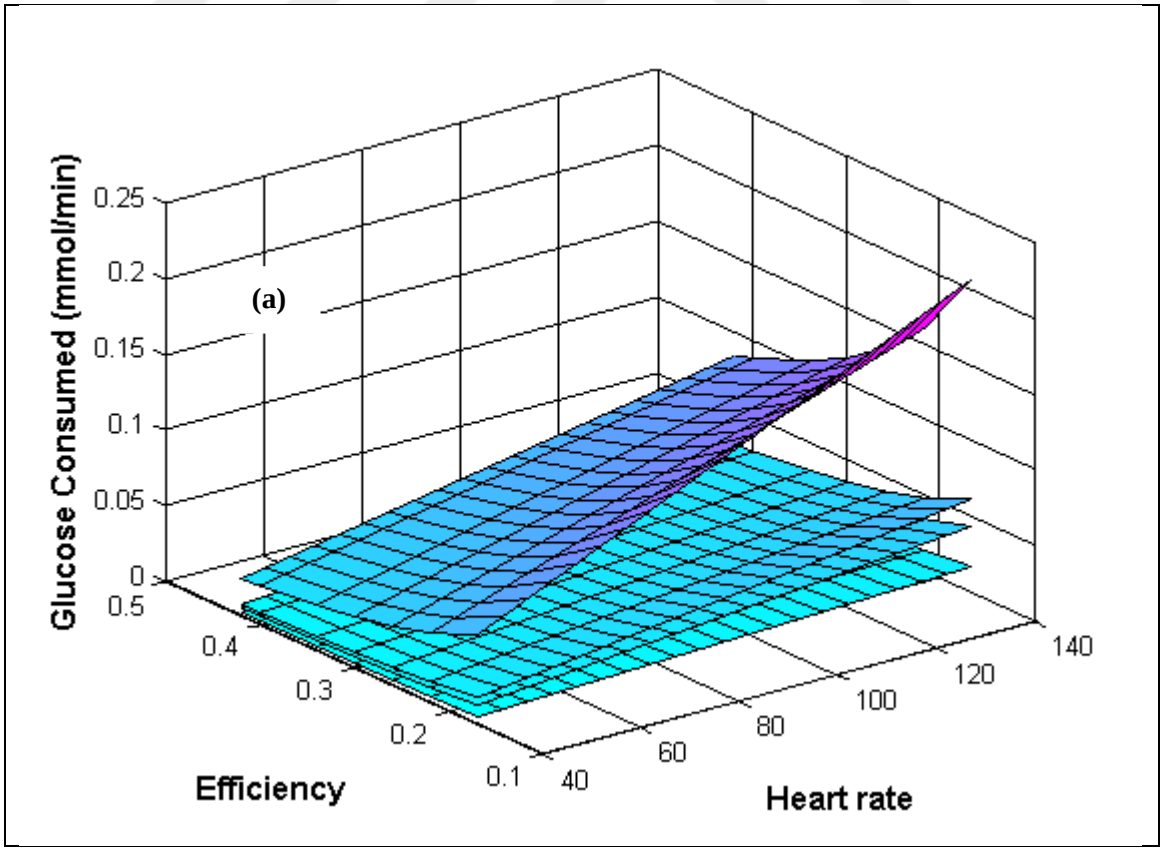
It was observed in Figure 3.11 that the left and the right ventricles of the healthy heart do approximately 3.5 and 2.7 times more work, respectively, than their counterparts in the enlarged heart. Such a major change in the work performance may be observed because of several reasons. The limiting cases may include:

i) Coronary arteries may deliver less nutrients, while the heart rate, muscle work efficiency, exergy destruction and entropy generation rates remain the same.

ii) Coronary arteries may deliver the same amounts of nutrients, while the heart rate remains the same, but muscle work efficiency decreases causing increase in exergy destruction and entropy generation rates. The decrease of the muscle work efficiency may be caused by the decline of either the metabolic efficiency or the mechanical efficiency of the cardiac muscles or both as depicted in Fig 1.1.

These limiting cases may arise at the onset of the health problem, but later cause propagates to affect all of the factors stated here, e.g., a problem starting by metabolic inefficiency will inevitably extend to cause mechanical efficiency and then cause changes in the heart rate, muscle work efficiency, exergy destruction and entropy generation rates.

Behavior of the left and the right ventricles are described in Figure 3.12 for both the healthy and the enlarged hearts. The enlarged heart could use considerably less glucose and performed less work at the same heartbeat, and generated less entropy in comparison with the healthy heart at the same efficiency. Equation 1.8 implies that entropy generation increases with the substrate utilization; therefore, the right ventricles of the people with healthy heart generated more than 2.5 times of the entropy than those of the people with enlarged heart. This ratio was 3.4 when the entropy was discussed for the left ventricle. When the second law efficiency was 0.40, glucose consumption increased linearly with the heartbeat rate; on the other hand, when the efficiency was low, glucose consumption increased more rapidly with the heart rate than what is described by the linear relation.



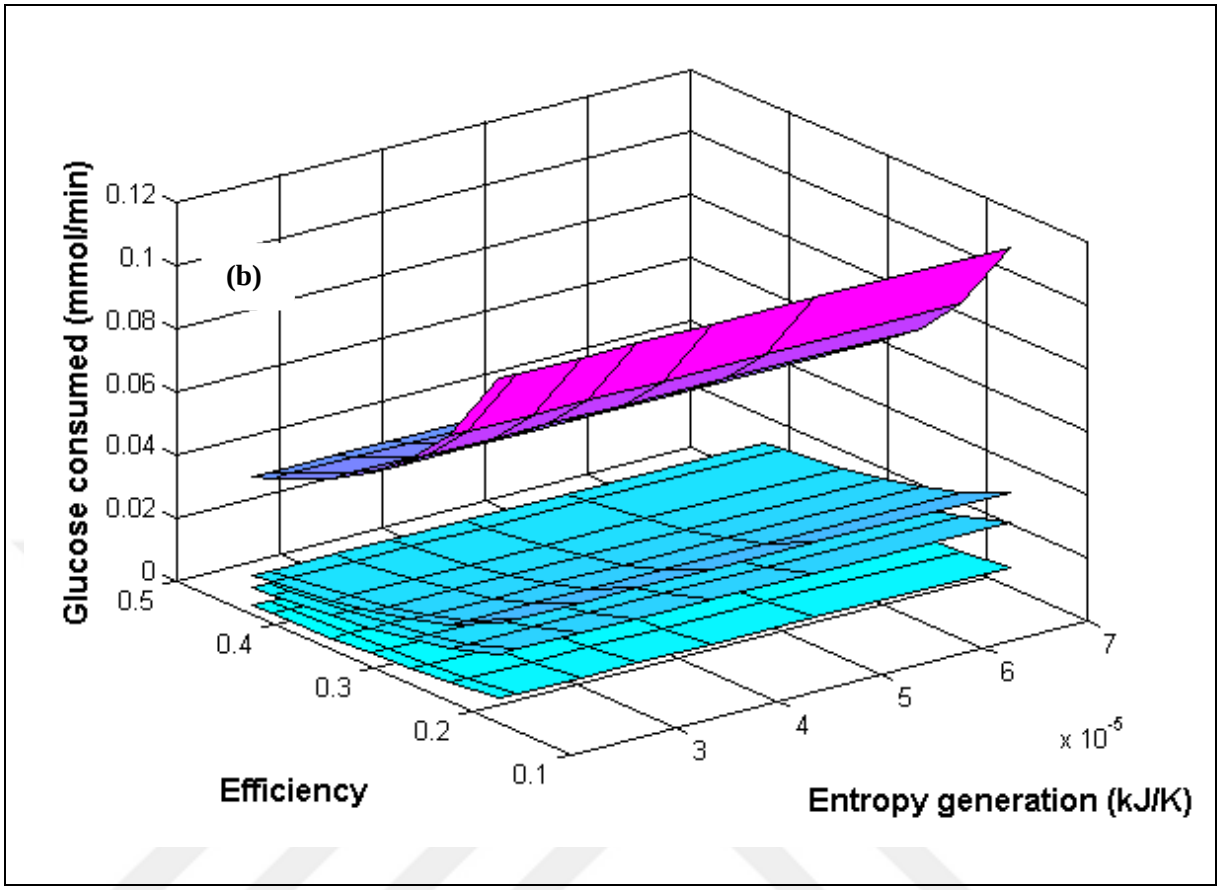


Figure 3.12. Change of glucose consumption rate (a) With efficiency and heartbeat rate and (b) With efficiency and entropy generation rate in the healthy and enlarged left and right ventricles (cycle time of is 0.8 sec)

Glucose consumption rate increases with the heart rate (Figure 3.12(a)); Figure 3.12(b) is plotted for a constant heartbeat rate of 75 beats/min; therefore, the maximum glucose consumption is lower in Figure 3.12(b) than that of Figure 3.12(a).

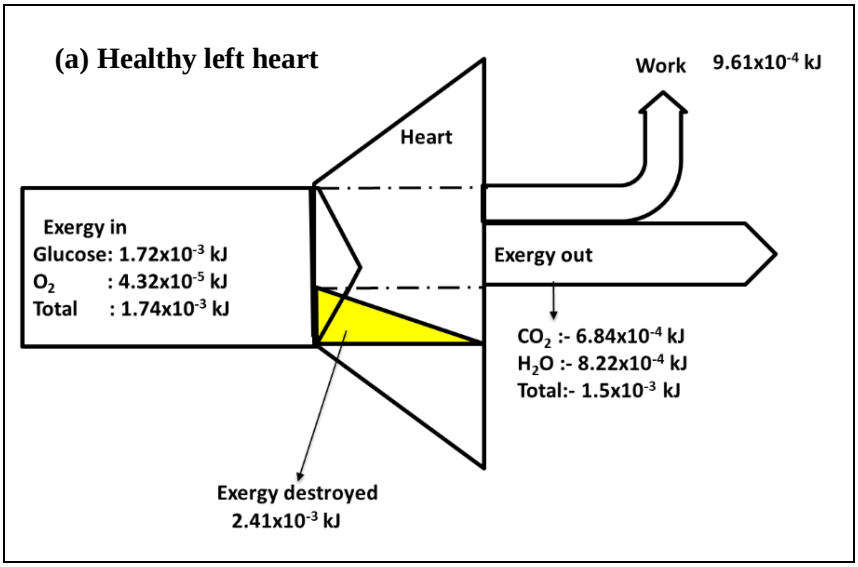
3.6. ENERGY METABOLISM IN HEART FAILURE

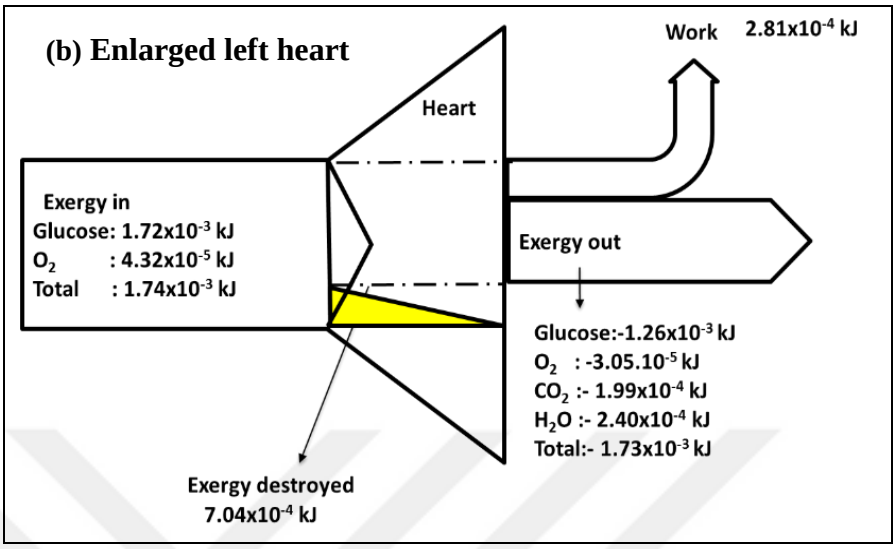
Heart muscle is an extremely oxidative tissue that generates more than 90 per cent of its energy through from mitochondrial respiration. Mitochondria captures nearly 30 per cent of cardiac muscle space and are well structured below the sarcolemma and in lines among the muscle filaments such that a continuous diffusion space occurs between mitochondria and the center of myofilaments. The heart uses above 90 per cent of its oxidative capacity during the period of the maximal exercise, in such a case there is no excess capacity for providing

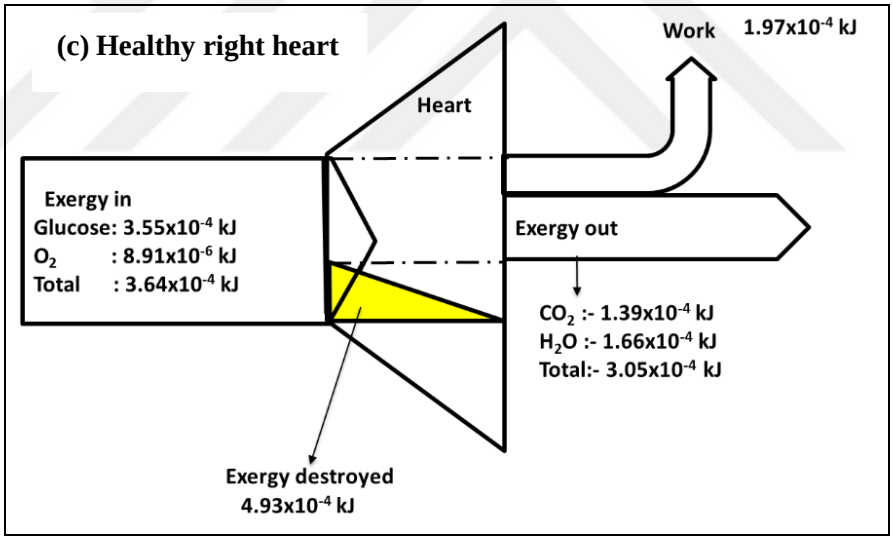
energy over utilization [148]. There is a strong association in vitro and in vivo between oxygen using up and cardiac work that takes place at steady global cellular ATP and phosphocreatine concentrations. Ca^{2+} plays an essential role in starting and regulating the strength of cardiac contraction. Therefore, a relation between the Ca^{2+} handling in the tissue and heart failure have been recognized [149-151], but postulation of the control of the excitation and contraction by Ca^{2+} ions only by ignoring the discussion provided here is not adequate [152].

One of the great magnitude irregularity damaging high energy phosphate production in the enlarged heart is a reduction in coronary circulation that can restrict nutrient and oxygen transfer to the cardiomyocytes at high workloads [153,154]. Energy need of the cardiac muscles can be satisfied only under aerobic situations [155]. The heart is capable of providing its energy supplies primarily from the oxidation of fatty acids and also glucose, lactate and other substrates available for oxidation [156]. With a down - regulation of the enzymes included in fatty acid oxidation, the main myocardial energy substrates exchange from fatty acids to glucose, in heart failure [153,154].

In Figure 3.13 it was assumed that the blood arrives to the right and left healthy and right and left enlarged hearts with the same glucose and oxygen concentrations. The left and the right healthy hearts utilize all the available glucose and oxygen; whereas the enlarged heart does not have the ability of utilizing all the available glucose and oxygen. Therefore, a fraction of the glucose and oxygen exits the heart without being utilized.




(b) Enlarged left heart
Work 2.81x10-4 kJ
Heart
Exergy in
Glucose: 1.72x10-3 kJ
O2 : 4.32x10-5 kJ
Total : 1.74x10-3 kJ
Exergy out
Glucose:-1.26x10-3 kJ
O2 : -3.05.10-5 kJ
CO2 :- 1.99x10-4 kJ
H2O :- 2.40x10-4 kJ
Total:- 1.73x10-3 kJ
Exergy destroyed
7.04x10-4 kJ


(c) Healthy right heart
Work 1.97x10-4 kJ
Heart
Exergy in
Glucose: 3.55x10-4 kJ
O2 : 8.91x10-6 kJ
Total : 3.64x10-4 kJ
Exergy out
CO2 :- 1.39x10-4 kJ
H2O :- 1.66x10-4 kJ
Total:- 3.05x10-4 kJ
Exergy destroyed
4.93x10-4 kJ

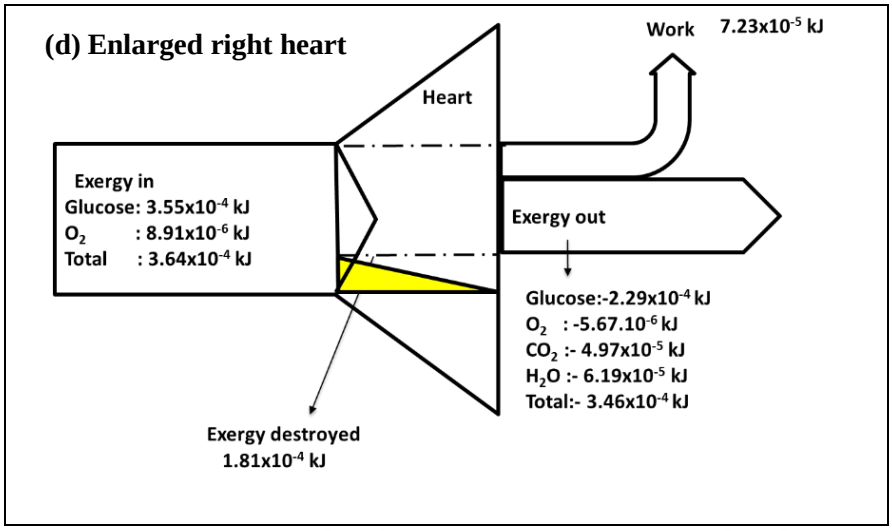


Figure 3.13. Exergy charts for the operation of (a) Healthy left, (b) Enlarged left, (c) Healthy-right and (d) Enlarged right hearts

It is generally believed that the main myocardial energy resource changes from fatty acid β-oxidation to glycolysis for the duration of hypertrophy, e.g., enlargement of the volume of the heart due to increase of the cell size, a reversion to the fetal energy substrate preference model. Early change from fatty acid to carbohydrate metabolism in hypertrophy causes in advanced efficiency of the heart providing glucose can be oxidized [156]. In hypertrophy, a proliferation in glycolysis and glycolytic enzymes is seen, but rates of glucose oxidation are decreased and more lactate accumulates. Metabolic adjustment becomes unsatisfactory with a lesser capacity to oxidize glucose leading to reduced efficiency, as the activity of renovation develops in the direction of the uncompensated condition [157].

The reduced oxidative capacity of the enlarged cardiac muscles will restrict cardiac work as a minimum for high workloads as described in Figure 3.11, because of the severe association between oxygen consumption and work. On the other hand, even in basal situations the cellular levels of ATP and PCr in addition to the PCr/ATP ratio, all of which are regulated by oxidative phosphorylation, are changed in heart failure [98]. Chronic heart failure is linked with morphological irregularities of mitochondria for example enlarged number, decreased size and compromised structural integrity [158]. In animal models of heart failure and in tissue from heart failure patients, mitochondria have been reported to be structurally atypical and reduced in number, or more abundant but littler in size. In addition, function of

these mitochondria appears to be damaged and expression of the mechanism of the electron transport chain is decreased [159].

The enlarged heart is not capable of sustaining its energetic reserve. Changes in myocardial high-energy phosphates were recognized in animal models and person hearts with left ventricle hypertrophy or heart failure. A reduction in PCr/ATP ratio is reliably described in the person with enlarged heart and investigational heart failure, even at average workloads. Creatine, creatine transporter, PCr and ATP are considerably decreased [160,161], and the reduction in the PCr/ATP ratio is a forecaster of death in inborn heart failure [162].

In addition to reduced energy generation, heart failure generates damage also in energy transfer and utilization. Reduction in the creatine kinase activity and fluxes, change in the isoenzyme pattern are characteristics of cardiac failure [163-166,162,167-169]. Moreover, the efficiency of the ATPases depends on sufficient energy supply and the effective extraction of the end products of ATP hydrolysis. Certainly, ATP and ADP exert a kinetic (through affinity and inhibition constants) in addition to a thermodynamic (through free energy of ATP hydrolysis) control on energy transduction [98].

There is some indication for energy wasting at the cellular level in cardiac dysfunction besides reduced energy generation. The enlarged heart has a decreased mechanical efficiency that increases the energy cost of force production and the energy requirements of the heart [170-173]. Accompanied by another cellular deficiency, the generalized decline in metabolic fluxes of the enzymatic systems involved in energy transfer supplies a mechanism by which energy restriction may be a key reason triggering cardiac failure [98].

3.7. SKELETAL MUSCLE METABOLISM IN HEART FAILURE

Patients suffering from heart failure continuously make a complaint of early muscular weakness and exercise intolerance because heart failure influences the rest of the body as well. Changes in energy metabolism that influence both cardiac and skeletal muscles via generalized metabolic myopathy, e.g., a muscle fiber disease [174].

The enlargement of the cardiac pump stimulates neurohormonal processes influencing the whole body strength which affects the cardiac and skeletal muscle function and structure. A decreased maximal exercise capacity of heart failure patients associates inadequately with

central hemodynamics. This has led to the conclusion that the factors outside the heart may also be involved in the muscle faintness and enlarged fatigability of the patients. Consideration has thus been concentrated on factors such as changes in vascular function and intrinsic skeletal muscle irregularities that may appear in this disease [175,176]. There are relatively few studies on skeletal muscle function in heart failure. Damaged calcium homeostasis, decreased force and slower contractile kinetics were observed that were not related to intrinsic contractile proteins [177,178]. Therefore, changes in the contractile protein profile may not be the main factor for exercise intolerance in heart failure. Alternatively, great metabolic deficiencies have been explained in skeletal muscles from heart failure patients and animals. Skeletal muscles in heart failure show a reduced mitochondrial volume that associates with the aerobic capacities of the patients, proposing a main payment of changed oxidative metabolism to exercise intolerance in heart failure [179]. It is generally recognized that heart failure influences mitochondrial capacity and regulation, and phosphotransfer systems in both cardiac and skeletal muscles [180,181]. These metabolic failures may result from reduced transcription of mitochondrial proteins or from increased mitochondrial degradation. Heart failure stimulates histological, metabolic and functional adjustments in the inspiratory muscles as well. This inspiratory muscle faintness, which takes place in 30 per cent to 50 per cent of the heart failure patients, is related with diminution in the functional capacity, decline in the quality of life and with a poor prognosis in these individuals [91].

Failure in energy metabolism are progressively thought as an essential determining factor in the development of the disorder, in spite of the variety of source and of clinical indication of heart failure [171,182,183]. Heart failure is a multi-organ disorder, disturbing diverse cell types and generating multiple neuro-hormonal activations. Inside muscle cells, this pathology influences most intracellular organelles and pathways. In myocytes, calcium and energy homeostasis are intrinsically linked so that influencing one will automatically be reflected on the other. The reduced efficiency of mechanical transduction and insufficient calcium uptake and release result in a mismatch between energy generation and utilization, and may affect calcium homeostasis and contractility. It is therefore not surprising that improving calcium homeostasis results in improved cardiac energetics [184] and that in turn improving myocardial energetics regulates calcium cycling [185,186]. Steady failure in several steps in myocardial energetic signaling, along with compromised compensatory

mechanisms, triggers the failure of the whole cardiac energetic system, finally contributing to myocardial dysfunction [98].

4. CONCLUSIONS

The lifespan entropy generation by the masseter muscle, in association with the chewing process of the food, is related with the life expectancy of a person who uptakes nutrients energy as recommended by the Institute of energy and an obese person. The results of this study imply that the lifespan entropy as suggested by Hershey [32], Hershey and Wang [33], and Silva and Annamalai [16,17] is indeed correct even at the individual muscle level. Friction is one of the major reasons which causes both wear out and entropy generation in mechanical systems; lifespan entropy generation may be regarded as the biological counterpart of the same phenomena. Stress building on the vital organs during the lifespan entropy generation has recently been assessed by Annamalai and Silva [18]. Ageing of the masseter muscle, which contribute to the process of chewing the food, may be quantified in terms of its entropy generation, supporting the definition of the concept of the entropic-age, expected lifespan and senile death [32,33]. Although carried out in a totally different context, the results of this study points the same fact as Silva and Annamalai [17] that the life expectancy is related with the food consumption, and beyond the basic level needed, as in the case of obesity, food consumption reduces the life expectancy [17]. The available knowledge about how the ageing process develops is very limited. This process is highly heterogeneous and unlikely to be expressed with a single pathway. Cesari *et al.* [187] Steinsaltz *et al*. [188] and Toussaint *et al.* [1] models are among the major theoretical studies regarding ageing. Lifespan entropy generation model offers a different and reliable view within this context. Garland [37] draws attention multidisciplinary research achieves are needed for further understanding of cancer. Since aging and cancer are expected to develop via similar mechanisms [25] the same argument is also valid for the ageing research. While working on the literature survey, it caught our attention that the thermodynamic analyses, which have great potential of making it possible to understand, and possibly slow down the ageing phenomena, are not benefited at the level they actually deserve. Therefore a simple level of mathematics has been used in this study to comply with the 80 per cent – 20 per cent rule, e.g., to attain 80 per cent of the potential benefits at the 20 per cent level of the possible modeling complexity. The detailed discussion about the 80- 20 per cent rule is available elsewhere [189].

The second law muscle work efficiency is defined as the product of the metabolic and mechanical work efficiencies. Thermodynamic analyses based on the exercise data pertinent to healthy young people are used to simulate the changes occurring in the second law muscle work efficiency during aging. The muscle work efficiency decreases as a result of the reduction of the metabolic energy generation in the mitochondria with aging. The reduction of the mitochondrial energy generation causes deterioration of the muscle structure, which causes a decline in the muscle work efficiency. Exergy loss and entropy generation in the muscles increase as a response to the attempts to higher work production in the aging muscle systems. Thermodynamic analysis of the skeletal and cardiac muscles may provide additional information to what medical sciences can provide in case of cardiac problems.

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APPENDIX A: THE CHEWING ANALYSIS IMPLEMENTED IN MATLAB PROGRAMMING LANGUAGE

Contents

M-file

```
clc
clear variables

% LOAD DATA
saliva_production=5; % 5 grams per minute
chewing_duration = 20; % in seconds
total_cycle=25;      % total cycle

% ORAL CAVITY SYSTEM INPUT DATA

% Almond
almond.lipid.mass_in=0.4942;
almond.lipid.enthalpy_in=-2.717;
%almond.lipid.entropy_in=0.0027615;
almond.lipid.exergy_in=39.54;
almond.protein.mass_in=0.2122;
almond.protein.enthalpy_in=-6.805;
almond.protein.entropy_in=0.188;
almond.protein.exergy_in=16.27;
```

```
almond.carbonhydrate.mass_in=0.0389;
almond.carbonhydrate.enthalpy_in=-6.474;
almond.carbonhydrate.entropy_in=0.392;
almond.carbonhydrate.exergy_in=17.52;
% almond.starch.mass_in=0.122;
% almond.starch.enthalpy_in=5.2524;
% almond.starch.entropy_in=0.016982;
% almond.starch.exergy_in=-0.0119;
almond.fiber.mass_in=0.122;
almond.fiber.enthalpy_in=-5.654;
almond.fiber.entropy_in=0.0011718;
almond.fiber.exergy_in=18.86;
almond.mineral.mass_in=0.0173;
almond.mineral.enthalpy_in=0;
almond.mineral.entropy_in=0.0016424;
almond.mineral.exergy_in=9.38;
almond.water.mass_in=0.047;
almond.water.enthalpy_in=-15.819;
almond.water.entropy_in=0.0005053;
almond.water.exergy_in=0.05;

% Concentration data

almond.lipid.mol_in=almond.lipid.mass_in/282.46;
almond.protein.mol_in=almond.protein.mass_in/147.13;
almond.carbonhydrate.mol_in=almond.carbonhydrate.mass_in/342.29;
almond.fiber.mol_in=almond.fiber.mass_in/162.14;
almond.mineral.mol_in=almond.mineral.mass_in/39.09;
almond.water.mol_in=almond.water.mass_in/18.01;
sum_in=almond.lipid.mol_in+almond.protein.mol_in+almond.carbonhy
drate.mol_in+almond.fiber.mol_in+almond.mineral.mol_in+almond.wa
ter.mol_in;

% Almond_fields=fieldnames(almond);
% for i=1:numel(Almond_fields)
% sum_in=sum(almond.(Almond_fields[48]).mol_in);
% end
```

```
almond.lipid.fraction_in=almond.lipid.mol_in/sum_in;
almond.protein.fraction_in=almond.protein.mol_in/sum_in;
almond.carbonhydrate.fraction_in=almond.carbonhydrate.mol_in/sum
_in;
almond.fiber.fraction_in=almond.fiber.mol_in/sum_in;
almond.mineral.fraction_in=almond.mineral.mol_in/sum_in;
almond.water.fraction_in=almond.water.mol_in/sum_in;


% Saliva
saliva.mass_in=saliva_production*chewing_duration/60;
saliva.enthalpy_in=-15.819;
saliva.entropy_in=0.0005053;
saliva.exergy_in=0.05;
saliva.water.mol_in=saliva.mass_in/18.01;
saliva.water.fraction_in=1;

% ORAL CAVITY SYSTEM OUTPUT DATA

% Almond
almond.lipid.mass_out=0.4942;
almond.lipid.enthalpy_out=-2.717;
almond.lipid.entropy_out=0.0027615;
almond.lipid.exergy_out=39.54;
almond.protein.mass_out=0.2122;
almond.protein.enthalpy_out=-6.805;
almond.protein.entropy_out=0.188;
almond.protein.exergy_out=16.27;
almond.carbonhydrate.mass_out=0.0389;
almond.carbonhydrate.enthalpy_out=-6.474;
almond.carbonhydrate.entropy_out=0.392;
almond.carbonhydrate.exergy_out=17.52;

% almond.starch.mass_in=0.122;
% almond.starch.enthalpy_in=5.2524;
% almond.starch.entropy_in=0.016982;
% almond.starch.exergy_in=-0.0119;

almond.fiber.mass_out=0.122;
almond.fiber.enthalpy_out=-5.654;
```

```
almond.fiber.entropy_out=0.0011718;
almond.fiber.exergy_out=18.86;
almond.mineral.mass_out=0.0173;
almond.mineral.enthalpy_out=0;
almond.mineral.entropy_out=0.0016424;
almond.mineral.exergy_out=9.38;
almond.water.mass_out=0.047+saliva.mass_in;
almond.water.enthalpy_out=-15.819;
almond.water.entropy_out=0.0005053;
almond.water.exergy_out=0.05;

% Concentration data

almond.lipid.mol_out=almond.lipid.mass_out/282.46;
almond.protein.mol_out=almond.protein.mass_out/147.13;
almond.carbonhydrate.mol_out=almond.carbonhydrate.mass_out/342.2
9;
almond.fiber.mol_out=almond.fiber.mass_out/162.14;
almond.mineral.mol_out=almond.mineral.mass_out/39.09;
almond.water.mol_out=almond.water.mass_out/18.01;
sum_out=almond.lipid.mol_in+almond.protein.mol_out+almond.carbon
hydrate.mol_out+almond.fiber.mol_out+almond.mineral.mol_out+almo
nd.water.mol_out;

% Almond_fields=fieldnames(almond);
% for i=1:numel(Almond_fields)
% sum_out=sum(almond.(Almond_fields{i}).mol_out);
% end

almond.lipid.fraction_out=almond.lipid.mol_out/sum_out;
almond.protein.fraction_out=almond.protein.mol_out/sum_out;
almond.carbonhydrate.fraction_out=almond.carbonhydrate.mol_out/s
um_out;
almond.fiber.fraction_out=almond.fiber.mol_out/sum_out;
almond.mineral.fraction_out=almond.mineral.mol_out/sum_out;
almond.water.fraction_out=almond.water.mol_out/sum_out;

% Saliva

saliva.mass_out=saliva_production*chewing_duration/60;
```

```
 saliva.enthalpy_out=-15.819;
 saliva.entropy_out=0.0005053;
 saliva.exergy_out=0.05;



% BLOOD CYCLE INPUT DATA
% blood.glucose.mass_in ==> will be calculated in bloodcycle.m

blood.glucose.enthalpy_in=-7.035;
blood.glucose.entropy_in=0.0011601;
blood.glucose.exergy_in=11.47;

% blood.oxygen.mass_in ==> will be calculated in bloodcycle.m

blood.oxygen.enthalpy_in=-0.366;
blood.oxygen.entropy_in=0.0064083;
blood.oxygen.exergy_in=0.27;

% BLOOD CYCLE OUTPUT DATA

% blood.co2.mass_in ==> will be calculated in bloodcycle.m

blood.co2.enthalpy_out=-15.923;
blood.co2.entropy_out=0.0048864;
blood.co2.exergy_out=-3.11;

% blood.water.mass_in ==> will be calculated in bloodcycle.m

blood.water.enthalpy_out=-15.919;
blood.water.entropy_out=0.0005053;
blood.water.exergy_out=-9.24;

% CONTRALATERAL WORK
% enter the data

% disp('Calculating Contralateral Work...')
Lc = [0 0.2 0.4 0.6 0.75 0.8 0.9 0.95 1.0 1.05 1.2 1.3 1.35 1.4
1.5 1.6...
```

```
    1.65 1.75 1.8 1.9 2.1 2.3 2.5 2.85 3.0 3.15 3.3 3.5 3.5 3.5
3.5...
    3.5 3.5 3.5 3.5 3.5 3.5 3.4 3.3 3.25 3.2 2.9 2.6 1.75 0.0 0
0 0 ...
    0 0 0 0 0 0 0 0]; % muscle length (mm)
Fc= [1 0.8 0.6 0.8 1.1 1.5 1.7 2 2.4 2.8 3 3.25 3.5 3.8 4.2 4.35
4.5 ...
    4.65 4.8 4.9 5 5.2 5.4 5.3 5.2 5.1 5 4.9 4.8 4.7 4.6 4.55
4.5 ...
    4.25 4 4.75 5 6 7 10 14 17.5 20 23 24 23 22 22.5 20 15 13.5
...
    10.5 6 4 3 1]; % Force (N)

% calculate the muscle work

for i=2:length(Fc)
     deltaW1(i)=(Fc(i)*(Lc(i)-Lc(i-1))); % incremental work
end

W1 = sum(deltaW1);

% IPSILATERAL WORK
% enter the data

% disp('Calculating Ipsilateral Work...')

Li =[0 0.5 1 1.3 1.4 1.5 1.6 1.7 1.8 1.9 2 2.2 2.4 2.4 2.1 ...
    1.8 1.3 0.8 0.8 0 0]; % muscle length (mm)
Fi= [1 1 1.5 2.0 2.3 2.6 3.0 3.5 4 4.5 5 5 4.5 4 3.5 3.3 ...
    2.6 2.3 2.3 3.6 1]; % Force (N)

% calculate the muscle work

for i=2:length(Fi)
     deltaW2(i)=(Fi(i)*(Li(i)-Li(i-1))); % incremental work
end

W2 = sum(deltaW2);

W=(W1+W2)/0.69;
```

```
% ORAL ENERGY BALANCE
% disp('Calculating the Oral Energy Balance...')
Almond_fields=fieldnames(almond);

mh_almond_in=0;
for i=1:numel(Almond_fields)
    mh_almond_in = mh_almond_in+...
        almond.(Almond_fields{i}).mass_in *
almond.(Almond_fields{i}).enthalpy_in;
end

mh_saliva_in=saliva.mass_in * saliva.enthalpy_in;

mh_almond_out=0;
for i=1:numel(Almond_fields)  %water mass_out
    mh_almond_out = mh_almond_out+...

almond.(Almond_fields{i}).mass_out*almond.(Almond_fields{i}).ent
halpy_out;
end

%mh_saliva_out=saliva.mass_out * saliva.enthalpy_out;

W_total=((W/1000)*total_cycle)/1000;
W_net=W_total*0.69;
mh_in = mh_almond_in + mh_saliva_in;
mh_out = mh_almond_out;

Q_oral = mh_out - mh_in - W_net;

% BLOOD-CYCLE ANALYSIS
% disp('Performing Blood-Cycle Analysis...')

ii=1;
for ii =1:26;
    efficiency=0.17:0.01:0.42;
total_chemical_energy = abs(W_total) / efficiency(ii); %in kJ
one_mol_ATP_energy = 3868; % ATP produced for 1 mol glucose
```

```
mol_glucose = total_chemical_energy / one_mol_ATP_energy ; % in
mol

blood.glucose.mass_in=mol_glucose*180; %1 mol glucose = 180 gr
blood.oxygen.mass_in=mol_glucose*6*32; %1 mol O2 = 32 gr
blood.co2.mass_out=mol_glucose*6*44;   %1 mol CO2 = 44 gr
blood.water.mass_out=mol_glucose*6*18; %1 mol H2O = 18 gr

reaction_time = 20 ; % in seconds
glucose_reaction_rate = (mol_glucose/reaction_time)*...
                        (60*1000); % in milimol per minute

 flow_diameter = 1.9; % in milimeters
 flow_velocity = 254; % milimeters per second
 v_blood = (pi*(flow_diameter/2)^2)*flow_velocity*(60/1e6);
%litres/mm

 glucose_absorbed = glucose_reaction_rate / v_blood; % in
mmol/lt

% BLOOD - ENERGY CYCLE
% disp('Calculating the Energy Balance for Blood...')


 Blood_fields=fieldnames(blood);

mh_blood_in=0;
for i=1:2 %glukose & oxygene
    mh_blood_in = mh_blood_in+...

blood.(Blood_fields{i}).mass_in*blood.(Blood_fields{i}).enthalpy
_in;
end

mh_blood_out=0;
for i=3:4 %co2 & water
    mh_blood_out = mh_blood_out+...

blood.(Blood_fields{i}).mass_out*blood.(Blood_fields{i}).enthalp
y_out;
```

```
end

Q_blood = mh_blood_out - mh_blood_in + abs(W_total); %Düzeltildi

% BLOOD - EXERGY BALANCE
% disp ('Calculating the Exergy Loss for Blood...')

 T_out=298;
 T_in=310;
Blood_fields=fieldnames(blood);

mb_blood_in=0;
for i=1:2
    mb_blood_in = mb_blood_in+...
        blood.(Blood_fields{i}).mass_in *
blood.(Blood_fields{i}).exergy_in;
end

mb_blood_out=0;
for i=3:4
    mb_blood_out = mb_blood_out+...
        blood.(Blood_fields{i}).mass_out *
blood.(Blood_fields{i}).exergy_out;
end

x_destroyed_blood = mb_blood_in - mb_blood_out - abs(W_total) -
...
            Q_blood*(1-(T_out/T_in)); % in kJ

Sgen_blood(ii)=x_destroyed_blood/298;
%ii=ii+1;
End

% ORAL - EXERGY BALANCE
% disp ('Calculating the Exergy Loss for Blood...')

Almond_fields=fieldnames(almond);

mb_almond_in=0;
for i=1:numel(Almond_fields)
```

```
    mb_almond_in = mb_almond_in+...
        almond.(Almond_fields{i}).mass_in *
almond.(Almond_fields{i}).exergy_in;
end

mb_almond_out=0;
for i=1:numel(Almond_fields)-1
    mb_almond_out = mb_almond_out+...
        almond.(Almond_fields{i}).mass_out *
almond.(Almond_fields{i}).exergy_out;
end
mb_almond_concentration_in=0;
for i=1:numel(Almond_fields)
    mb_almond_concentration_in = mb_almond_concentration_in+...
        almond.(Almond_fields{i}).mol_in *8.314*310*log(
almond.(Almond_fields{i}).fraction_in);
end
mb_saliva_concentration_in=saliva.water.mol_in*8.314*310*log(sal
iva.water.fraction_in);
mb_almond_concentration_out=0;
for i=1:numel(Almond_fields)-1
    mb_almond_concentration_out =
mb_almond_concentration_out+...
        almond.(Almond_fields{i}).mol_out
*8.314*310*log(almond.(Almond_fields{i}).fraction_out);
end
x_destroyed_oralc = mb_almond_in - mb_almond_out + abs(W_total)
- ...
            Q_oral*(1-
(T_out/T_in))+(mb_almond_concentration_in+mb_saliva_concentratio
n_in-mb_almond_concentration_out)/1000; % in kJ

Sgen_oralc=x_destroyed_oralc/298;


% LIFE EXPECTANCY
% disp ('Calculating Expected Life...')

age = 20:1:100;
age = age' ;
```

```
reference_age = 25;

% Survival Probability

St = [ 98.3 98.2 98.1 98.0 97.9 97.8 97.6 97.5 97.4 97.2 ...
       97.1 96.9 96.8 96.6 96.4 96.2 96.1 95.8 95.6 95.4 ...
       95.2 94.9 94.6 94.4 94.1 93.7 93.4 93.0 92.6 92.2 ...
       91.8 91.3 90.8 90.2 89.6 89.0 88.3 87.5 86.6 85.7 ...
       84.6 83.5 82.3 81.0 79.6 78.2 76.6 75.0 73.3 71.5 ...
       69.6 67.6 65.5 63.3 60.9 58.5 55.9 53.2 50.5 47.6 ...
       44.6 41.4 38.2 34.9 31.4 28.0 24.7 21.6 18.7 16.0 ...
       13.5 11.3 9.3 7.5 6 4.7 3.6 2.7 1.9 1.4 1];

for i = 1:numel(St)-1
    Lt(i) = (St(i)+St(i+1))/2;
end

% Person-years lived at and above age 25 (T25)
T25 = sum(Lt(find(age==reference_age):end));

S25 = St(find(age==reference_age));

% Life Expectancy

E25 =   T25 / S25 ;

life = round(reference_age + E25);


% ENTROPY GENERATION FOR NORMAL AND OBESE PERSON
% Calorie of 1 gram almond
% disp ('Calculating Entropy Generation for Normal and Obese
Person...')

age = 1:life; age=age';
% age_2=1:90; age_2=age_2';
almond_cal = 5.85 * 4.18; % kcal

%NORMAL
```

```
% 10

% EFF=0.17

epd_normal_10_1 = [100;120;120;130;130;130;130;130;180;180;...
              180;180;180;220;220;220;220;220;250;250;...
              250;250;250;250;250;250;250;250;250;250;...
              230;230;230;230;230;230;230;230;230;230;...
              230;230;230;230;230;230;230;230;230;230;...
              210;210;210;210;210;210;210;210;210;210;...
              210;210;210;210;210;210;210;210;210;210;...
              210;210;210;210;210;210] * 4.18;

entpd_normal_10_1 = (epd_normal_10_1 / almond_cal) *
Sgen_blood(1) ;
epy_normal_10_1=365*cumsum(epd_normal_10_1);
entpy_normal_10_1=365*cumsum(entpd_normal_10_1);
epd_fit_normal_10_1=fit(age,epd_normal_10_1,'fourier2');
epd_fit_normal_10_1=epd_fit_normal_10_1(age);
entpd_fit_normal_10_1=fit(age,entpd_normal_10_1,'fourier2');
entpd_fit_normal_10_1=entpd_fit_normal_10_1(age);

% EFF=0.3

epd_normal_10_2 = [100;120;120;130;130;130;130;130;180;180;...
              180;180;180;220;220;220;220;220;250;250;...
              250;250;250;250;250;250;250;250;250;250;...
              230;230;230;230;230;230;230;230;230;230;...
              230;230;230;230;230;230;230;230;230;230;...
              210;210;210;210;210;210;210;210;210;210;...
              210;210;210;210;210;210;210;210;210;210;...
              210;210;210;210;210;210] * 4.18;

entpd_normal_10_2 = (epd_normal_10_2 / almond_cal) *
Sgen_blood(14) ;
epy_normal_10_2=365*cumsum(epd_normal_10_2);
entpy_normal_10_2=365*cumsum(entpd_normal_10_2);
epd_fit_normal_10_2=fit(age,epd_normal_10_2,'fourier2');
epd_fit_normal_10_2=epd_fit_normal_10_2(age);
entpd_fit_normal_10_2=fit(age,entpd_normal_10_2,'fourier2');
```

```
entpd_fit_normal_10_2=entpd_fit_normal_10_2(age);

% EFF=0.42

epd_normal_10_3 = [100;120;120;130;130;130;130;130;180;180;...
              180;180;180;220;220;220;220;220;250;250;...
              250;250;250;250;250;250;250;250;250;250;...
              230;230;230;230;230;230;230;230;230;230;...
              230;230;230;230;230;230;230;230;230;230;...
              210;210;210;210;210;210;210;210;210;210;...
              210;210;210;210;210;210;210;210;210;210;...
              210;210;210;210;210;210] * 4.18;

entpd_normal_10_3 = (epd_normal_10_3 / almond_cal) *
Sgen_blood(26) ;
epy_normal_10_3=365*cumsum(epd_normal_10_3);
entpy_normal_10_3=365*cumsum(entpd_normal_10_3);
epd_fit_normal_10_3=fit(age,epd_normal_10_3,'fourier2');
epd_fit_normal_10_3=epd_fit_normal_10_3(age);
entpd_fit_normal_10_3=fit(age,entpd_normal_10_3,'fourier2');
entpd_fit_normal_10_3=entpd_fit_normal_10_3(age);

% 50

% EFF=0.17

epd_normal_50_1 = [500;600;600;650;650;650;650;650;900;900;...
              900;900;900;1100;1100;1100;1100;1100;1100;1250;...

1250;1250;1250;1250;1250;1250;1250;1250;1250;1250;...

1150;1150;1150;1150;1150;1150;1150;1150;1150;1150;...

1150;1150;1150;1150;1150;1150;1150;1150;1150;1150;...

1050;1050;1050;1050;1050;1050;1050;1050;1050;1050;...

1050;1050;1050;1050;1050;1050;1050;1050;1050;1050;...
              1050;1050;1050;1050;1050;1050] * 4.18;
```

```
entpd_normal_50_1 = (epd_normal_50_1 / almond_cal) *
Sgen_blood(1) ;
epy_normal_50_1=365*cumsum(epd_normal_50_1);
entpy_normal_50_1=365*cumsum(entpd_normal_50_1);
epd_fit_normal_50_1=fit(age,epd_normal_50_1,'fourier2');
epd_fit_normal_50_1=epd_fit_normal_50_1(age);
entpd_fit_normal_50_1=fit(age,entpd_normal_50_1,'fourier2');
entpd_fit_normal_50_1=entpd_fit_normal_50_1(age);



% EFF=0.3

epd_normal_50_2 = [500;600;600;650;650;650;650;650;900;900;...
             900;900;900;1100;1100;1100;1100;1100;1100;1250;...

1250;1250;1250;1250;1250;1250;1250;1250;1250;1250;...

1150;1150;1150;1150;1150;1150;1150;1150;1150;1150;...

1150;1150;1150;1150;1150;1150;1150;1150;1150;1150;...

1050;1050;1050;1050;1050;1050;1050;1050;1050;1050;...

1050;1050;1050;1050;1050;1050;1050;1050;1050;1050;...
             1050;1050;1050;1050;1050;1050] * 4.18;

entpd_normal_50_2 = (epd_normal_50_2 / almond_cal) *
Sgen_blood(14) ;
epy_normal_50_2=365*cumsum(epd_normal_50_2);
entpy_normal_50_2=365*cumsum(entpd_normal_50_2);
epd_fit_normal_50_2=fit(age,epd_normal_50_2,'fourier2');
epd_fit_normal_50_2=epd_fit_normal_50_2(age);
entpd_fit_normal_50_2=fit(age,entpd_normal_50_2,'fourier2');
entpd_fit_normal_50_2=entpd_fit_normal_50_2(age);



% EFF=0.42

epd_normal_50_3 = [500;600;600;650;650;650;650;650;900;900;...
```

```
            900;900;900;1100;1100;1100;1100;1100;1100;1250;...

1250;1250;1250;1250;1250;1250;1250;1250;1250;1250;...

1150;1150;1150;1150;1150;1150;1150;1150;1150;1150;...

1150;1150;1150;1150;1150;1150;1150;1150;1150;1150;...

1050;1050;1050;1050;1050;1050;1050;1050;1050;1050;...

1050;1050;1050;1050;1050;1050;1050;1050;1050;1050;...
            1050;1050;1050;1050;1050;1050] * 4.18;

entpd_normal_50_3 = (epd_normal_50_3 / almond_cal) *
Sgen_blood(26) ;
epy_normal_50_3=365*cumsum(epd_normal_50_3);
entpy_normal_50_3=365*cumsum(entpd_normal_50_3);
epd_fit_normal_50_3=fit(age,epd_normal_50_3,'fourier2');
epd_fit_normal_50_3=epd_fit_normal_50_3(age);
entpd_fit_normal_50_3=fit(age,entpd_normal_50_3,'fourier2');
entpd_fit_normal_50_3=entpd_fit_normal_50_3(age);

% 80

% EFF=0.17

epd_normal_80_1 =
[800;960;960;1040;1040;1040;1040;1040;1440;1440;...

1440;1440;1440;1760;1760;1760;1760;1760;2000;2000;...

2000;2000;2000;2000;2000;2000;2000;2000;2000;2000;...

1840;1840;1840;1840;1840;1840;1840;1840;1840;1840;...

1840;1840;1840;1840;1840;1840;1840;1840;1840;1840;...

1680;1680;1680;1680;1680;1680;1680;1680;1680;1680;...

1680;1680;1680;1680;1680;1680;1680;1680;1680;1680;...
```

```
            1680;1680;1680;1680;1680;1680] * 4.18;

entpd_normal_80_1 = (epd_normal_80_1 / almond_cal) *
Sgen_blood(1) ;
epy_normal_80_1=365*cumsum(epd_normal_80_1);
entpy_normal_80_1=365*cumsum(entpd_normal_80_1);
epd_fit_normal_80_1=fit(age,epd_normal_80_1,'fourier2');
epd_fit_normal_80_1=epd_fit_normal_80_1(age);
entpd_fit_normal_80_1=fit(age,entpd_normal_80_1,'fourier2');
entpd_fit_normal_80_1=entpd_fit_normal_80_1(age);



% EFF=0.3

epd_normal_80_2 =
[800;960;960;1040;1040;1040;1040;1040;1440;1440;...

1440;1440;1440;1760;1760;1760;1760;1760;2000;2000;...

2000;2000;2000;2000;2000;2000;2000;2000;2000;2000;...

1840;1840;1840;1840;1840;1840;1840;1840;1840;1840;...

1840;1840;1840;1840;1840;1840;1840;1840;1840;1840;...

1680;1680;1680;1680;1680;1680;1680;1680;1680;1680;...

1680;1680;1680;1680;1680;1680;1680;1680;1680;1680;...
            1680;1680;1680;1680;1680;1680] * 4.18;

entpd_normal_80_2 = (epd_normal_80_2 / almond_cal) *
Sgen_blood(14) ;
epy_normal_80_2=365*cumsum(epd_normal_80_2);
entpy_normal_80_2=365*cumsum(entpd_normal_80_2);
epd_fit_normal_80_2=fit(age,epd_normal_80_2,'fourier2');
epd_fit_normal_80_2=epd_fit_normal_80_2(age);
entpd_fit_normal_80_2=fit(age,entpd_normal_80_2,'fourier2');
entpd_fit_normal_80_2=entpd_fit_normal_80_2(age);
```

```
% EFF=0.42

epd_normal_80_3 =
[800;960;960;1040;1040;1040;1040;1040;1440;1440;...

1440;1440;1440;1760;1760;1760;1760;1760;2000;2000;...

2000;2000;2000;2000;2000;2000;2000;2000;2000;2000;...

1840;1840;1840;1840;1840;1840;1840;1840;1840;1840;...

1840;1840;1840;1840;1840;1840;1840;1840;1840;1840;...

1680;1680;1680;1680;1680;1680;1680;1680;1680;1680;...

1680;1680;1680;1680;1680;1680;1680;1680;1680;1680;...
             1680;1680;1680;1680;1680;1680] * 4.18;

entpd_normal_80_3 = (epd_normal_80_3 / almond_cal) *
Sgen_blood(26);
epy_normal_80_3=365*cumsum(epd_normal_80_3);
entpy_normal_80_3=365*cumsum(entpd_normal_80_3);
epd_fit_normal_80_3=fit(age,epd_normal_80_3,'fourier2');
epd_fit_normal_80_3=epd_fit_normal_80_3(age);
entpd_fit_normal_80_3=fit(age,entpd_normal_80_3,'fourier2');
entpd_fit_normal_80_3=entpd_fit_normal_80_3(age);

% 10

% EFF=0.17

epd_obese_10_1 = [ 110;132;132;143;143;143;143;143;198;198;...
              198;198;198;242;242;242;242;242;275;275;...
              275;275;275;275;275;275;275;275;275;275;...
              253;253;253;253;253;253;253;253;253;253;...
              253;253;253;253;253;253;253;253;253;253;...
              231;231;231;231;231;231;231;231;231;231;...
              231;231;231;231;231;231;231;231;231;231;...
              231;231;231;231;231;231] * 4.18;
```

```
epy_obese_10_1=365*cumsum(epd_obese_10_1);
entpd_obese_10_1 = (epd_obese_10_1 / almond_cal) * Sgen_blood(1)
;
entpy_obese_10_1=365*cumsum(entpd_obese_10_1);
epd_fit_obese_10_1=fit(age,epd_obese_10_1,'fourier2');
epd_fit_obese_10_1=epd_fit_obese_10_1(age);
entpd_fit_obese_10_1=fit(age,entpd_obese_10_1,'fourier2');
entpd_fit_obese_10_1=entpd_fit_obese_10_1(age);

% EFF=0.3

epd_obese_10_2 = [ 110;132;132;143;143;143;143;143;198;198;...
              198;198;198;242;242;242;242;242;275;275;...
              275;275;275;275;275;275;275;275;275;275;...
              253;253;253;253;253;253;253;253;253;253;...
              253;253;253;253;253;253;253;253;253;253;...
              231;231;231;231;231;231;231;231;231;231;...
              231;231;231;231;231;231;231;231;231;231;...
              231;231;231;231;231;231] * 4.18;

epy_obese_10_2=365*cumsum(epd_obese_10_2);
entpd_obese_10_2 = (epd_obese_10_2 / almond_cal) *
Sgen_blood(14) ;
entpy_obese_10_2=365*cumsum(entpd_obese_10_2);
epd_fit_obese_10_2=fit(age,epd_obese_10_2,'fourier2');
epd_fit_obese_10_2=epd_fit_obese_10_2(age);
entpd_fit_obese_10_2=fit(age,entpd_obese_10_2,'fourier2');
entpd_fit_obese_10_2=entpd_fit_obese_10_2(age);

% EFF=0.42

epd_obese_10_3 = [ 110;132;132;143;143;143;143;143;198;198;...
              198;198;198;242;242;242;242;242;275;275;...
              275;275;275;275;275;275;275;275;275;275;...
              253;253;253;253;253;253;253;253;253;253;...
              253;253;253;253;253;253;253;253;253;253;...
              231;231;231;231;231;231;231;231;231;231;...
              231;231;231;231;231;231;231;231;231;231;...
              231;231;231;231;231;231] * 4.18;
```

```
epy_obese_10_3=365*cumsum(epd_obese_10_3);
entpd_obese_10_3 = (epd_obese_10_3 / almond_cal) *
Sgen_blood(26) ;
entpy_obese_10_3=365*cumsum(entpd_obese_10_3);
epd_fit_obese_10_3=fit(age,epd_obese_10_3,'fourier2');
epd_fit_obese_10_3=epd_fit_obese_10_3(age);
entpd_fit_obese_10_3=fit(age,entpd_obese_10_3,'fourier2');
entpd_fit_obese_10_3=entpd_fit_obese_10_3(age);

% 50

% EFF=0.17

epd_obese_50_1 = [ 550;660;660;715;715;715;715;715;990;990;...
              990;990;990;1210;1210;1210;1210;1210;1375;1375;...

1375;1375;1375;1375;1375;1375;1375;1375;1375;1375;...

1265;1265;1265;1265;1265;1265;1265;1265;1265;1265;...

1265;1265;1265;1265;1265;1265;1265;1265;1265;1265;...

1155;1155;1155;1155;1155;1155;1155;1155;1155;1155;...

1155;1155;1155;1155;1155;1155;1155;1155;1155;1155;...
              1155;1155;1155;1155;1155;1155] * 4.18;

epy_obese_50_1=365*cumsum(epd_obese_50_1);
entpd_obese_50_1 = (epd_obese_50_1 / almond_cal) * Sgen_blood(1)
;
entpy_obese_50_1=365*cumsum(entpd_obese_50_1);
epd_fit_obese_50_1=fit(age,epd_obese_50_1,'fourier2');
epd_fit_obese_50_1=epd_fit_obese_50_1(age);
entpd_fit_obese_50_1=fit(age,entpd_obese_50_1,'fourier2');
entpd_fit_obese_50_1=entpd_fit_obese_50_1(age);

% EFF=0.3

epd_obese_50_2 = [ 550;660;660;715;715;715;715;715;990;990;...
              990;990;990;1210;1210;1210;1210;1210;1375;1375;...
```

```
1375;1375;1375;1375;1375;1375;1375;1375;1375;1375;...

1265;1265;1265;1265;1265;1265;1265;1265;1265;1265;...

1265;1265;1265;1265;1265;1265;1265;1265;1265;1265;...

1155;1155;1155;1155;1155;1155;1155;1155;1155;1155;...

1155;1155;1155;1155;1155;1155;1155;1155;1155;1155;...
              1155;1155;1155;1155;1155;1155] * 4.18;

epy_obese_50_2=365*cumsum(epd_obese_50_2);
entpd_obese_50_2 = (epd_obese_50_2 / almond_cal) *
Sgen_blood(14) ;
entpy_obese_50_2=365*cumsum(entpd_obese_50_2);
epd_fit_obese_50_2=fit(age,epd_obese_50_2,'fourier2');
epd_fit_obese_50_2=epd_fit_obese_50_2(age);
entpd_fit_obese_50_2=fit(age,entpd_obese_50_2,'fourier2');
entpd_fit_obese_50_2=entpd_fit_obese_50_2(age);

% EFF=0.42

epd_obese_50_3 = [ 550;660;660;715;715;715;715;715;990;990;...
              990;990;990;1210;1210;1210;1210;1210;1375;1375;...

1375;1375;1375;1375;1375;1375;1375;1375;1375;1375;...

1265;1265;1265;1265;1265;1265;1265;1265;1265;1265;...

1265;1265;1265;1265;1265;1265;1265;1265;1265;1265;...

1155;1155;1155;1155;1155;1155;1155;1155;1155;1155;...

1155;1155;1155;1155;1155;1155;1155;1155;1155;1155;...
              1155;1155;1155;1155;1155;1155] * 4.18;

epy_obese_50_3=365*cumsum(epd_obese_50_3);
entpd_obese_50_3 = (epd_obese_50_3 / almond_cal) *
Sgen_blood(26) ;
```

```
entpy_obese_50_3=365*cumsum(entpd_obese_50_3);
epd_fit_obese_50_3=fit(age,epd_obese_50_3,'fourier2');
epd_fit_obese_50_3=epd_fit_obese_50_3(age);
entpd_fit_obese_50_3=fit(age,entpd_obese_50_3,'fourier2');
entpd_fit_obese_50_3=entpd_fit_obese_50_3(age);

% 80

% EFF=0.17

epd_obese_80_1 = [
880;1056;1056;1144;1144;1144;1144;1144;1584;1584;...

1584;1584;1584;1936;1936;1936;1936;1936;2200;2200;...

2200;2200;2200;2200;2200;2200;2200;2200;2200;2200;...

2024;2024;2024;2024;2024;2024;2024;2024;2024;2024;...

2024;2024;2024;2024;2024;2024;2024;2024;2024;2024;...

1848;1848;1848;1848;1848;1848;1848;1848;1848;1848;...

1848;1848;1848;1848;1848;1848;1848;1848;1848;1848;...
              1848;1848;1848;1848;1848;1848] * 4.18;

epy_obese_80_1=365*cumsum(epd_obese_80_1);
entpd_obese_80_1 = (epd_obese_80_1 / almond_cal) * Sgen_blood(1)
;
entpy_obese_80_1=365*cumsum(entpd_obese_80_1);
epd_fit_obese_80_1=fit(age,epd_obese_80_1,'fourier2');
epd_fit_obese_80_1=epd_fit_obese_80_1(age);
entpd_fit_obese_80_1=fit(age,entpd_obese_80_1,'fourier2');
entpd_fit_obese_80_1=entpd_fit_obese_80_1(age);

% EFF=0.3

epd_obese_80_2 =
[880;1056;1056;1144;1144;1144;1144;1144;1584;1584;...
```

```
1584;1584;1584;1936;1936;1936;1936;1936;2200;2200;...

2200;2200;2200;2200;2200;2200;2200;2200;2200;2200;...

2024;2024;2024;2024;2024;2024;2024;2024;2024;2024;...

2024;2024;2024;2024;2024;2024;2024;2024;2024;2024;...

1848;1848;1848;1848;1848;1848;1848;1848;1848;1848;...

1848;1848;1848;1848;1848;1848;1848;1848;1848;1848;...
             1848;1848;1848;1848;1848;1848] * 4.18;

epy_obese_80_2=365*cumsum(epd_obese_80_2);
entpd_obese_80_2 = (epd_obese_80_2 / almond_cal) *
Sgen_blood(14) ;
entpy_obese_80_2=365*cumsum(entpd_obese_80_2);
epd_fit_obese_80_2=fit(age,epd_obese_80_2,'fourier2');
epd_fit_obese_80_2=epd_fit_obese_80_2(age);
entpd_fit_obese_80_2=fit(age,entpd_obese_50_2,'fourier2');
entpd_fit_obese_80_2=entpd_fit_obese_80_2(age);

% EFF=0.42

epd_obese_80_3 = [
880;1056;1056;1144;1144;1144;1144;1144;1584;1584;...

1584;1584;1584;1936;1936;1936;1936;1936;2200;2200;...

2200;2200;2200;2200;2200;2200;2200;2200;2200;2200;...

2024;2024;2024;2024;2024;2024;2024;2024;2024;2024;...

2024;2024;2024;2024;2024;2024;2024;2024;2024;2024;...

1848;1848;1848;1848;1848;1848;1848;1848;1848;1848;...

1848;1848;1848;1848;1848;1848;1848;1848;1848;1848;...
             1848;1848;1848;1848;1848;1848] * 4.18;
```

```
epy_obese_80_3=365*cumsum(epd_obese_80_3);
entpd_obese_80_3 = (epd_obese_80_3 / almond_cal) *
Sgen_blood(26) ;
entpy_obese_80_3=365*cumsum(entpd_obese_80_3);
epd_fit_obese_80_3=fit(age,epd_obese_80_3,'fourier2');
epd_fit_obese_80_3=epd_fit_obese_80_3(age);
entpd_fit_obese_80_3=fit(age,entpd_obese_80_3,'fourier2');
entpd_fit_obese_80_3=entpd_fit_obese_80_3(age);

% RESULTS

disp('Contralateral Analysis Results:')

disp(['Total work for ' num2str(total_cycle)...
      ' cycles is ' num2str(abs(W_total)) ' kJ.'])

disp(['Glucose absorbed to the cell is ' ...
    num2str(glucose_absorbed) ' mmol/lt.'])

disp(['Q for blood is ' num2str(Q_blood) ' kj.'])

disp(['Q for oral cavity is ' num2str(Q_oral) ' kj.'])

disp(['Sgen for normal 10% eff=0.17 is '
num2str(entpy_normal_10_1(76)) ' kj/K.'])
disp(['Sgen for obese 10% eff=0.17 is '
num2str(entpy_obese_10_1(76)) ' kj/K.'])

disp(['Sgen for normal 10% eff=0.3 is '
num2str(entpy_normal_10_2(76)) ' kj/K.'])
disp(['Sgen for obese 10% eff=0.3 is '
num2str(entpy_obese_10_2(76)) ' kj/K.'])

disp(['Sgen for normal 10% eff=0.42 is '
num2str(entpy_normal_10_3(76)) ' kj/K.'])
disp(['Sgen for obese 10% eff=0.42 is '
num2str(entpy_obese_10_3(76)) ' kj/K.'])
```

```
disp(['Sgen for normal 50% eff=0.17 is '
num2str(entpy_normal_50_1(76)) ' kj/K.'])
disp(['Sgen for obese 50% eff=0.17 is '
num2str(entpy_obese_50_1(76)) ' kj/K.'])

disp(['Sgen for normal 50% eff=0.3 is '
num2str(entpy_normal_50_2(76)) ' kj/K.'])
disp(['Sgen for obese 50% eff=0.3 is '
num2str(entpy_obese_50_2(76)) ' kj/K.'])

disp(['Sgen for normal 50% eff=0.42 is '
num2str(entpy_normal_50_3(76)) ' kj/K.'])
disp(['Sgen for obese 50% eff=0.42 is '
num2str(entpy_obese_50_3(76)) ' kj/K.'])

disp(['Sgen for normal 80% eff=0.17 is '
num2str(entpy_normal_80_1(76)) ' kj/K.'])
disp(['Sgen for obese 80% eff=0.17 is '
num2str(entpy_obese_80_1(76)) ' kj/K.'])

disp(['Sgen for normal 80% eff=0.3 is '
num2str(entpy_normal_80_2(76)) ' kj/K.'])
disp(['Sgen for obese 80% eff=0.3 is '
num2str(entpy_obese_80_2(76)) ' kj/K.'])

disp(['Sgen for normal 80% eff=0.42 is '
num2str(entpy_normal_80_3(76)) ' kj/K.'])
disp(['Sgen for obese 80% eff=0.42 is '
num2str(entpy_obese_80_3(76)) ' kj/K.'])

disp(['X_destroyed for blood is ' num2str(x_destroyed_blood) '
kj'])

disp(['X_destroyed for oral cavity is '
num2str(x_destroyed_oralc) ' kj'])

disp(['Expected life is ' num2str(life) ' years.'])

% PLOTTING
```

```
% Entropy generation vs 2nd Law Efficiency

figure;
plot(efficiency,Sgen_blood,'LineStyle','-
.','Marker','x','color','blue','LineWidth',2)
xlabel('2nd law efficiency')
ylabel('Sgen,muscle')
title('Entropy Generation versus 2nd law Efficiency')
ylim([0 5e-5]); % limit of the range of the y axis
grid on

% Work loop

figure;
plot(Lc,Fc,'k-', 'LineWidth',2.85)
hold on
plot(Li,Fi,'r-','LineWidth',2.85)
xlabel('L (mm)')
ylabel('F (N)')
title('Work Loop')
legend('Contralateral','Ipsilateral','Location','NorthEast');
ylim([0 25]); % limit of the range of the y axis
grid on
% hold on
% plot(L,F,'k-', 'LineWidth',2.85)
% xlabel('L (mm)')
% ylabel('F (N)')
% title('Workloop (Ipsilateral)')
% ylim([-20 50]); % limit of the range of the y axis
% grid on
% Sgen cumulative & Energy Expenditure
% 10

% EFF=0.17

figure
plot(age,entpy_normal_10_1,'LineStyle','-
','Marker','o','color','red','LineWidth',2);
hold on
```

```
plot(age,entpy_obese_10_1,'LineStyle','-
.','Marker','o','color','yellow','LineWidth',2);
grid on
hold on

% EFF=0.3

plot(age,entpy_normal_10_2,'LineStyle','-
','Marker','x','color','green','LineWidth',2);
hold on
plot(age,entpy_obese_10_2,'LineStyle','-
.','Marker','x','color','blue','LineWidth',2);
grid on
hold on


% EFF=0.42

plot(age,entpy_normal_10_3,'LineStyle','--
','Marker','+','color','black','LineWidth',2);
hold on
plot(age,entpy_obese_10_3,'LineStyle',':','Marker','+','color','
magenta','LineWidth',2);
grid on
title('Entropy Comparison of Normal and Obese Person 10% solid
consumed');
xlabel('age (years)');
ylabel('Entropy (kjoules/Kelvin) Cumulative');
% ylabel('Energy (joules) per day');
% set(get(x(2),'ylabel'), 'string', 'Entropy (kjoules/Kelvin)
Cumulative');
legend('Entropy normal 2nd law eff=0.17','Entropy obese 2nd law
eff=0.17','Entropy normal 2nd law eff=0.3','Entropy obese 2nd
law eff=0.3','Entropy normal 2nd law eff=0.42','Entropy obese
2nd law eff=0.42','Location','NorthWest');
% legend(y2,'Entropy normal','Location','East');
% 50

% EFF=0.17
```

```
figure
plot(age,entpy_normal_50_1,'LineStyle','-
','Marker','o','color','red','LineWidth',2);
hold on
plot(age,entpy_obese_50_1,'LineStyle','-
.','Marker','o','color','yellow','LineWidth',2);
grid on
hold on

% EFF=0.3

plot(age,entpy_normal_50_2,'LineStyle','-
','Marker','x','color','green','LineWidth',2);
hold on
plot(age,entpy_obese_50_2,'LineStyle','-
.','Marker','x','color','blue','LineWidth',2);
grid on
hold on

% EFF=0.42

plot(age,entpy_normal_50_3,'LineStyle','--
','Marker','+','color','black','LineWidth',2);
hold on
plot(age,entpy_obese_50_3,'LineStyle',':','Marker','+','color','
magenta','LineWidth',2);
grid on
title('Entropy Comparison of Normal and Obese Person 50% solid
consumed');
xlabel('age (years)');
ylabel('Entropy (kjoules/Kelvin) Cumulative');
legend('Entropy normal 2nd law eff=0.17','Entropy obese 2nd law
eff=0.17','Entropy normal 2nd law eff=0.3','Entropy obese 2nd
law eff=0.3','Entropy normal 2nd law eff=0.42','Entropy obese
2nd law eff=0.42','Location','NorthWest');

% 80

% EFF=0.17
figure
```

```
plot(age,entpy_normal_80_1,'LineStyle','-
','Marker','o','color','red','LineWidth',2);
hold on
plot(age,entpy_obese_80_1,'LineStyle','-
.','Marker','o','color','yellow','LineWidth',2);
grid on
hold on

% EFF=0.3

plot(age,entpy_normal_80_2,'LineStyle','-
','Marker','x','color','green','LineWidth',2);
hold on
plot(age,entpy_obese_80_2,'LineStyle','-
.','Marker','x','color','blue','LineWidth',2);
grid on
hold on

% EFF=0.42

plot(age,entpy_normal_80_3,'LineStyle','--
','Marker','+','color','black','LineWidth',2);
hold on
plot(age,entpy_obese_80_3,'LineStyle',':','Marker','+','color','
magenta','LineWidth',2);
grid on
title('Entropy Comparison of Normal and Obese Person 80% solid
consumed');
xlabel('age (years)');
ylabel('Entropy (kjoules/Kelvin) Cumulative');
legend('Entropy normal 2nd law eff=0.17','Entropy obese 2nd law
eff=0.17','Entropy normal 2nd law eff=0.3','Entropy obese 2nd
law eff=0.3','Entropy normal 2nd law eff=0.42','Entropy obese
2nd law eff=0.42','Location','NorthWest');

% Energy Expenditure

epd_normal =
[1000;1200;1200;1300;1300;1300;1300;1300;1800;1800;...
```

```
1800;1800;1800;2200;2200;2200;2200;2200;2500;2500;...

2500;2500;2500;2500;2500;2500;2500;2500;2500;2500;...

2300;2300;2300;2300;2300;2300;2300;2300;2300;2300;...

2300;2300;2300;2300;2300;2300;2300;2300;2300;2300;...

2100;2100;2100;2100;2100;2100;2100;2100;2100;2100;...

2100;2100;2100;2100;2100;2100;2100;2100;2100;2100;...
             2100;2100;2100;2100;2100;2100] * 4.18;

epd_fit_normal=fit(age,epd_normal,'fourier2');
epd_fit_normal=epd_fit_normal(age);

epd_obese = [
1250;1500;1500;1625;1625;1625;1625;1625;2250;2250;...

2250;2250;2250;2750;2750;2750;2750;2750;3125;3125;...

3125;3125;3125;3125;3125;3125;3125;3125;3125;3125;...

2875;2875;2875;2875;2875;2875;2875;2875;2875;2875;...

2875;2875;2875;2875;2875;2875;2875;2875;2875;2875;...

2625;2625;2625;2625;2625;2625;2625;2625;2625;2625;...

2625;2625;2625;2625;2625;2625;2625;2625;2625;2625;...
             2625;2625;2625;2625;2625;2625] * 4.18;
epd_fit_obese=fit(age,epd_obese,'fourier2');
epd_fit_obese=epd_fit_obese(age);

figure
plot(age,epd_fit_normal,'LineStyle','--
','Marker','o','color','red','LineWidth',2);
hold on
```

```
plot(age,epd_fit_obese,'LineStyle','--
','Marker','o','color','yellow','LineWidth',2);
grid on
title('Daily Dietary Intake for Normal and Obese Person');
xlabel('age (years)');
ylabel('Energy (joules) per day');
legend('Normal Person','Obese Person','Location','NorthWest');
```

Contralateral Analysis Results:

Total work for 25 cycles is 0.0023027 kJ.

Glucose absorbed to the cell is 0.098411 mmol/lt.

Q for blood is -0.0041982 kj.

Q for oral cavity is 0.0015889 kj.

Sgen for normal 10% eff=0.17 is 40.3632 kj/K.

Sgen for obese 10% eff=0.17 is 44.3995 kj/K.

Sgen for normal 10% eff=0.3 is 19.3895 kj/K.

Sgen for obese 10% eff=0.3 is 21.3284 kj/K.

Sgen for normal 10% eff=0.42 is 11.5531 kj/K.

Sgen for obese 10% eff=0.42 is 12.7085 kj/K.

Sgen for normal 50% eff=0.17 is 201.4386 kj/K.

Sgen for obese 50% eff=0.17 is 221.9975 kj/K.

Sgen for normal 50% eff=0.3 is 96.7661 kj/K.

Sgen for obese 50% eff=0.3 is 106.6421 kj/K.

Sgen for normal 50% eff=0.42 is 57.6577 kj/K.

Sgen for obese 50% eff=0.42 is 63.5423 kj/K.

Sgen for normal 80% eff=0.17 is 322.9054 kj/K.

Sgen for obese 80% eff=0.17 is 355.1959 kj/K.

Sgen for normal 80% eff=0.3 is 155.1158 kj/K.

Sgen for obese 80% eff=0.3 is 170.6273 kj/K.

Sgen for normal 80% eff=0.42 is 92.4251 kj/K.

Sgen for obese 80% eff=0.42 is 101.6677 kj/K.

X_destroyed for blood is 0.003438 kj

X_destroyed for oral cavity is 0.028474 kj

Expected life is 76 years.

APPENDIX B: GLUCOSE CONSUMPTION, EXERGY DESTRUCTION AND ENTROPY GENERATION FOR THE CASE WHERE THE 2ND LAW EFFICIENCIES ARE η_{II}=0.17, η_{II}=0.3 AND η_{II}=0.42 IMPLEMENTED IN MATLAB PROGRAMMING LANGUAGE

M-file

```
clear all
clc
close all

% introduce the data 0.30

wData30 = [122 329 122 207 30 60 90 100 150 238 387 200 33 65 98
131 20 40 20 40];
gData30 = [6.308 17.01 6.308 10.70 1.551 3.102 4.653 5.170 7.755
12.30 20.01 10.34 1.706 3.360 5.067 6.773 1.034 2.068 1.034
2.068];
exData30 = [305.5 823.8 305.5 518.3 75.12 150.2 225.3 250.4 375.6
595.9 969.1 500.8 82.63 162.7 245.4 328 50.08 100.1 50.08 100.1];
entData30 = [1.025 2.764 1.025 1.739 0.252 0.504 0.756 0.840
1.260 1.999 3.252 1.680 0.277 0.546 0.823 1.100 0.168 0.336 0.168
0.336];

% introduce the data 0.17

wData17 = [122 329 122 207 30 60 90 100 150 238 387 200 33 65 98
131 20 40 20 40];
gData17 = [11.13 30.02 11.13 18.88 2.737 5.474 8.212 9.124 13.68
21.71 35.31 18.24 3.011 5.931 8.942 11.95 1.824 3.649 1.824
3.649];
exData17 = [636 1715 636 79.16 156.4 312.8 469.2 521.3 782 1240
2017 1042 172 338.8 510.9 682.9 104.2 208.5 104.2 208.5];
entData17 = [2.134 5.755 2.134 3.621 0.524 1.049 1.574 1.749
2.624 4.163 6.770 3.498 0.577 1.137 1.714 2.291 0.349 0.699 0.349
0.699];
```

```
% introduce the data 0.42

wData42 = [122 329 122 207 30 60 90 100 150 238 387 200 33 65 98
131 20 40 20 40];
gData42 = [4.505 12.15 4.505 7.645 1.107 2.215 3.323 3.693 5.539
8.790 14.29 7.386 1.218 2.400 3.619 4.838 0.738 1.477 0.738
1.477];
exData42 = [182 490.8 182 308.8 44.75 89.51 134.2 149.1 223.7 355
577.3 298.3 49.23 96.97 146.2 195.4 29.83 59.67 29.83 59.67];
entData42 = [0.610 1.647 0.610 1.036 0.150 0.300 0.450 0.500
0.750 1.191 1.937 1.001 0.165 0.325 0.490 0.655 0.100 0.200 0.100
0.200];

% plot the data
figure;
plot(wData30(1:3), gData30(1:3),'+',wData30(4),gData30(4),'o',
...
    wData30(5:7), gData30(5:7),'*',wData30(8:10),
gData30(8:10),'x', ...
    wData30(11:12), gData30(11:12),'s',wData30(13:16),
gData30(13:16),'p',...
    wData30(17:20),
gData30(17:20),'v','MarkerSize',10,'LineWidth',2);
hold on
plot(wData17(1:3), gData17(1:3),'+',wData17(4),gData17(4),'o',
...
    wData17(5:7), gData17(5:7),'*',wData17(8:10),
gData17(8:10),'x', ...
    wData17(11:12), gData17(11:12),'s',wData17(13:16),
gData17(13:16),'p',...
    wData17(17:20),
gData17(17:20),'v','MarkerSize',10,'LineWidth',2);
grid on;
h=legend('Arm (Volianitis and Secher, 2002)','Leg (Volianitis and
Secher, 2002)','Arm (Ahlborg and Urstad, 1991)','Leg (Ahlborg and
Urstad, 1991)','Arm (Elfegoun et al., 2006)','Leg (Pedersen et
al., 1999)','Leg (Strange, 1999)','Location','Northwest');
set(h,'FontSize',8);
```

```
% put the labels
xlabel('W (J)','FontSize',16); hold on
ylabel('Glucose consumed (mmol/min)','FontSize',16);
% title('Title','FontSize',16);
set(gca,'FontSize',16);

%plot lines
hold on
plot(wData30(1:3), gData30(1:3),'k',wData30(4),gData30(4),'k',
...
    wData30(5:7), gData30(5:7),'k',wData30(8:10),
gData30(8:10),'k', ...
    wData30(11:12), gData30(11:12),'k',wData30(13:16),
gData30(13:16),'k',...
    wData30(17:20),
gData30(17:20),'k','MarkerSize',10,'LineWidth',2);


figure;
plot(wData30(1:3), exData30(1:3),'+',wData30(4),exData30(4),'o',
...
    wData30(5:7), exData30(5:7),'*',wData30(8:10),
exData30(8:10),'x', ...
    wData30(11:12), exData30(11:12),'s',wData30(13:16),
exData30(13:16),'p',...
    wData30(17:20),
exData30(17:20),'v','MarkerSize',10,'LineWidth',2);
grid on;
h=legend('Arm (Volianitis and Secher, 2002)','Leg (Volianitis and
Secher, 2002)','Arm (Ahlborg and Urstad, 1991)','Leg (Ahlborg and
Urstad, 1991)','Arm (Elfegoun et al., 2006)','Leg (Pedersen et
al., 1999)','Leg (Strange, 1999)','Location','Northwest');
set(h,'FontSize',8)

% put the labels

xlabel('W (J)','FontSize',16); hold on
ylabel('Exergy Destroyed (Joule)','FontSize',16);
```

```
% title('Title','FontSize',16);

set(gca,'FontSize',16);

%plot lines
cykr=corrcoef(wData,gData)
```

APPENDIX C: WORK PERFORMANCE, EXERGY DESTRUCTION AND ENTROPY GENERATION FOR THE CASE WHERE THE SECOND LAW MUSCLE WORK EFFICIENCY DECREASES LINEARLY IMPLEMENTED IN MATLAB PROGRAMMING LANGUAGE

M-file

```
clear all; close all; clc;

t=20:10:80;
eff=[0.42 0.38 0.34 0.3 0.26 0.22 0.17];
W=[1624.56 1469.84 1315.12 1160.4 1005.68 850.96 657.56];
Exergy=[2423.66 2584.37 2745.08 2905.79 3066.5 3227.21 3428.09];
Entropy=[8133.1 8672.4 9211.6 9750.9 10290.2 10829.5 11503.6];

figure
[hx,h1,h2] = plotyy(t,eff,[t',t',t'],[W',Exergy',Entropy']);

set(hx(1),'YColor',[0 0 1],'LineWidth',2,'FontSize',16,'Ylim',[0
0.6],'YTick',[0 0.1 0.2 0.3 0.4 0.5 0.6],'YTickLabel',[0 0.1 0.2
0.3 0.4 0.5 0.6]);

set(hx(2),'YColor',[0 0.5
0],'LineWidth',2,'FontSize',16,'Ylim',[0 12000],'YTick',[0 2000
4000 6000 8000 10000 12000],'YTickLabel',[0 2000 4000 6000 8000
10000 12000]);

set(h2,'Color',[0 0.5 0],'LineWidth',2);
set(h1,'Color',[0 0 1],'LineWidth',2);
grid on
set(h2(3),'LineStyle','-.')
set(h2(2),'LineStyle',':')
t1=text(50,0.08,'W (kJ/mol glucose)');
set(t1,'FontSize',16,'Color',[0 0.5 0]);
t2=text(50,0.17,'Exergy destruction (kJ/mol glucose)');
set(t2,'FontSize',16,'Color',[0 0.5 0]);
```

```
t3=text(50,0.47,'Entropy generation (J/K)/(mol glucose)');
set(t3,'FontSize',16,'Color',[0 0.5 0]);
t4=text(50,0.31,'2nd law efficiency');
set(t4,'FontSize',16,'Color',[0 0.5 0]);

ylabel('2nd Law Efficiency');
xlabel('Age (Years)');
%title('Path 1')
```

APPENDIX D: WORK PERFORMANCE, EXERGY DESTRUCTION AND ENTROPY GENERATION FOR THE CASE WHERE THE SECOND LAW MUSCLE WORK EFFICIENCY DECREASES GRADUALLY UNTIL THE AGE OF 70 AND THEN FASTER IMPLEMENTED IN MATLAB PROGRAMMING LANGUAGE

M-file

```
clear all; close all; clc;

t=20:10:80;
eff=[0.42 0.41 0.39 0.38 0.35 0.30 0.17];
W=[1624.56 1585.88 1508.52 1469.84 1353.8 1160.4 657.56];
Exergy=[2423.66 2463.84 2544.19 2584.37 2704.90 2905.79 3428.09];
Entropy=[8133.1 8267.9 8537.5 8672.4 9076.8 9750.9 11503.6];

figure
[hx,h1,h2] = plotyy(t,eff,[t',t',t'],[W',Exergy',Entropy']);

set(hx(1),'YColor',[0 0 1],'LineWidth',2,'FontSize',16,'Ylim',[0
0.6],'YTick',[0 0.1 0.2 0.3 0.4 0.5 0.6],'YTickLabel',[0 0.1 0.2
0.3 0.4 0.5 0.6]);

set(hx(2),'YColor',[0 0.5
0],'LineWidth',2,'FontSize',16,'Ylim',[0 12000],'YTick',[0 2000
4000 6000 8000 10000 12000],'YTickLabel',[0 2000 4000 6000 8000
10000 12000]);
set(h2,'Color',[0 0.5 0],'LineWidth',2);
set(h1,'Color',[0 0 1],'LineWidth',2);

grid on
set(h2(3),'LineStyle','-.')
set(h2(2),'LineStyle',':')

t1=text(50,0.09,'W (kJ/mol glucose)');
set(t1,'FontSize',16,'Color',[0 0.5 0]);
```

```
t2=text(50,0.16,'Exergy destruction(kJ/mol glucose)');
set(t2,'FontSize',16,'Color',[0 0.5 0]);
t3=text(50,0.49,'Entropy generation (J/K)/(mol glucose)');
set(t3,'FontSize',16,'Color',[0 0.5 0]);
t4=text(50,0.34,'2nd law efficiency');
set(t4,'FontSize',16,'Color',[0 0.5 0]);
ylabel('2nd Law Efficiency');
xlabel('Age (Years)');
%title('Path 2')
```

APPENDIX E: THE CHANGE OF GLUCOSE CONSUMPTION RATE WITH EFFICIENCY AND HEARTBEAT RATE IN THE HEALTHY AND ENLARGED LEFT AND RIGHT VENTRICLES IMPLEMENTED IN MATLAB PROGRAMMING LANGUAGE

M-file

```
clear all
close all

eff(1:7)= [0.17 0.22 0.26 0.3 0.34 0.4 0.42];
hr=40:5:140;
for i=1:7
    for j=1:21
        mglu(i,j)=((9.607E-04/eff(i))/3868)*1000*hr(j);
        Vglu(i,j)=mglu(i,j)/0.25;
    end
end

surf(hr,eff,mglu); hold on

eff(1:7)= [0.17 0.22 0.26 0.3 0.34 0.4 0.42];
hr=40:5:140;
for i=1:7
    for j=1:21
        mglu(i,j)=((2.812E-04/eff(i))/3868)*1000*hr(j);
        Vglu(i,j)=mglu(i,j)/0.25;
    end
end

surf(hr,eff,mglu); hold on
eff(1:7)= [0.17 0.22 0.26 0.3 0.34 0.4 0.42];
hr=40:5:140;
for i=1:7
    for j=1:21
        mglu(i,j)=((1.969E-04/eff(i))/3868)*1000*hr(j);
        Vglu(i,j)=mglu(i,j)/0.25;
    end
```

```
end

surf(hr,eff,mglu); hold on

eff(1:7)= [0.17 0.22 0.26 0.3 0.34 0.4 0.42];
hr=40:5:140;
for i=1:7
    for j=1:21
        mglu(i,j)=((7.23E-05/eff(i))/3868)*1000*hr(j);
        Vglu(i,j)=mglu(i,j)/0.25;
    end
end

surf(hr,eff,mglu); hold on

colormap cool
xlabel('Heart rate','fontsize',12,'fontweight','bold')
ylabel(' Efficiency','fontsize',12,'fontweight','bold')
zlabel('Glucose Consumed
(mmol/min)','fontsize',12,'fontweight','bold')
grid on
set(gca,'GridLineStyle','-')
```

APPENDIX F: THE CHANGE OF GLUCOSE CONSUMPTION RATE WITH EFFICIENCY AND ENTROPY GENERATION RATE IN THE HEALTHY AND ENLARGED LEFT AND RIGHT VENTRICLES IMPLEMENTED IN MATLAB PROGRAMMING LANGUAGE

M-file

```
clear all
close all

eff(1:7)= [0.17 0.22 0.26 0.3 0.34 0.4 0.42];
entropy=[9.23e-7.*75 6.69e-7.*75 5.37e-7.*75 4.41e-7.*75 3.68e-
7.*75 2.85e-7.*75 2.63e-7.*75]; for i=1:7
    for j=1:7
        mglu(i,j)=((9.607E-04/eff(i))/3868)*1000*75;
        Vglu(i,j)=mglu(i,j)/0.25;
    end
end

surf(entropy,eff,mglu); hold on

eff(1:7)= [0.17 0.22 0.26 0.3 0.34 0.4 0.42];
entropy= [9.23e-7.*75 6.69e-7.*75 5.37e-7.*75 4.41e-7.*75 3.68e-
7.*75 2.85e-7.*75 2.63e-7.*75];
for i=1:7
    for j=1:7
        mglu(i,j)=((2.812E-04/eff(i))/3868)*1000*75;
        Vglu(i,j)=mglu(i,j)/0.25;
    end
end
xlim([2e-5,7e-5])
surf(entropy,eff,mglu); hold on
eff(1:7)= [0.17 0.22 0.26 0.3 0.34 0.4 0.42];
entropy= [9.23e-7.*75 6.69e-7.*75 5.37e-7.*75 4.41e-7.*75 3.68e-
7.*75 2.85e-7.*75 2.63e-7.*75
for i=1:7
    for j=1:7
        mglu(i,j)=((1.969E-04/eff(i))/3868)*1000*75;
```

```
        Vglu(i,j)=mglu(i,j)/0.25;
    end
end

surf(entropy,eff,mglu); hold on
eff(1:7)= [0.17 0.22 0.26 0.3 0.34 0.4 0.42];
entropy= [9.23e-7.*75 6.69e-7.*75 5.37e-7.*75 4.41e-7.*75 3.68e-
7.*75 2.85e-7.*75 2.63e-7.*75for i=1:7
    for j=1:7
        mglu(i,j)=((7.23E-05/eff(i))/3868)*1000*75;
        Vglu(i,j)=mglu(i,j)/0.25;
    end
end

surf(entropy,eff,mglu); hold on

colormap cool

xlabel(' Entropy generation
(kJ/K)','fontsize',12,'fontweight','bold')
ylabel(' Efficiency','fontsize',12,'fontweight','bold')
zlabel('Glucose consumed
(mmol/min)','fontsize',12,'fontweight','bold')
grid on
set(gca,'GridLineStyle','-')
```