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How does obesity affect bioenergetics in human respiratory muscles?

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ABSTRACT

Background: Obesity prevalence is increasing worldwide, and it causes restrictions in the respiratory system. Bioenergetics is known as energy management in a living cell. Respiratory muscle can be considered a thermodynamic machine that converts chemical energy into mechanical work during each breathing cycle.
Objective: This research aimed to reveal glucose consumption, exergy destruction, and entropy generation in healthy, obese, and obesity hypoventilation syndrome (OHS) patients using thermodynamic analysis of the work of breathing.
Research methods & procedures: A human respiratory system was modeled thermodynamically for healthy, obese, and OHS patients. The systems were analyzed by applying the first and second laws of thermodynamics.
Results: Our simulations show that obese and OHS patients consume approximately 5 and 8 times more glucose, respectively, than their healthy counterparts: 0.2 mmol/min for healthy, 1.06 mmol/min for obese, and 1.59 mmol/min for OHS. Exergy destruction values for the healthy, obese, and OHS models were calculated as 6.41×10^{-3} (kJ/min), 4.85×10^{-2} (kJ/min), and 6.16×10^{-2} (kJ/min), respectively. Entropy generation values of the healthy, obese, and OHS models throughout the breathing cycle were 2.15×10^{-5} (kJ/K)/min, 1.63×10^{-4} (kJ/K)/min, and 2.07×10^{-4} (kJ/K)/min, respectively. Thermodynamic balances and calculations were performed to simulate the bioenergetic changes happening during breathing.
Conclusions: The obese and OHS models had significantly increased glucose consumption, exergy destruction, and entropy generation than the healthy model. The results point to the fact that respiratory performance is associated with obesity and excessive food consumption damages the respiratory system.

1. Introduction

Obesity is commonly regarded as a global fact that decreases life expectancy and increases morbidity. Obesity prevalence is increasing worldwide, and it causes restrictions in the respiratory system. Obesity hypoventilation syndrome (OHS) defines the combination of severe obesity and hypoventilation. Obese people without OHS are assumed to have uncomplicated or simple obesity [1]. Obesity may have substantial adverse effects on the respiratory system and adversely affect respiratory physiology. The obese person has considerably increased oxygen consumption, carbon dioxide production, and a damaged ventilation system. The leading respiratory problems of obesity involve an increased demand for ventilation, increased work of breathing, reduced respiratory compliance, and inefficiency of respiratory muscles. Obesity affects respiratory function both at rest and during exercise. Decreases in respiratory compliance, functional residual capacity, impaired respiratory mechanics, and expiratory reserve volume create a restrictive ventilatory defect [2–4].

Bioenergetics is known as energy administration in a living cell. Thermodynamic analyses are used to quantify the relative importance of different processes. The first law of thermodynamics declares that energy can be transformed from one form to another or transported through mass, heat, and work transfer, but can neither be created nor destroyed. The second law of thermodynamics explains entropy and is used to measure energy losses. Entropy is a measure of the randomness of a system [5–8]. Exergy is the maximum useful energy and is destroyed in any irreversible process resulting from entropy generation. The expression of exergy destruction determines the loss of useable energy caused by entropy generation. The generation of entropy in any irreversible process leads to the destruction of exergy [9].

Muscle contraction in biology is a prominent subject of thermodynamics. The respiratory muscle can be considered a thermodynamic machine, such as a piston, that converts chemical energy into

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Nomenclature

ex	Specific exergy kJ/kg
Ex	Total exergy kJ
h	Specific enthalpy kJ/kg
H	Total enthalpy kJ
m	Mass kg
Q	Heat kJ
s_{gen}	Entropy generation kJ/K
T	Temperature K
W	Work kJ
ΔE	Energy change kJ
η_{II}	The second law efficiency Fraction or percentage
μ	Chemical potential kJ/kmol
Subscripts	
0	Restricted dead state
ch	Chemical
gen	Generation
i	Any species
in	Inlet
out	Outlet

mechanical work during each breathing cycle [10]. In each respiratory cycle, the respiratory system acts as a thermal engine, in which the energy used is transformed into work by the respiratory muscles. Heat is generated as a by-product and is dissipated to the environment by generating entropy in each breathing cycle based on the second law of thermodynamics. Tissue function randomness rises due to entropy accumulation in this periodic process, which may lead to a reduction in work efficiency over time and finally, may lead to damage in the respiratory muscle. The second law efficiency for muscle work is a product of metabolic and mechanical work efficiencies [10,11].

The work of breathing (W) is the total consumption of energy required to achieve the action of breathing. Respiratory muscle W efficiency during breathing relies on the quality of the W performed, strength, blood pressure, heart rate, and additional issues [12,13]. Obese and OHS patients have decreases in total respiratory compliance and increases in total W.

In obese and OHS subjects, breathing efficiency is similar and can be nearly half the average level [1,14]. The increased W and oxygen consumption by respiratory muscles leads the respiratory muscles in obese people to become tired and to have reduced strength. Although respiratory muscle W efficiency plays a central role in restricting the breathing of obese patients, the physiological mechanisms responsible for this reduction have not been fully elucidated. Moreover, the effects of obesity on mortality in COPD (chronic obstructive pulmonary disease) and heart failure have not been completely resolved [3,15–17].

Energy losses leading to reduced respiratory W efficiency can be calculated by thermodynamic analysis of the respiratory muscles. Thus, the losses because of irreversibility in each breathing cycle in the system and the sources of these losses can be identified numerically.

In recent years, several studies have been published based on the thermodynamic analysis of living systems. However, there are a limited number of studies on respiratory thermodynamics [10,13,18–20]. It has been determined from the literature survey that no research has been performed regarding the respiratory thermodynamics in obesity. Therefore, this study was carried out to determine the thermodynamic changes in respiratory mechanics and to characterize the structure of damaged respiratory mechanics thermodynamically in obese patients.

From the thermodynamic viewpoint, the human respiratory system may be considered an energy exchange device. The available energy (exergy) transfer for continuing breathing activity depends on physiological, ambient, and individual components (i.e., the heart, the lungs, blood vessels, and blood). In this work, the authors are interested in observing the effect of physiological conditions, inspiratory air conditions, and individual component contribution on the performance of the respiratory system for healthy, obese, and OHS patients.

To that end, we performed a thermodynamic analysis of the key energetic mechanisms that may be altered in obesity and attempted to realize how mechanical and thermodynamic behaviors may be altered. The aim of this study is to determine the glucose consumption, exergy destruction, and entropy generation in healthy, obese, and OHS subjects using a thermodynamic analysis of the W. This research consisted of proposing a human thermal system model to represent healthy, obese, and OHS respiration, given their physiological differences. Employing thermal analysis permitted observing different anatomies' responses to typical breathing conditions.

2. Materials and methods

In this study, a human respiratory system was modeled for healthy, obese, and OHS respiratory muscle. The first and second laws of thermodynamics were used to analyze the systems. For the first law of thermodynamics, the mass balance was performed using the energy balance equation, which allowed us to measure glucose consumption by the respiratory muscles. The glucose consumed by the respiratory muscles varied by the second law efficiency. Applying the second law of thermodynamics, entropy generation was calculated, which allowed us to measure energy losses. The equations used in the thermodynamic model are given in Table 1. Energy, exergy, and entropy equilibriums were applied around the respiratory muscle systems as a function of the second law efficiency (η_{II}) to calculate the glucose consumption, exergy destruction, and entropy generation.

The W data was adapted from the literature [1], which had been determined under resting conditions. The W may be computed concerning the oxygen cost of breathing or the pulmonary pressure manifold by the exchange in pulmonary volume. It is measured in joules/L, joules/min, and sometimes kg/m/min. In respiratory physiology, the measurement of the W is work = pressure x volume and is similar to the usual definition of work in physical science (work = force x distance). The W values range from 2.4 to 7.5 J/min and from 0.2 to 0.9 J/L in healthy subjects at rest and account for 5% of total body oxygen consumption in a healthy resting state but can increase significantly during acute illness [12,21].

The ventilatory parameters [22], breathing efficiency, second law efficiency [23], and the W in the healthy, obese, and OHS models are given in Table 2. The amounts of glucose consumption, exergy destruction, and entropy generation were calculated for the healthy, obese, and OHS model subjects using the second law efficiency as 0.42,

Table 1
Equations used in the thermodynamic model.

Equation		Equation number
Energy (Mass) balance	$Q - W + \sum_i (mh)_{in} - \sum_i (mh)_{out} = \Delta E = 0$	(1)
Exergy destruction	$Ex_{destroyesd} = Q\left(1 - \frac{T_0}{T}\right) - W + (mex)_{in} - (mex)_{out}$	(2)
Specific exergy	$ex = ex_{ch} + h - T_0 s - \sum x_i \mu_i^0$	(3)
Entropy generation	$s_{gen} = \frac{Ex_{destroyed}}{T_0}$	(4)
Glycolysis	$C_6H_{12}O_6 \xrightarrow{30ADP+30P \rightarrow 30ATP} 6CO_2 + 6H_2O$	(5)
W production	$ATP \xrightarrow{musclecont raction} ADP + P_I$	(6)
First Law Efficiency	$\eta_I = \frac{W}{\Delta H_{glycolysis}}$	(7)
Second Law Efficiency	$\eta_{II} = \frac{W}{W_{\max}} = \frac{W}{\Delta G_{glycolysis}}$	(8)

Table 2
Ventilatory parameters, breathing efficiency, second law efficiency, and the work of breathing in healthy, obese, and OHS subjects.

Variable	Unit	Healthy	Obese	OHS
RR [1]	Breaths/min	13.0	17.0	20.0
VE [22]	L/min	9.9	12.6	15.00*
Mechanical Efficiency [1]	%	2.1	0.8	1.0
2nd law efficiency [23]	%	0.42	0.17	0.30
Work [1]	J/L	0.43	0.74	1.64
W*	kJ/min	4.3×10^{-3}	9.3×10^{-3}	24.6×10^{-3}

RR, respiratory rate; VE, minute ventilation; W, work of breathing per minute; *, calculated.

0.17, and 0.30, respectively.

The experimental results performed for the first law efficiencies with animals such as mice and frogs are reported in the range of 0.14 and 0.35, while the second law efficiencies are in the range of 0.17 and 0.42, respectively [6,23]. Since specific data on the respiratory muscles are not available, the above-described efficiencies were assumed to be similarly applicable for the respiratory muscles and were utilized in calculations. In this thermodynamic model, the surrounding air condition was taken as 25 °C and the body temperature as 37 °C. The study is limited to the consumption of glucose for producing ATP in the respiratory system's muscle cells. Only aerobic respiration has been considered in this work.

2.1. Mathematical model

Respiration is the generation of ATP (adenosine 5'-triphosphate) energy by the chemical degradation of nutrients in different ways, taken from foodstuffs and found in the body. Oxygen and nutrients are transported to cells through the bloodstream. The nutrients carried to the cells are burned by oxygen, and consequently, the energy state created by the combination of nutrients and oxygen causes respiration [24].

Work done by the respiratory muscles using ATP (adenosine triphosphate) is produced in the metabolic pathways (Fig. 1). ATP is broken down into ADP (adenosine diphosphate) to relax the actin-myosin complex following the contraction of muscle fibers [25]. The conversion of glucose to the metabolic end-products generates heat, which equals nearly 2/3 of the enthalpy change and is a cause of the entropy accumulation.

2.1.1. Energy balance

In the energy balance equation (Equation 1) around the respiratory muscle system, i=1, 2, 3, and 4 correspond to mean glucose ($C_6H_{12}O_6$), oxygen (O_2), carbon dioxide (CO_2), and water (H_2O), respectively. Enthalpy of formation (ΔE) = 0 during respiratory muscle contraction under steady-state conditions. After substituting the ΔE, the heat released during the W from the respiratory muscle was calculated. We calculated the mass exchanges following Çatak et al. (2015) [6] that lists the thermodynamic properties and data of each constituent (absolute entropy, chemical composition, chemical exergy, enthalpy of formation, heat capacity, and mass) entering and leaving through the muscle system boundaries.

2.1.2. Exergy destruction

Exergy destroyed (Equation 2) in the blood is calculated following Dinçer and Çengel (2001) [5]. T_0 is the reference temperature and T is the boundary temperature of the respiratory muscle system. Based on their thermophysical state and chemical composition, the specific exergy of the constituents is calculated from Equation 3, where, μ_i^0 is the pure species' chemical potential and $Ex_{destroyed}$ refers to the exergy loss

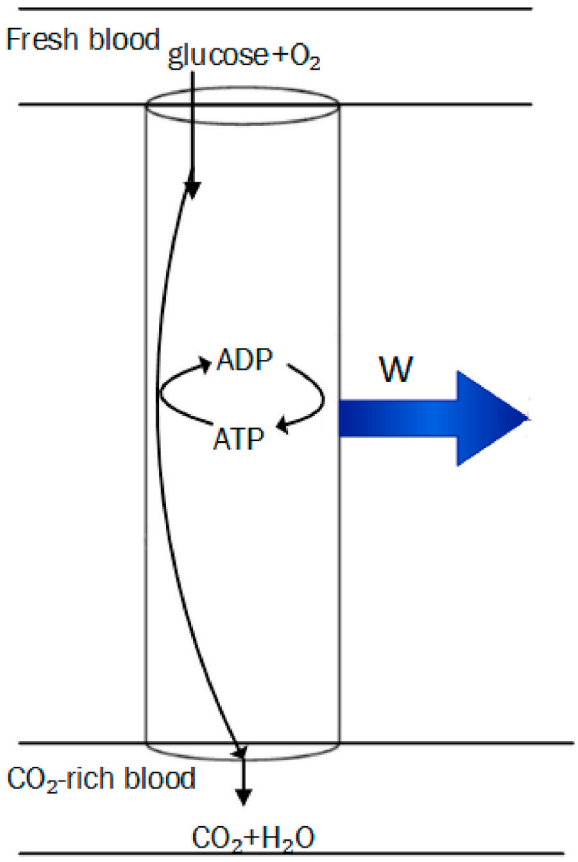

Fig. 1. The schematic description of the muscle contraction process.

in the bloodstream, which equals (entropy accumulation in the bloodstream) x (body temperature). $T_0 = T_{out}$ is the reference temperature and $T = T_{in}$ is the boundary temperature of the respiratory muscle system. In this analysis, it has been assumed that the respiratory system is at a fixed temperature of 37 °C. The exergy of formation is given in Çatak et al. (2015) [6] for each chemical species when $T_0 = T_{out} = 298$ K and $T = T_{in} = 310$ K. Initially, the heat released from the respiratory muscle during the W transferred by the bloodstream was calculated. Later, the exergy loss in the blood was calculated.

2.1.3. Entropy generation

We calculated the entropy generation (s_{gen}) (J/K) using Equation 4, assuming transference of heat from the bloodstream to the respiratory muscle at 37 °C (310 K) and from the respiratory muscle to the air at 25 °C (298 K). After substituting the value of exergy destroyed and the respiratory muscle temperature, the entropy generated was calculated.

The respiratory muscle produces the W by the consumption of the internal energy of the nutrients. The number of ATP moles produced depends on the kind of nutrients and the metabolic pathway used as a result of catabolism. For example, during oxidation of glucose, 30 to 38 moles of ATP are formed. The complex energy metabolism may be simplified using one chemical reaction, glycolysis, assuming that only glucose is catabolized, and 30 moles of ATP are produced by the muscle cells (Equation 5). Accordingly, the production of W can be described by Equation 6.

Hill (1938) did a pioneering study regarding the work of muscle [26], which showed that both force and the heat released could be expressed as a function of the contraction velocity during muscle contraction.

2.1.4. Efficiency

For the period of working contraction, biochemical energy in the

form of ATP is transformed into external work [27]. The performance of muscle work is related to metabolic energy consumption, measured as enthalpy, in other words, heat plus work. One of the efficiency measures, the First Law Efficiency, is the ratio of Work done to Enthalpy (ΔH) generated [28]. The First Law Efficiency, also known as Mechanical Efficiency, is described by Equation 7.

On the other hand, the ratio of the produced work to the maximum available work is known as the Second Law Efficiency (Equation 8). Nonetheless, the first law of thermodynamics is valid even in irreversible processes, and energy is still conserved in this process. On the contrary, the second law of thermodynamics says that something has been lost and cannot be repaired. The second law has advanced to determine the efficiency of machines to explain the theoretical limits of machines' efficiency and states that no machine is fully efficient. Some available energy is lost as heat, inner reorganization of chemical complexes, and other fluctuations in entropy.

3. Results and discussion

The values of the glucose consumed, exergy destroyed, and entropy generated in the respiratory muscles of the healthy, obese, and OHS modeled patients were calculated using the second law efficiency (Equation 8) and given in Table 3.

The mass of glucose consumed by the model respiratory muscle was calculated for the W values for the subjects. Along with the first law of thermodynamics performing energy balance analysis, the glucose consumed for the W of healthy, obese, and OHS models was calculated as 0.20 mmol/min, 1.06 mmol/min, and 1.59 mmol/min, respectively. It was observed that obese and OHS modeled patients consumed approximately 5 and 8 times more glucose, respectively, than healthy counterparts.

As a result of the thermodynamic analysis of the W performed by the healthy, obese, and OHS models, values of the exergy destruction were calculated as 6.41×10^{-3} (kJ/min), 4.85×10^{-2} (kJ/min), and 6.16×10^{-2} (kJ/min), respectively. Entropy generations for healthy, obese, and OHS model simulations through the breathing cycle were 2.15×10^{-5} (kJ/K)/min, 1.63×10^{-4} (kJ/K)/min, and 2.07×10^{-4} (kJ/K)/min, respectively.

The thermodynamic analysis shows that the entropy generation and exergy loss in the respiratory muscles increased in response to higher W production in the obese and OHS respiratory muscle systems. When the W was increased, exergy destruction also increased. Entropy generation increased when the W increased in the respiration of obese and OHS models. Due to entropy accumulation, the randomness of muscle tissue function increases and eventually results in damage to the respiratory muscle [10].

The results reveal that obese and OHS modeled patients consume more energy than their healthy counterparts did to achieve a similar breathing process. These findings may be due to higher exergy destruction and higher entropy generation at lower second law W efficiencies. As long as the efficiency rises, more of the chemical exergy of glucose is transformed into work. Thus, the glucose amount required to obtain a certain amount of W declines as long as the efficiency rises.

Sharp et al. (1964) [15] found that obese patients have decreased total respiratory compliance and increased total W. Obesity is related to various respirational problems like pneumonia, asthma, COPD, pulmonary embolic disease, and obstructive sleep apnea syndrome. Obesity also affects several respiratory physiological parameters, spirometric measures, diffusing capacity and gas exchange, compliance, resistance, neuromuscular strength, lung volume, bronchial hyperactivity, and upper airway mechanical function. These, sequentially, might affect the W, ventilator driving, and exercise capacity and cause sleep-breathing anomalies [4].

In this study, obesity was associated with an increase of entropy in the respiratory system. The production of metabolic heat in the respiratory muscle reflected the generation of entropy. Even though the accumulation of entropy inside the system might be neglected for a single breathing process, a similar assumption is wrong for repeated processes throughout life. Thus, as the body mass increases, respiratory muscle cell structure becomes dissimilar to those from a healthy body mass [29]. Associated changes in body composition establish the basis of many metabolic disorders.

The reaction of food with oxygen produces heat. Since heat production is an irreversible process, it always occurs with entropy production. Therefore, a person generates entropy by consuming foods. Epidemiological findings reveal that specific neurodegenerative disorders are lesser in countries with a low per capita food intake such as Japan than countries with a high per capita food intake such as the United States. Therefore, calorie restriction can stimulate noteworthy alterations in the lessening of acute disorders in relatively obese humans. As a result, in the case of obese people, calorie restriction may increase lifespan [30].

Caloric restriction improves lifespan by raising metabolic stability. McCay et al. (1935) showed that calorie restriction delays aging and prolongs average and maximal lifespan [31,32], but it was later recognized in flies, yeast, and worms as well. Calorie restriction prolongs lifespan by delaying age-associated chronic diseases, decreasing metabolic rate and oxidative stress, and changing neuroendocrine and sympathetic nervous system function in organisms.

Calorie restriction intentionally reduces calorie intake, and this should be differentiated from malnutrition, inappropriate diet, or deficiency. Remarkably, calorie restriction extends the lifespan of several classes, for example, several species of rodents, decreases the level of DNA destruction in aged animals, and proliferates the steady-state mRNA level of various genes included in energy metabolism [33]. The concept that caloric restriction diminishes the rate of aging, extends the period of youth, delays the beginning of age-related pathologies, and prolongs the lifespan of mammals has been a managing theory in gerontology for years. Plentiful postulations have been suggested to clarify the biological foundation of how calorie restriction extends the lifespan of responsive genotypes. The most frequently recorded are delayed growth, increased damage/repair function, decreased body temperature/metabolic rate, changes in IGF/insulin/TOR signaling, decreased immunologic and hormonal variations, variations in gene expression, reduced reactive oxygen species (ROS) generation/oxidative stress, increased autophagy and apoptosis, and decreased body fat [34].

The lifespan entropy theory proposes that living beings might have a limited lifespan entropy generation capacity and die when they reach this limit. In a study by Çatak et al. (2015) [6], the lifespan entropy generation by the masseter muscle, concerning the chewing process of foodstuffs, was associated with the life expectancy of an obese subject. The masseter muscle of a man ingesting foodstuffs according to the

Table 3
Variations of the glucose consumption, exergy destruction, and entropy generation rates in healthy, obese, and OHS modeled subjects using the second law efficiency.

Subject	η_{II}	$m_{glucose}$ (mol/min)	$m_{glucose}$ (mmol/min)	Glucose concentration in blood (mmol/L)	$Ex_{destroyed,muscle}$ (kJ/min)	$S_{gen,muscle}$ (kJ/K)/min
Healthy	0.42	2.64×10^{-6}	0.20	3.97×10^{-2}	6.41×10^{-3}	2.15×10^{-5}
Obese	0.17	1.41×10^{-5}	1.06	2.12×10^{-1}	4.85×10^{-2}	1.63×10^{-4}
OHS	0.30	2.12×10^{-5}	1.59	3.18×10^{-1}	6.16×10^{-2}	2.07×10^{-4}

OHS, obesity hypoventilation syndrome; η_{II}, second law efficiency; $m_{glucose}$, mass of glucose; $Ex_{destroyed,muscle}$, exergy destruction in respiratory muscle; $S_{gen,muscle}$, entropy generation in respiratory muscle.

guidelines of the Institute of Medicine (USA) may generate nearly 1×10^5 J/K of entropy while chewing his foodstuffs throughout his 76 years of expected lifetime. According to the results, the masseter muscles of an obese subject who consumes 10% more food generates a similar entropy nearly 5 years earlier.

Aging may be stated as the increasing deterioration in the functional ability of essential organs. Calorie restriction decreases oxidative damage by diminishing energy fluctuation and thus affects the aging process. Part of this reduction is the result of a decrease in energy consumption and the thermal effect of food, while another part is due to the decreased size of the mass to be metabolized.

Carbohydrates in the form of glucose ($C_6H_{12}O_6$) are the primary fuel of most living things. To estimate the metabolism based on energy and exergy, obtaining the biothermodynamical properties of the energy substrates like enthalpy change (Δh) and Gibbs free energy change (Δg) of oxidation reactions is vital. The first law of thermodynamics is a conservation law that states that the form of energy may alter; however, the total amount is always conserved. On the other hand, the second law is a dissipation law that defines an amount, the entropy (S), that we usually characterize as disorder; equilibrium is not expected. The second law also states that entropy will increase in any irreversible process. It is noteworthy that it is the second law that forces biochemical reactions. The first law tells us that the total energy credited to work, heat, and alterations in biochemical components will be steady. However, it does not tell us whether such a reaction will occur, or what the relative distributions of the forms of energy will be if it does. To determine the tendency of the reaction, we have to use the second law, which says that entropy must increase [35]. The second law of thermodynamics was applied to biological systems for the first time to validate the minimum entropy generation theory or the theory of Prigogine and Wiame (1946). In this view, all organisms tend to have minimum entropy generation. Consequently, the minimum entropy generation theory was confirmed for various species ranging from fish to humans [36]. The study by Sorgüven and Özilgen (2015) supplies an analogy between the Carnot engine and muscle to understand the process of producing muscle work and assess the maximum muscle efficiency in physiological conditions [37].

A significant feature of biological systems is that they flourish on irreversibility. Therefore, they are not in thermal, biochemical, and mechanical balance with their surroundings. To maintain the lifecycle, living beings expend high-energy foods irreversibly, producing heat and entropy. However, entropy is carried to the environment through a series of waste streams that involve sweating and heat transfer through the skin, maintaining the living organism in the settled thermal state [38].

The obtained results from the present study for the human body can be used to assess the quality of the energy conversion processes that take place in its various systems, organs, and even cells. The exergy analysis of the respiratory system is a tool that can present signs of healthiness and quality of life. In the study, mathematical modeling of the respiration process was performed in healthy, obese, and obesity hypoventilation syndrome subjects.

In this study, critical energetic mechanisms were analyzed thermodynamically. The available knowledge about how the obesity-related respiratory disorder process develops is limited in the literature. Due to the abnormal respiratory physiology in obese patients, thermodynamic assessment of the respiratory muscles can provide additional information regarding respiratory disorders. The entropy generation model presents a different and reliable vision in this context. Multidisciplinary research is needed to better understand respiratory disorders. Evaluating the W by respiratory muscle through thermodynamic analysis can provide additional information in determining the work performance related to the respiratory system.

4. Conclusions

In this study, the bioenergetics of breathing in obesity was assessed thermodynamically and expressed numerically. Entropy generation and exergy destruction by the respiratory muscle was associated with the obese person in relation to the respiratory process. The obese and OHS modeled patients had significantly higher glucose consumption, exergy destruction, and entropy generation than the healthy one. The results indicate that respiratory performance is associated with obesity, and excessive food consumption damages the respiratory system. In conclusion, a thermodynamic assessment of the respiratory muscles in the obese population may provide additional information regarding respiratory disorders.

This study highlights the importance of exergy analysis of the respiratory system in the obese for estimating healthiness and quality of life and provides data for the global literature, which consequently unpacks the critical role of accurate bioenergetics data. We hope that these new values will serve as a useful means to assess obesity in the general population.

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Credit author statement

Jale Çatak: Conceptualization, Methodology, Writing- Original draft preparation, Visualization, Formal analysis, Investigation, Data curation, Software, Writing- Reviewing and Editing. Elif Develi: Conceptualization, Methodology, Writing- Original draft preparation, Visualization, Formal analysis, Investigation, Data curation, Software, Writing- Reviewing and Editing. Serkan Bayram: Conceptualization, Methodology, Writing- Original draft preparation, Visualization, Formal analysis, Investigation, Data curation, Software, Writing- Reviewing and Editing.

Declaration of competing interest

There are no conflicts of interest.

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