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Energy Expenditure Measured by Indirect Calorimetry in Obesity

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1. Introduction

Obesity is a multifactorial disease characterized by excessive deposition of fat in adipose tissue, which may be due to excessive energy intake, and or changes in body energy expenditure, resulting in positive energy balance [1].

Mika Horie et al [2] demonstrated that obese women had higher total energy expenditure (TEE), compared with normal weight. However, this increase may be due to increased basal metabolic rate (BMR) due to higher fat-free mass (FFM) and energy demand during physical activity. However, Melo et al [3] found that obese individuals are economical, the metabolic point of view. Therefore, the energy expenditure (EE) per kilogram of body weight at the give time is lower in obese individuals.

The low metabolic rate, expressed relative to FFM seems to be a risk factor for weight gain [4]. In a prospective study in Pima Indians, Ravussin et al. [5] showed that both the low resting metabolic rate (RMR) and low TEE increased risk of weight gain. The basal energy expenditure (BEE) and resting (REE) can be obtained through BMR and RMR, respectively, multiplied by 24 hours (1440 minutes).

There are several methods for the assessment of EE with different levels of precision, including indirect calorimetry, which measures the metabolic rate by the determination of oxygen consumption (O_2) (with a spirometer), the production of carbon dioxide (CO_2) and excretion of urinary nitrogen, for a given period of time [6]. This technique relies on the fact that all the O_2 consumed and CO_2 produced is due to the oxidation of the three major energy substrates, which are fats, carbohydrates and proteins [7].

Recognizing the need to estimate EE in institutions that have no indirect calorimetry, researchers have proposed the use of specific equations, developed from calorimetry studies in groups of individuals with similar clinical characteristics [8]. Although the estimate of EE

is the most common method, the predictive equations might generate errors [9]. Shetty [10] considers that the equations used to estimate the BEE in normal weight adults have reasonable precision (coefficient of variation 8%).

In clinical practice it is impracticable to measure the calorimetric methods for EE, so the international use of the equations was recommended, modified from a compilation of data carried out by Schofield [11]. Studies conducted in different ethnic groups found that these equations provide high BEE estimates, particularly for residents in the tropics [12-14]. Wahrlich and Anjos [14] confer these differences to the fact that equations have been developed mostly from population samples of North America and Europe which show differences in body composition, and live in different environmental conditions.

It is known that in populations with severe obesity is actually more difficult to fit the equations, because there is the difficulty in choosing the weight to be applied in the equation, which may influence a lot the results [15]. The use of current weight leads to the overestimation of the results independent of the equation to be applied, and the use of ideal or adjusted weight can result in the underestimation of energy needs [16].

Considering that obesity is a chronic disease of epidemiological importance, nutritional intervention studies have been developed in order to propose strategies for the prevention and treatment of this disease. The chronic imbalance between energy intake and EE results in positive energy balance and body weight gain. One way of evaluating the influence of dietary components in the EE, it through the measurement of energy metabolism by indirect calorimetry.

Obesity is also considered multifactorial, with genetic and environmental causes. In this sense, also studied the influence of candidate genes to obesity in metabolic variables, and indirect calorimetry is used in these studies.

The purpose of this chapter is to assess the importance of indirect calorimetry in the assessment of EE in obese individuals, both in study of nutrition intervention and influence of genes in EE, and in the validation and adequacy of existing prediction equations, which were not created for this population.

2. Indirect calorimetry

Indirect calorimetry remains a gold standard in measuring EE in the clinical settings. Indirect calorimetry offers a scientifically-based approach to customize a patient's energy needs and nutrient delivery to maximize the benefits of nutrition therapy. With recent advances in technology, indirect calorimeters are easier to operate, more portable, and affordable. Increased utilization of indirect calorimetry would facilitate individualized patient care and should lead to improved treatment outcomes [17].

Indirect calorimetry is considered a standard method, after validation by comparison with the direct calorimetry [18], however, its use is restricted to research due to the demanding cost and time for its conclusion [14], requiring the use of prediction equations in clinical practice.

According to Green (1994) [19], this technique is based on the principles that there are no considerable reserves of O_2 in the body, the O_2 uptake reflects the oxidation of nutrients, that all the chemical energy in the body comes from the oxidation of carbohydrates, fats and proteins, and that the ratio of O_2 consumption and CO_2 produced for the oxidation of these macronutrients are fixed. After determining the concentration of O_2 inspired and CO_2 expired, the calculation of RMR can be done with the equation of Weir (1949) [20, 21].

Given the difficulties associated with urine collection 24 hours, and found little difference between the results using the complete formula and simplified (2%), many authors have chosen to use the equation of Weir (1949) disregarding urinary losses of nitrogen [22].

The amount of O_2 used for oxidation and CO_2 production will depend on the substrate being oxidized. The respiratory quotient (RQ = Volume CO_2/Volume O_2) varies with the nutrients are being oxidized [14]. The table below describes the values of RQ complexes corresponding to the use of energy substrates [22].

RQ	Interpretation
0.67	Ethanol oxidation
0.71	Fat oxidation
0.82	Protein oxidation
0.85	Oxidation of a mixed diet
1.0	Carboydrate oxidation
1.0 – 1.2	Lipogenesis

Adapted of Materese (1997) [22].

Table 1. Values for interpretation of respiratory quotient (RQ) according to substrate oxidation.

The RQ is divided into non-protein RQ, which reflects the participation of carbohydrates and fats, and protein RQ, which represents the use of proteins. The rate of protein oxidation is obtained by determining the amount of nitrogen excreted in the urine during the test [7, 8].

The evaluation of RMR should only be initiated after a period of rest to minimize possible effects of recent physical activities such as dressing, driving or walking. In practice, it is recommended around twenty minutes of rest, for longer periods can cause the individual to sleep or get impatient [14].

During the measurement of RMR, the ambient temperature should be maintained in the neutral thermal zone, ie between 25 and 26°C, by Henry & Emery (1986) [14]. In order to evaluate the effects of temperature on EE, Dauncey (1981) [23] subjected women to direct calorimetry for thirty (30) hours at 22°C and 28°C. The evaluation of the RMR morning for 30 minutes, after twelve hours of fasting, demonstrated that the production of heat at the lowest temperature (22°C) was significantly greater (mean of 11 ± 3.2%) compared to the higher temperature (28ºC). Wahrlich & Anjos (2001) [14] point out that at ambient temperatures below the neutral zone, the use of protective clothing may be sufficient to prevent the increase in EE caused by the cold.

Compher et al. (2006) [24] conducted a systematic literature review to determine the optimal conditions for obtaining reliable measures of RMR by indirect calorimetry. Based on this survey, the following information was highlighted by the authors: 1) food, ethanol, caffeine and nicotine affect RMR for a variable number of hours after consumption, so its use should be controlled prior to measurement, 2) daily activities increased the RMR, however, a short rest period (20 minutes) before the test is sufficient, 3) moderate or vigorous physical activities presented most impact on the metabolism and therefore should be avoided in hours prior to measurement of RMR; 4) the measure with a duration of ten minutes with the first five minutes disregarded, and the remaining five minutes with a coefficient of variation of up to 10% guarantee accurate measurements of RMR, 5) the trial site should be physically comfortable and should be evaluated for ten to twenty minutes of rest before the start of measurement.

For research on assessment of RMR recommended: fasting for at least 6 hours, abstaining from caffeine during the night, nicotine and alcohol for at least 2 hours, of moderate physical activity for at least 2 hours, and vigorous physical activity for 14 hours [24].

In relation to the proper position for holding the indirect calorimetry, the authors emphasize that the most important is to ensure that each individual is physically comfortable during the test, and that measures are always taken in the same position. However, they warn that some positions require increased muscle tone and, therefore, could influence the measurement of RMR, and for research that has attempted to evaluate the RMR should keep the individual in the supine posture with or slightly higher [24].

The time required for obtaining an accurate measurement of RMR is only about five to ten minutes, discarding the first five minutes, provided that no changes occur in over 10% VO_2 and VCO_2 and 5% in RQ. Accordingly, one measure would be sufficient to describe the RMR for twenty-four hours. However, if you cannot guarantee the stability of the readings, the combination of two or three repetitions increase the precision of the measurements for the extrapolation to twenty-four hours [8, 24].

For studies involving analysis of the thermic effect of food (TEF), the indirect calorimetry should be conducted for a period of 6 hours, as measured by shorter periods are not able to fully assess the TEF [24].

With regard to environmental characteristics, it is recommended that the room temperature is comfortable, that is, between 20 and 25°C, the environment is quiet, with soft lighting and humidity control [24].

In females, should be avoided that the energy metabolism assessments are performed during the luteal phase because this phase of the menstrual cycle are described changes such as water retention, increased of body weight and energy demand, changes in lipid profile and metabolism of some nutrients (vitamin D, calcium, magnesium and iron), emotional hypersensitivity, aches and changes in eating behavior [25].

So, indirect calorimetry is a simple and affordable tool for measuring EE and for quantifying the utilization of macronutrients. Its use is becoming increasingly widespread, but it is

necessary to know its methodological features and its theoretical and practical limitations. Indirect calorimetry measures the rate of REE, the major component of the TEE. Coupling the measurement of body composition to that of REE expands the diagnostic potential of indirect calorimetry. Once the lean and fat compartments have been measured, it is possible to establish on the basis of REE whether an individual is hyper- or hypometabolic. The clinical applications are practically unlimited [26].

Although the basic principles of indirect calorimetry are well established, it is important to recognize that there are several potential pitfalls in the methodology and data interpretation that must be appreciated to properly understand and apply the results derived from this technique. One must recognize that the fundamental measurement provided by indirect calorimetry is the net disappearance rate of a substrate regardless of the metabolic interconversions that the substrate may undergo before its disappearance from its metabolic pool. Under most circumstances, direct oxidation represents the major route by which a substrate disappears from its metabolic pool, and the two terms are often used interchangeably. However, under conditions when rates of gluconeogenesis, ketogenesis, or lipogenesis are elevated, the presumed equivalence between oxidation and disappearance may no longer apply, even though the actual measurements derived from indirect calorimetry remain valid. When indirect calorimetry is combined with other in vivo metabolic techniques (e.g., the insulin clamp or radioisotope turnover methods) it can provide a powerful tool for noninvasively examining complex metabolic processes [27].

Indirect calorimetry can also evaluate BEE. The main difference between REE and BEE is that REE is measured after the individual dislocation to the exam site, necessitating the prior resting period of 30 minutes to neutralize the effects of the physical activity performed [28]. Study found that REE is 10-15% higher than the BEE [29].

3. Utilization of indirect calorimetry in obesity

3.1. Evaluation of prediction equations

Kross et al [30] evaluated the accuracy of multiple regression equations to estimate REE in critically ill patients, especially for obese patients. A total of 927 patients were identified, including 401 obese patients. There were bias and poor agreement between measured REE and REE predicted by the Harris-Benedict, Owen, American College of Chest Physicians, and Mifflin equations (p > 0.05). There was poor agreement between measured and predicted REE by the Ireton-Jones equation, stratifying by sex. Ireton-Jones was the only equation that was unbiased for men and those in weight categories 1 and 2. In all cases except Ireton-Jones, predictive equations underestimated measured REE. The authors concluded that none of these equations accurately estimated measured REE in this group of mechanically ventilated patients, most underestimating energy needs. The authors concluded that is necessary to develop predictive equations for adequate assessment of energy needs.

Ullah et al [31] compared measured REE using the bedside with indirect calorimetry commonly used prediction equations, considering that the accuracy of prediction equations for estimating REE in morbidly obese patients is unclear. A total of 31 morbidly obese patients (46 kg/m^2) were studied. Pre-operative REE with indirect calorimetry was measured and compared with estimated REE using the Harris-Benedict and Schofield equations. All patients subsequently underwent a Roux-en-Y gastric bypass and were repeated measurements at six weeks and three months following surgery. The equations overestimated REE by 10% and 7%, by Harris-Benedict and Schofield equations, respectively. After weight loss the difference between the estimated and measured REE reduced to 1.3%. The accuracy improved after surgery induced weight loss, confirming their validity for the normal weight population. The study demonstrated that indirect calorimetry should be used in morbid obesity.

Cross-sectional study developed by our research group (unpublished data) with 92 women (35.60 ± 6.66 years) with excess body weight (34.41 ± 4.71 kg/m^2), Brazilian and Spanish. This study assessed the women in a metabolic unit, after fasting for 12 hours without performing strenuous physical activity in the last 24 hours and with minimal effort. The evaluation was performed using the open-circuit respiratory hood with indirect calorimetry (Deltatrac Metabolic Monitor-R3D) [6]. For the calculation of EE, it was used the values of the following volumes; inspired O_2 (VO_2), expired CO_2 (VCO_2) (ml / min) and urinary nitrogen [6-28], obtained by the calorimeter. In Brazilian women, it was found that the estimates obtained by Harris-Benedict, Shofield, FAO / WHO /ONU and Henry & Rees did not differ from REE of indirect calorimetry, which presented higher values than the equations proposed by Owen, Mifflin-St Jeor and Oxford. In Spanish women, also the equations proposed by Owen, Mifflin-St Jeor and Oxford presented EE lower than the indirect calorimetry, while the other equations did not differ from the indirect calorimetry. Both are women, Brasilian and Spanish, the best equations were FAO / WHO / ONU, Harris-Benedict Shofield and Henry & Rees.

Study aimed to validate the published predictive equations for REE in 76 normal weight (44.8 kg, 19.0 kg/m^2) and 52 obese (64.0 kg, 25.9 kg/m^2) Korean children and adolescents in the 7-18 years old age group. The open-circuit indirect calorimetry using a ventilated hood system was used to measure REE. Sixteen REE predictive equations were included, which were based on weight and/or height of children and adolescents, or which were commonly used in clinical settings despite its use based on adults. For the obese group, the Molnar, Mifflin, Liu, and Harris-Benedict equations provided the accurate predictions of > 70% (87%, 79% 77%, and 73%, respectively). On the other hand, for non-obese group, only the Molnar equation had a high level of accuracy (bias of 0.6%, RMSPE of 90.4 kcal/d, and accurate prediction of 72%). The accurate prediction of the Schofield (W/WH), WHO (W/WH), and Henry (W/WH) equations was less than 60% for all groups [32].

Alves et al. (2009) [33] compared the RMR obtained by indirect calorimetry with predict equations (Harris-Benedict (HB) and Ireton-Jones (IJ)) in 44 patients with excess body weight. The nearest RMR in fasting was obtained with the equation HB using the current

body weight (1.873 ± 484 kcal / day and 1798 ± 495 kcal / day for HB and indirect calorimetry, respectively). However, the authors emphasize the need to employ the indirect calorimetry for the determination of EE of obese, because despite the similarity found between the absolute REE measured by indirect calorimetry and the prediction equation, there are significant ranges of variability, suggesting that the ideal method and more accurate to obtain the actual REE in this population is the indirect calorimetry.

3.2. Evaluation of the effect of nutritional interventions and obesity candidate genes in energy expenditure

Study with 60 obese women (34.59 ± 7.56 years) was conducted in order to evaluate the influence of fat diet and peroxisome proliferator-activated (PPARγ2) and β2-adrenergic receptor genes on energy metabolism. It was found that polymorphism in PPARgamma2 resulted in increased in fat oxidation, regardless of genotype of β2-adrenergic receptor gene. Polyunsaturated fatty acids (PUFA) intake can assist in weight loss, but the genotype of the genes assessed determines the type of fat that should be ingested [34].

The same research group developed another study with sixty obese women (30–46 years) which were divided into two groups depending on the genotype of PPARγ2 (Pro12Pro and Pro-12Ala/Ala12Ala). At baseline and after two nutritional (short- or long-term) interventions, measurement of anthropometrical and body composition (bioelectrical impedance) variables, dietary assessments, energy metabolism (indirect calorimetry) measurements as well as biochemical and molecular (PPARγ2 genotype) analyses were performed. All women received a high-fat test meal to determine the post-prandial metabolism (short term) and an energy-restricted diet for 10 weeks to determine the effect of diet in long term. The Pro12Ala polymorphism in the PPARγ2 gene influenced energy metabolism in the assayed short- and long-term situations since the response to both nutritional interventions differed according to the genotype. The results suggest that fat oxidation and EE may be lower in Pro12Pro carriers compared to Pro12Ala/Ala12Ala genotypes, while in obese women with Pro12Ala/Ala12Ala polymorphisms in the PPARγ2 gene fat oxidation was negatively correlated with the monounsaturated fatty acids (MUFA) and PUFA (%) intake [35].

The difference in structure of fatty acids, including the chain length, degree of unsaturation and the position of the double bond, can affect the rate of oxidation of fatty acids. Medium chain saturated fatty acids (MCSFA) are more easily oxidized than the long chain saturated fatty acids (LCSFA), while unsaturated fatty acids (UFA) are more easily oxidized compared to saturated chain acids (SFA) with the same chain length [36].

Using indirect calorimetry studies also indicate that the PUFA shows a higher oxidation compared to SFA, both in men and in obese normal [37, 38]. Piers et al (2002) [39] found that changes in the type of fat dietary may have a beneficial effect on reducing body weight in men who consume high fat content, since the oxidation rate postprandial (assessed by Indirect calorimetry) of nutrient is increased after a high MUFA meal, compared with the SFA.

Casas-Agustench et al (2009) [40] aimed to compare the acute effects of three fatty meals with different fat quality on postprandial thermogenesis and substrate oxidation. Evaluated twenty-nine healthy men aged between 18 and 30 years in randomized crossover trial, comparing the thermogenic effects of three isocaloric meals: high in PUFA from walnuts, high in MUFA from olive oil, and high in SFA from fat-rich dairy products. Indirect calorimetry was used to determine RMR, RQ, 5-H postprandial EE and substrate oxidation. Five hours postprandial thermogenesis was higher by 28% after the high PUFA meal (p = 0.039) and by 23% higher after the high MUFA meal (p = 0.035), compared with the high SFA meal. Increased fat oxidation rates no significantly after the two meals rich in UFA and decreased non significantly after the high SFA meal. Postprandial RQ, carbohydrate and protein oxidation measures were similar among meals. The authors concluded that fat quality determined the thermogenic response to a fatty meal but clear effects on substrate oxidation.

Another study was conducted to evaluate whether postprandial abnormalities of EE and / or lipid oxidation are present in healthy, normal-weight individuals with a strong family history of obesity and thus at high risk to become obese. They conducted a case-control study. A total of 16 healthy young men participated in the study. Eight individuals had both parents overweight (father's and mother's body mass index > 25 kg / m^2) and eight had both parents with normal body weight (father's and mother's body mass index < 25 kg / m^2). The group of individuals with overweight parents was similar to that with normal-weight parents (control group) in terms of body mass index and FFM. EE was measured by indirect calorimetry, and blood samples were taken for the evaluation of metabolic variables in the fasting state and every hour for 8 h after a standard fat-rich meal (protein 15%, 34% carbohydrate, 51 fat %, 4090 kJ). Fasting and postprandial EE, and fasting fat and carbohydrate oxidation were both in similar groups. On the contrary, postprandial carbohydrate oxidation (incremental area under curve) was significantly higher and that of fat oxidation lower in the group of individuals with overweight parents. They concluded that normal-weight individuals with a strong family history of obesity present a reduced fat oxidation in the postprandial period. These metabolic characteristics may be considered the early predictors of weight gain and are genetically determined probably [41].

Differences in meal-induced thermogenesis and macronutrient oxidation between lean (*n* = 19) and obese (*n* = 22) women after consumption of two different isocaloric meals, one rich in carbohydrate (CHO) and one rich in fat were examined. Women were studied on two occasions, one week apart. In one visit they consumed a CHO-rich meal and in the other visit a fat-rich meal. The two meals were isocaloric and were given in random order. REE and macronutrient oxidation rates were measured and calculated in the fasting state and every hour for 3 h after meal consumption. Meal-induced thermogenesis was not different between lean and obese subjects after the CHO-rich (*p* = 0.89) or fat-rich (*p* = 0.32) meal, but it was significantly higher after the CHO-rich compared with the fat-rich meal in the lean and the obese individuals (*p* < 0.05). Protein oxidation rate increased slightly but significantly after the test meals in both groups (*p* < 0.01). Fat oxidation rate decreased after consumption of the CHO-rich meal (*p* < 0.001), whereas it increased after consumption of the

fat-rich meal in both groups ($p < 0.01$). CHO oxidation rate increased in both groups after consumption of the CHO-rich meal ($p < 0.001$). Oxidation rates of protein, fat, and CHO during the experiment were not significantly different between lean and obese participants. In conclusion, it was verified that meal-induced thermogenesis and macronutrient oxidation rates were not significantly different between lean and obese women after consumption of a CHO-rich or a fat-rich meal [42].

In a parallel-arm, long-term feeding trial, 24 lean and 24 overweight participants received a daily peanut oil load in a milk shake equivalent to 30% of their REE for eight weeks to evaluate the effects of peanut oil intake on appetite, EE (indirect calorimetry at baseline and week 8), body composition, and lipid profile. Energy intake increased significantly in the overweight but not in the lean participants. A statistically significant body weight gain (median 2.35 kg) was also observed among the overweight subjects, although this corresponded to only 43% of the theoretical weight gain. In the overweight participants, the REE was significantly increased by 5% over the intervention, but no significant difference was observed in the lean subjects. As expected, REE was significantly higher in the overweight than in the lean participants. No marked differences of appetite were observed over time in either group or between overweight and lean participants. These data indicate that ingestion of peanut oil elicits a weaker compensatory dietary response among overweight compared with lean individuals. Body weight increased, albeit less than theoretically predicted [43].

The effects of a moderate-fat diet, high in MUFAs, and a low-fat (LF) diet on EE and macronutrient oxidation before and after a 6-mo controlled dietary intervention were compared. Twenty-seven overweight (body mass index 28.1 $\pm$ 0.4 kg/m^2) nondiabetic subjects (18–36 years) followed an 8-wk low-calorie diet and a 2-wk weight-stabilizing diet and then were randomly assigned to a MUFA ($n = 12$) or LF ($n = 15$) diet for 6 mo. Substrate oxidation and 24-h EE were measured by whole-body indirect calorimetry. The first measurement (0 mo) was taken during the weight-stabilizing diet, and the second measurement was taken after the 6-mo intervention. A tendency was seen toward a lower 24-h EE with the MUFA than with the LF diet ($p = 0.0675$), but this trend did not remain after adjustment for the initial loses of fat mass and FFM ($p = 0.2963$). Meal-induced thermogenesis was significantly ($p < 0.05$) lower with the MUFA than with the LF diet. Despite a slightly lower meal-induced thermogenesis, the MUFA diet had an effect on 24-h EE that was not significantly different from that of the LF diet after a 6-mo controlled dietary intervention [44].

Study with 24 healthy, overweight men (body mass index between 25 and 31 kg/m^2) compared the effects of diets rich in medium-chain triglycerides (MCTs) or long-chain triglycerides (LCTs) on body composition, EE, substrate oxidation, subjective appetite, and *ad libitum* energy intake. At baseline and after four weeks of each dietary intervention, EE was measured using indirect calorimetry. Average EE was 0.04 $\pm$ 0.02 kcal/min greater ($p < 0.05$) on day 2 and 0.03 $\pm$ 0.02 kcal/min (not significant) on day 28 with functional oil (64.7% MCT oil) compared with olive oil consumption. Similarly, average fat oxidation was greater ($p = 0.052$) with functional oil compared with olive oil intake on day 2 but not day 28.

Consumption of a diet rich in MCTs results in greater loss of adipose tissue compared with LCTs, perhaps due to increased EE and fat oxidation observed with MCT intake [45].

A controlled randomized dietary trial was conducted with 26 overweight or moderately obese men and women (body mass index 28-33 kg/m^2) to test the hypothesis that n-3-polyunsaturated fatty acids (n-3-PUFA) lower body weight and fat mass by reducing appetite and *ad libitum* food intake and/or by increasing EE. Diets were administered in an isocaloric fashion for 2 weeks followed by 12 weeks of *ad libitum* intake. The n-3-PUFA and control diets were identical in all regards except for the fatty acid composition. Both groups lost similar amounts of weight when these diets were consumed *ad libitum* for 12 weeks [mean (SD): -3.5 (3.7) kg in the control group vs. -2.8 (3.7) kg in the n-3-PUFA group, $F(1,24)$ = 13.425, p = 0.001 for time effect; $F(1,24)$ = 0.385, p = 0.541 for time × group interaction]. No differences were founds between the n-3-PUFA and control groups with regard to appetite as measured by visual analogue scale, *ad libitum* food intake or, REE as measured by indirect calorimetry, diurnal plasma leptin concentrations, or fasting ghrelin concentrations. These results suggest that dietary n-3-PUFA do not play an important role in the regulation of food intake, EE, or body weight in humans [46].

4. Conclusion

Indirect calorimetry is useful technique in the metabolic evaluation of obese individuals. Despite some methodological limitations, is still the best way to estimate this variable in this population, which is useful both in studies of dietary intervention, intended to propose new strategies for prevention and treatment of obesity, and for validation of predictive equations for energy expenditure in this population.

Abbreviations

BEE - basal energy expenditure
CHO – carbohydrate
EE - energy expenditure
FFM - fat-free mass
LCSFA - long chain saturated fatty acids
LCT - long-chain triglycerides
LF – low-fat
MCSFA - Medium chain saturated fatty acids
MCT - medium-chain triglycerides
MUFA - monounsaturated fatty acids
PPARγ2 - peroxisome proliferator-activated
PUFA - polyunsaturated fatty acids
REE – resting energy expenditure
RMR - resting metabolic rate
RQ - respiratory quotient
SFA – saturated fatty acids

TEE - energy expenditure
TEF - thermic effect of food
UFA - unsaturated fatty acids
VO$_2$ - inspired O$_2$
VCO$_2$ - expired CO$_2$

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