Abstract—The second law muscle work efficiency is obtained by multiplying the metabolic and mechanical work efficiencies. Thermodynamic analyses are carried out with 19 sets of arms and legs exercise data which were obtained from the healthy young people. These data are used to simulate the changes occurring during aging. The muscle work efficiency decreases with aging as a result of the reduction of the metabolic energy generation in the mitochondria. The reduction of the mitochondrial energy efficiency makes it difficult to carry out the maintenance of the muscle tissue, which in turn causes a decline of the muscle work efficiency. When the muscle attempts to produce more work, entropy generation and exergy destruction increase. Increasing exergy destruction may be regarded as the result of the deterioration of the muscles. When the energetic efficiency is 0.42, exergy destruction becomes 1.49 folds of the work performance. This proportionality becomes 2.50 and 5.21 folds when the exergetic efficiency decreases to 0.30 and 0.17 respectively.

Keywords—Aging mitochondria, entropy generation, exergy destruction, muscle work performance, second law efficiency.

I. INTRODUCTION

The biological systems may be considered as open systems living under near steady state conditions. Living organisms transform chemical energy of the nutrients to other forms of biomolecules. During this process, heat is generated as a byproduct and dissipated to the surroundings via generating entropy on the basis of the second law of thermodynamics [1].

Thermodynamic analyses test the feasibility of processes. First law of thermodynamics states that energy can neither be created nor destroyed, but converted from one form to another or transferred due to mass, heat and work transfer. The second law of thermodynamics defines the entropy to account for the losses, e.g., dissipation of the energy, involved in a processes because of internal irreversibility via entropy production [1]. The muscle work efficiency is the determining factor of the entropy generation rate progresses with the increase of the exercise level.


Skeletal muscle may be regarded as a thermodynamic engine capable of converting chemical energy into mechanical work. The major functional capacity of a muscle is its capability to shorten its length against a load and as a result to do work [11].

The mechanical energy generated by a skeletal muscle is derived from energy released by the hydrolysis of adenosine triphosphate (ATP). A large amount of the energy derived from ATP is not converted into work, but released as heat [12].

As stated by [13], a promising area for the application of exergy analysis is the assessment of living organisms to resolve their exergy behavior aiming for a well understanding of living and aging processes from a thermodynamic approach, helping forecast their long term behaviors under altered situations. Muscle work efficiency during exercising depends on the quality of the work performed, strength, and style of muscle work, blood pressure, heart rate and additional issues. Skeletal muscle anomalies limit the aerobic capacity for the duration of the exercise [14].

There is a critical effect of mitochondria aging in the aging process, since it produces most of the energy, e.g., ATP, needed for the activities of the cells. The loss of skeletal muscle protein mass during aging is among the reasons of the reduction of the number of mitochondria in the muscle cells [15]. Skeletal muscle mitochondrial dysfunction leads to metabolic disturbances, especially in elder individuals [16]. These disturbances lead to changes with cellular dynamics and are related with loss of muscle quality as well as increases in oxidative stress.

Even though skeletal muscle work efficiency possibly plays a key role in restricting movement of the elderly people, the physiological mechanisms responsible for this reduction is not completely understood. In the present study, we will perform thermodynamic analysis of the important energetic mechanisms that may change during aging and try to
understand how the mechanical and thermodynamic efficiencies may change.

II. METHODOLOGY

In order to calculate the glucose consumption, exergy destruction and entropy generation; mass, energy, exergy and entropy balances were performed around the arm and the leg muscles.

![Fig. 1 The schematic description of the muscle contraction process](image)

**A. Energy Balance**

Energy balance around the muscle system (Fig. 1) requires:

\[
Q - W + \sum_i (m_i h)_i - \sum_i (m_i h)_o = \Delta E = 0
\]

(1)

where \(i=1, 2, 3\) and 4 refer to glucose, oxygen, carbon dioxide and water, respectively. During contracting under the steady state conditions \(\Delta E = 0\), and we used the data from the literature about the arm and leg work. We calculated the heat released during work performance from the muscle after substituting the enthalpy of formation and the mass exchanges from [9], which lists the thermodynamic data of each species. Chemical composition, mass, heat capacity, enthalpy of formation, absolute entropy and chemical exergy of the constituents entering and leaving through the system boundaries are given in [9].

![Fig. 2 Schematic diagram of the ATP generation and work performance in the muscle](image)

**B. Exergy Destroyed in the Blood Stream**

Exergy destroyed in the blood stream is calculated from (2):

\[
Ex_{destroyed} = Q \left(1 - \frac{T_0}{T}\right) - W + \sum_i (mex)_i - \sum_i (mex)_o
\]

(2)

Here \(T\) is the temperature at the boundary of the muscle, and \(T_0\) is the reference temperature. The specific exergy of the species is calculated based on their chemical composition and thermophysical state:

\[
ex = ex_{cb} + h - T_0 s - \sum x_i \mu_i^0
\]

(3)

where, \(\mu_i^0\) is the chemical potential of the pure species and \(Ex_{destroyed}\) may be regarded as the exergy lost in the blood stream, and equals to (entropy accumulation in the blood stream) \(x\) (the body temperature). Here \(T = T_w\) is the temperature of the boundary of the muscle system, and \(T_0 = T_{out}\) is the reference temperature. Exergy of formation of each chemical is listed in [9], when \(T_0 = T_{out} = 298\) K, and \(T = T_w = 310\) K. The amount of heat released during the working process from the muscle and removed by the blood stream is calculated, and then the exergy lost in the bloodstream is calculated (Fig. 2) [9].

**C. Entropy Balance**

The entropy generation is calculated as:

\[
s_{gen} = \frac{Ex_{destroyed}}{T_0}
\]

(4)

The work is performed by the muscles by consuming ATP produced in the metabolic pathways; heat generation is an inevitable part of the process and equals to about 2/3 of the enthalpy change associated with conversion of glucose to metabolic end products and a cause of the entropy accumulation. After assuming that heat transfer from blood to the muscle occurs at 310 K, and from the muscle to air at 298 K, and assuming and substituting the exergy destroyed and the muscle cell temperature we may calculate \(s_{gen}\) (J/K) for the muscle.

Work done by the muscles is adapted from the data given in the literature [17]-[21]. The muscle cells produce work by consuming the internal energy of the nutrients which are carried to the reaction site by the O2 rich blood. The present study is limited with the consumption of the carbohydrate in form of glucose in the muscle cells to produce ATP, which is then dissociated into adenosine diphosphate (ADP) to reposition the myosin head, to achieve the relaxation of the actin myosin complex following the contraction of the muscle fibers [22].

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 employed. For example, 30 to 38 moles of ATP are 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:

\[
C_6H_{12}O_6 \rightarrow 30 \text{ADP} + 30 \text{P} \rightarrow 6 \text{CO}_2 + 6 \text{H}_2\text{O}
\] (5)

Similarly, work production can be represented as:

\[
\text{ATP} \xrightarrow{\text{muscle contraction}} \text{ADP} + P_i
\] (6)

Pioneering work in the field of muscle work was performed by [23], who experimentally showed that both force and the heat released during the contraction of a muscle could be written as a function of the contraction velocity.

The efficiency of muscle contraction quantifies the external work obtained from the input chemical energy [5]. Muscular work takes place by contraction and shortening against a load with the utilization of metabolic energy [24]. The first law efficiency, which is also called the mechanical or thermodynamic efficiency, is defined as:

\[
\eta_I = \frac{W}{\Delta H_{\text{glycolysis}}}
\] (7)

Recent studies have shown that the heat released during glycolysis and ATP hydrolysis may vary depending on metabolic conditions such as Ca2+ concentration [9]. The measured heat release varies between 879 to 4017 kJ for each mole of consumed glucose [9].

The first law is valid even in irreversible processes, where energy is still conserved; the second law states that something is lost, and unrecoverable [25]. The second law efficiency is defined as the ratio of the actually produced work performed to the maximum available work:

\[
\eta_{II} = \frac{W}{W_{\text{max}}} = \frac{W}{\Delta G_{\text{glycolysis}}}
\] (8)

Outcomes of the muscle work efficiency research are vastly valuable for diversity of disciplines for example rehabilitation, sports biomechanics, ergonomics, etc., and have broad use [26]. 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. If the energetic requirements surpass the capacity to provide energy, the level of activity cannot be maintained and the exercising muscles develop fatigue. An essential issue in the equilibrium between the rate of energy expenditure and the rate at which energy can be provided is the efficiency with which muscles can transform chemical energy into mechanical energy [27].

### III. RESULTS AND DISCUSSION

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 in the literature [24]. References [9] and [10] reported efficiencies within these ranges for the human muscular systems. Equation (9) was used to calculate the mass of the glucose consumed in the muscle 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 (8):

\[
m_{\text{glu cons}} = \frac{W}{\eta_{II}} \frac{180.16}{3.86} \frac{\text{kg}}{\text{kmoI}}
\] (9)

Numerical values of the work performed, glucose consumed, exergy destroyed and entropy generated for the given second law efficiencies of \(\eta_{II}=0.17\), \(\eta_{II}=0.3\) and 0.42 are listed in Tables I-III. While doing the same amount muscle work, decreases are observed in glucose consumption, exergy destruction and entropy generation with the increase of the 2nd law efficiency. These results imply that as the efficiency increases, a larger fraction of the chemical exergy of glucose is converted into work.

![Fig. 3 Exergy destruction as a function of the 2nd law efficiency in exercise under 100 J work load](image)

**TABLE I**

<table>
<thead>
<tr>
<th>Work (J)</th>
<th>(\eta_{II})</th>
<th>Glucose consumed (mmol/min)</th>
<th>Exergy destroyed (J)</th>
<th>Entropy generation (J/K)</th>
</tr>
</thead>
<tbody>
<tr>
<td>122</td>
<td>(\eta_{II}=0.17)</td>
<td>11.13</td>
<td>636</td>
<td>2.134</td>
</tr>
<tr>
<td>(\eta_{II}=0.30)</td>
<td>6.308</td>
<td>305.5</td>
<td>1.025</td>
<td></td>
</tr>
<tr>
<td>(\eta_{II}=0.42)</td>
<td>4.505</td>
<td>182</td>
<td>0.610</td>
<td></td>
</tr>
<tr>
<td>(\eta_{II}=0.17)</td>
<td>2.737</td>
<td>156.4</td>
<td>0.524</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>(\eta_{II}=0.30)</td>
<td>1.551</td>
<td>75.12</td>
<td>0.252</td>
</tr>
<tr>
<td>(\eta_{II}=0.42)</td>
<td>1.107</td>
<td>44.75</td>
<td>0.150</td>
<td></td>
</tr>
<tr>
<td>(\eta_{II}=0.17)</td>
<td>5.474</td>
<td>312.8</td>
<td>1.049</td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>(\eta_{II}=0.30)</td>
<td>3.102</td>
<td>150.2</td>
<td>0.504</td>
</tr>
<tr>
<td>(\eta_{II}=0.42)</td>
<td>2.215</td>
<td>89.51</td>
<td>0.300</td>
<td></td>
</tr>
<tr>
<td>(\eta_{II}=0.17)</td>
<td>8.212</td>
<td>469.2</td>
<td>1.574</td>
<td></td>
</tr>
<tr>
<td>90</td>
<td>(\eta_{II}=0.30)</td>
<td>4.653</td>
<td>225.3</td>
<td>0.736</td>
</tr>
<tr>
<td>(\eta_{II}=0.42)</td>
<td>3.323</td>
<td>134.2</td>
<td>0.450</td>
<td></td>
</tr>
</tbody>
</table>
Some recent studies show that the elderly adults utilize more energy than their younger counterparts to do the same activity due to their lower muscle work performance efficiency [28], [29]. Results of this study show that this observation is caused by higher exergy loss and entropy generation at lower second law muscle work performance efficiency, e.g. the situation prevailing in the muscles of elderly people. These results may also imply that young individuals who improve their muscle work efficiency by exercising may experience less exergy loss and less entropy generation in their muscles. Older adults usually show low levels of exercise efficiency [30].

The muscle efficiency decreases with the age as a consequence of increasing exergy destruction and entropy generation. In humans, cardiac output and stroke volume at maximal exercise and maximal work load capacity decrease in aged subjects [31].

Aging is related to entropy accumulation in the body [32]. Therefore, as time passes by, the aged muscle cell becomes different from that of a younger person’s cell [33]. The maximal capacity of the body to perform work may also be diminish by aging. Systematic aerobic exercise can increase the life span [34]. Thus, a connection between the positive influences of exercise and changes in ATP supply-to-demand mechanisms may occur in aging human. Reference [35] argues that the age-related increase in the levels of oxidative stress and damage on the mitochondrial biomolecules becomes progressively more apparent with aging. A decline in mitochondrial function may affect the production of the cellular energy, which in turn can restrict ATP dependent events in the cell.

While there is a wide-ranging of data proposing that mitochondrial energetic mechanisms decrease in advanced age, the relative importance of these alterations is being still argued. When mitochondria are more coupled, ATP is generated at a slower rate [36] that could be insufficient to encounter cellular energy needs, particularly during skeletal muscle contraction. In fact, in the older individuals it has been found that a lower speed of ATP generation is related with higher fatigability [37].

### Table II

**LEG EXERCISE- NUMERICAL VALUES OF THE GLUCOSE CONSUMPTION RATE, EXERGY DESTROYED AND THE ENTROPY GENERATED IN THE WORKING MUSCLES OF A PERSON DUE TO 2ND LAW EFFICIENCY RANGE AT GIVEN WORK DONE VALUES**

<table>
<thead>
<tr>
<th>Work (J)</th>
<th>ηII</th>
<th>Glucose consumed (mmol/min)</th>
<th>Exergy destroyed (J)</th>
<th>Entropy generation (J/K)</th>
</tr>
</thead>
<tbody>
<tr>
<td>207</td>
<td>0.17</td>
<td>18.88</td>
<td>1079</td>
<td>3.621</td>
</tr>
<tr>
<td>100</td>
<td>0.30</td>
<td>5.170</td>
<td>250.4</td>
<td>0.840</td>
</tr>
<tr>
<td>150</td>
<td>0.30</td>
<td>7.755</td>
<td>375.6</td>
<td>1.260</td>
</tr>
<tr>
<td>238</td>
<td>0.30</td>
<td>12.30</td>
<td>595.9</td>
<td>1.999</td>
</tr>
<tr>
<td>65</td>
<td>0.30</td>
<td>8.790</td>
<td>355</td>
<td>1.191</td>
</tr>
<tr>
<td>33</td>
<td>0.30</td>
<td>1.070</td>
<td>82.63</td>
<td>0.277</td>
</tr>
<tr>
<td>20</td>
<td>0.30</td>
<td>1.034</td>
<td>50.08</td>
<td>0.168</td>
</tr>
<tr>
<td>40</td>
<td>0.30</td>
<td>2.068</td>
<td>100.1</td>
<td>0.336</td>
</tr>
<tr>
<td>20</td>
<td>0.30</td>
<td>1.034</td>
<td>50.08</td>
<td>0.168</td>
</tr>
<tr>
<td>40</td>
<td>0.30</td>
<td>2.068</td>
<td>100.1</td>
<td>0.336</td>
</tr>
</tbody>
</table>

### Table III

**COMBINED ARM AND LEG EXERCISE- NUMERICAL VALUES OF THE GLUCOSE CONSUMPTION RATE, EXERGY DESTROYED AND THE ENTROPY GENERATED IN THE WORKING MUSCLES OF A PERSON DUE TO 2ND LAW EFFICIENCY RANGE AT GIVEN WORK DONE VALUES**

<table>
<thead>
<tr>
<th>Work (J)</th>
<th>ηII</th>
<th>Glucose consumed (mmol/min)</th>
<th>Exergy destroyed (J)</th>
<th>Entropy generation (J/K)</th>
</tr>
</thead>
<tbody>
<tr>
<td>329</td>
<td>0.17</td>
<td>17.01</td>
<td>823.8</td>
<td>2.764</td>
</tr>
<tr>
<td>387</td>
<td>0.30</td>
<td>20.01</td>
<td>969.1</td>
<td>3.252</td>
</tr>
<tr>
<td>200</td>
<td>0.30</td>
<td>10.34</td>
<td>500.8</td>
<td>1.680</td>
</tr>
</tbody>
</table>

### IV. Conclusion

The output of metabolic and mechanical work efficiency is the definition of the second law muscle work efficiency.
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. Mitochondrial depletion of metabolic energy production with aging reduces muscle work efficiency. The reduction of the mitochondrial energy generation causes deterioration of the muscle structure, which causes a decline in the muscle work efficiency. As a result in response to interventions for high work production in aging muscle systems, exergy loss and entropy production in the muscles increase.

In summary; the changes occurring during skeletal muscle aging are:

1. Exercise efficiency decreases with aging
2. Mitochondrial content of the cellular energy generation declines with aging
3. The cellular energy generation, e.g., the ATP production rate, decreases and ATP dependent cellular physiological activity is restricted
4. Maximal contractility of the muscles decreases and skeletal muscle cells lose their capacity to adapt to physiological stress imposed by aging
5. Skeletal muscle protein mass decreases, muscle performance decreases and muscle work efficiency attained a lower level
6. More energy is utilized to achieve the same muscular activity in comparison with a young person
7. Skeletal muscle mitochondrial dysfunction leads to metabolic disturbances
8. Muscle quality and work efficiency has declined

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