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Thermogenic response to food in trained women

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Thermogenic response to food in trained women

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SOMMAIRE

La thermogénèse induite par l'alimentation (TIA) est l'augmentation du métabolisme basal après l'ingestion d'un repas. La TIA comprend deux composantes: la composante obligatoire, soit les processus de la digestion, de l'absorption , du transport et du stockage des éléments nutritifs et la composante facultative, attribuée au système nerveux sympathique. Chez l'animal, cette composante a lieu au niveau du tissu adipeux brun, et chez l'homme elle dépend des stimulations oropharyngées.

Au cours des deux dernières décennies, on a vu une augmentation significative des études sur la TIA et cela est dû en grande partie aux résultats des investigations suggérant qu'elle joue un rôle important dans la régulation du poids corporel.

Il a été démontré que l'entraînement physique à long terme produit des adaptations physiologiques et métaboliques importantes. L'augmentation de la capacité aérobique maximale, la diminution de la graisse corporelle et l'utilisation accrue des lipides dans le métabolisme oxydatif sont des phénomènes essentiels qui ont une influence dans la performance des athlètes.

Plusieurs chercheurs ont étudié les effets de l'entraînement physique sur la TIA. Toutefois les résultats demeurent controversés. D'autres études qui ont porté sur la corrélation de la TIA et des facteurs tels que la capacité aérobique maximale, la composition corporelle sont tout aussi discordantes. Les résultats controversés peuvent être expliqués par des facteurs d'ordre méthodologique tels que 1) les critères de sélection des sujets: sexe, niveau d'entraînement, composition corporelle 2) le délai entre la mesure de la TIA et le dernier entraînement 3) la composition du repas et la quantité du repas 4) la durée de la mesure de la TIA 5) l'absence de mesure des deux composantes de la TIA

6) des facteurs génétiques. Par conséquent, il nous a semblé important d'étudier la TIA chez des femmes athlètes dans une étude où ces facteurs méthodologiques seraient rigoureusement contrôlés.

Ce mémoire présente les résultats de l'étude dans laquelle nous avons comparé les deux composantes de la TIA (obligatoire et facultative) chez des femmes athlètes (coureuses, n = 12) et chez des femmes sédentaires (contrôle, n = 13). Des analyses de régression ont été faites en considérant la capacité aérobique maximale, le pourcentage de graissse corporelle, le niveau et le type d'entraînement, dans le but d'estimer les facteurs pouvant expliquer les variations de la TIA.

De plus, dans une étude complémentaire, la TIA totale a été mesurée dans un groupe de nageuses (n = 12) pour vérifier la TIA chez des femmes entraînées mais dont le type d'entraînement a des conséquences différentes au niveau de la composition corporelle et le VO_{2max} chez ces athlètes.

L'étude principale, suggère que les femmes entraînées ont une thermogénèse obligatoire plus élevée comparée aux femmes sédentaires. La capacité aérobique maximale est associée à cette composante ($r = 0.454$, $p = 0.06$). Cette corrélation était plus élevée chez le groupe contrôle ($r = 0.617$, $p = 0.01$), que chez les athlètes. Concernant le pourcentage de graisse et la thermogénèse obligatoire, aucun "effet confondant" dû au pourcentage de graisse n'a été observé par cette étude.

De plus, cette étude confirme que l'oxydation des lipides est presque doublée chez les femmes coureuses comparées aux sédentaires au repos.

L'étude complémentaire menée auprès 12 nageuses a démontré que, comparativement aux coureuses, le pourcentage de masse adipeuse était

significativement plus élevée et le VO_{2max} significativement plus bas chez les nageuses.

De plus, la TIA des nageuses était comparable à celle du groupe contrôle (183± 43 vs 185 ± 50 kJ/6hrs respectivement.

L'étude effectuée chez les nageuses, montre donc la nécessité de contrôler et de spécifier le type d'entraînement dans des études chez des femmes athlètes.

Finalement, cette étude suggère que le métabolisme et l'utilisation des substrats sont associés à la capacité aérobique maximale, mais non à la composition corporelle.

SUMMARY

The thermogenic response to food (TRF), is the increase in the resting metabolic rate (RMR) after food ingestion. The TRF has two components: the obligatory component (OTRF) which is involved in the process of digestion, absorption, transport and storage of nutrients and the facultative component (FTRF) which has been attributed to the sympathetic nervous system (SNS). In animals, this component is located in the brown adipose tissue. In humans, it depends upon oropharyngeal stimulations.

In the last two decades, there has been an increased number of studies on TRF. This is due to the suggestion from investigators that TRF plays an important role in weight regulation.

It has been demonstrated that chronic physical training generates important metabolic and physiological adaptations. Increased maximal aerobic power (VO_{2max}), diminished body fat and increased lipid oxidation are essential factors which influence athletes performance.

Various investigators have studied the effects of physical training on TRF. However, results are still controversial. Studies analyzing the interaction of TRF and parameters like VO_{2max} and/or body composition are in disagreement as well. The controversial results may be explained by methodological factors such as 1) the criteria to select the subjects: sex, training level, body composition 2) the delay between the TRF measurement and the last training bout 3) the quantity and composition of the test meal 4) the extent of the TRF measurement 5) the absence in the measure of both components of TRF 6) genetic factors. Therefore, we considered it important to examine TRF in female athletes in a study in which the methodological factors were rigorously controlled.

This memoir presents the results of the study in which we analyzed both components of TRF (obligatory and facultative) in female athletes (runners, n = 12) and sedentary females (control, n = 13). Multiple regression analyses were calculated considering the VO2max, body percentage of fat, the type and level of training in order to explain the TRF variations.

In addition, in a sub-study, 12 female swimmers were also assessed for body composition, VO2max and TRF. Swimmers showed a significantly higher percentage of body fat and a significantly lower VO2max in comparison with runners. TRF in female swimmers was similar than TRF in sedentary females (183 ± 43 vs 185 ± 50 kJ/6 hours) respectively.

The main study suggests that female runners exhibited a higher OTRF in comparison with sedentary females. Also, this component indicated an association with VO2max ($r = 0.454$, $p = 0.06$). This correlation was more significant with the sedentary group. ($r= 0.617$, $p = 0.01$). No " confounding effect" was observed in this study due to the percentage of body fat, and OTRF.

This study confirmed the fat oxidation, which was just about double in female runners compared to female sedentary subjects at rest.

The study on female swimmers indicates the necessity to control and specify the training archetype in further studies of female athletes.

Finally, this study suggests that the metabolic cost of nutrient utilization is associated with VO2max and not body composition.

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LIST OF ABBREVIATIONS

ANS:	Autonomous nervous system
AT:	Adaptative thermogenesis
ATP	Adenosine triphosphate
BAT:	Brown adipose tissue
BIA	Bioelectrical impedance analysis
BMI:	Body mass index
BMR:	Basal metabolic rate
BW:	Body weight
CT	Combine training program
CV	Coefficient of variation
DIT	Diet induced thermogenesis
DLW:	Double level water
DNA	Deoxyribonucleic acid
DZ:	Dyzygotic
EE:	Energy expenditure
Ex:	Exercise
FFM:	Fat free mass
FM:	Fat mass
FTRF	Facultative thermogenic response to food
kg:	Kilogram
kJ	Kilojoule
L	Litters
LBM	Lean body mass
LC:	Low carbohydrate
LF:	Low fat
LPL:	Lipoprotein lipase
mg:	Milligram
ml:	Milliliter

MSNA:	Muscle sympathetic nerve activity
MZ:	Monozygotic
N:	Nitrogen
NE	Norepinephrine
NIR	Near-infrared spectrophotometry
npQR:	Non protein respiratory quotient
Nx:	Non-exercise
OC:	Oral contraceptive
OTRF	Obligatory thermogenic response to food
PA:	Physical activity
PNS:	Parasympathetic nervous system
REE:	Resting energy expenditure
RER:	Respiratory exchange rate
RMR:	Resting metabolic rate
RQ:	Respiratory quotient
SMR:	Sleeping metabolic rate
SNS:	Sympathetic nervous system
SPA:	Spontaneous physical activity
ST	Strength training
T	Testosterone
TDEE:	Total daily energy expenditure
TEM:	Thermic effect of a meal
TIA:	Thermogénèse indute par l'alimentation
TRF:	Thermogenic response to food
UC	Urinary cholesterol
VCO_2:	Carbon dioxide production
VMHSNS	Ventromedial hypothalamus-sympathetic nervous system
VO_2:	Oxygen consumption
VO_{2max}:	Maximal aerobic power or maximal oxygen consumption

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INTRODUCTION

The energy balance model follows the first law of thermodynamics. Its principle, described by Rubner in 1883 has been expressed as:

(energy stored = energy in- energy out)

In the past two decades, investigators have focused on the regulation of the macronutrient balance, especially the fat balance and also, daily energy expenditure in humans. Daily energy expenditure involves four components: resting metabolic rate (RMR), the thermogenic response to food (TRF), the effect of physical activity (PA) and other stimuli or adaptative thermogenesis (AT).The rationale of these studies has been to explain weight gain, assumed as positive balance and weight loss, explained as energy deficit.

The physiological control of energy balance may involve the autonomous nervous system and both its components: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) activity. In obese subjects, it has been suggested that they may have lower energy expenditure rates, associated with a diminished SNS (Garrel, D.R. and de Jonge, L., 1994). In both animals and humans, inactivity is often associated with obesity. In trained subjects, the physiological, hormonal and metabolic changes may affect the relation between SNS and PNS.

The TRF has been used to explain the heat production (energy) following the ingestion of a meal. The study of TRF dates back to the age of Aristotle, who described the concept of "vital heat" as a source of life and all of its powers "of nutrition, of sensation, of movement and thought". The rise in energy expenditure starts immediately after the ingestion of a meal, and returns to its resting value after approximately 4-8 hours (Flatt, J.P., 1978).

TRF has two components: the obligatory component which involves the process of the digestion, the absorption and stocking of nutrients. The facultative component may be regulated by the SNS. In animals, this component is located in the brown adipose tissue (Himms-Hagen,). In humans, it may depend on oropharyngeal stimulus (Garrel, D.R., and de Jonge, L., 1994).

The literature on TRF and obesity has been studied extensively (de Boer, J.O. et al., 1987; D'Alession, D.A., et al., 1988; Anton-Kuchly, et al., 1985). In trained subjects it is controversial (Poehlman, E.T., 1989). The lack of control in the methodology may explain the discrepancies.

This study explored both components of TRF in trained and untrained female subjects at rest and assessed the role of body composition and VO_{2max} as possible influencing factors for each component of TRF. This study, also compared the difference in substrate oxidation in trained and untrained female subjects.

CHAPTER I: LITERATURE REVIEW

1. ENERGY METABOLISM

The cells in the human body have the potential to transform the chemical energy contained in foodstuffs into other forms of energy. This energy could be released as: mechanical energy for movement, thermal energy to maintain normal body temperature, electrical energy for the nerves signal conduction.
The sum of the total processes by which the energy of foodstuffs are transformed and oxidized to carbon dioxide and water is called metabolism. (Ferranini, E., 1988; Wootton, S.,1988).

Since " energy may neither be created nor destroyed", the human body is busy transforming energy, combusting foodstuffs from its own reserves or from dietary intake, and producing heat and mechanical work. Different factors will modify energy metabolism. (Bursztein, S., et al., 1989).

1.1 Energy balance in humans

The nutritional dilemma in energy metabolism implicates the balancing of energy intake against energy expenditure (Wootton, W., 1988).

Body weight is the criterion most conventional for the evaluation of the nutritional status of human beings. Weight gain is assumed as a positive balance of energy whereas weight loss would be considered as evident energy deficit (Heymsfield, S.B. and Williams P.J., 1988).

1.2 The energy balance equation

The notion of energy balance seems simple. This model follows the first law of thermodynamics. Its principle, described by Rubner in 1883 has been expressed as:

Rate of energy stores = rate of energy intake - rate of energy expenditure

This model of energy exchange has been the subject of numerous studies (Durnin, J. et al.; 1973; Jéquier, E., Schutz, Y., 1983; Ravussin, E., et al., 1986; Jéquier, E., et al., 1987; Van, E. et al., 1984; Ravussin, E., Swinburn, B.A., 1993; Girardier, L., 1994; Tremblay, A., 1993).

1.3 Macronutrient balance

In the last two decades, investigators have focused on the regulation of the macronutrient balance (carbohydrate, protein and lipid) (Flatt, J.P., 1987; Jéquier, E. 1990; Tremblay, A. 1993; Ravusin, E. Swinburn, A.B. 1993).

1.3.1 Carbohydrates

Carbohydrates are usually the main source of dietary calories. The stores of this nutrient are low, even if glucose is the unique energy substrate for the brain under normal free living conditions (Tremblay, A., 1993). If the immediate demand for energy is satisfied, carbohydrates may be stored in the liver as glycogen. Carbohydrates are transported as glucose in the blood to other tissues in the body. Only about 50kcal (210 kJ) are accessible immediately as glucose within the blood. The liver stores of glycogen will provide approximately 250-300 kcal (1050-1250 kJ) while glycogen in muscle in the average male will

provide (albeit to muscle tissues only) about 400-500 kcal (1700-2100 kJ). (Wootton, S.,1988).

Carbohydrate balance is accurately maintained due to its rapid adjustment of carbohydrate oxidation in response to its intake. Carbohydrates depend on a metabolic pathway to remove an excess in intake (Tremblay, A., & Alméras, N., 1995).

Because there is an obligatory requirement for glucose in several organs, such as the brain, people who eat low-carbohydrate, high-fat diets may spontaneously increase their food intake. Increasing the proportion of carbohydrate energy while decreasing that of fat in the everyday diet has substantial scientific support in relation to energy balance (Jequier, E., 1994).

1.3.2 Proteins

Protein intake is usually 15% of total energy intake. Under normal feeding conditions, protein oxidation is balanced by protein intake on a day to day basis (Abbott, W.G.H., et al., 1988; Young, V.R., & Marchini, J.S., 1990). This nutrient has good potential to stimulate satiety and to prevent overfeeding (Young, V.R. & Marchini, J. S., 1990).

Protein stores in the body account for one third of the total stored calories in an average 70 kg man . Protein stores may increase in size in response to growth hormone, androgens, physical as well as weight training (Abbott, W.G.H., 1988). To some degree, if there is a chronic energy imbalance induced by an incongruous protein intake and protein oxidation, the amino acids' carbon fraction is transfered to the carbohydrate reservoir via gluconeogenesis (Tappy, L. et al., 1992).

Barenys, M., et al., (1993), appraised the influence of physical exercise and protein intake on RMR and post-prandial energy expenditure in 16 healthy, normal weight, 15 year old males. The boys were assigned to two dietary groups receiving the same energy intake (1.3 of their RMR). Group A received (13 % protein, 39 % fat and 48 % carbohydrates; group B experienced a diet with 30 % protein, 32 % fat, and 38 % carbohydrates. Post-prandial post-exercise thermogenesis was indicated as kcal/3 h. Their results suggested that in normal-weight-adolescents, a hyperproteic diet followed by moderately-intense exercise generates an increase in EE ($p < 0.05$;), and decreases RQ ($p< 0.01$) in the post-prandial post-exercise period and is followed by an enhanced RMR the following day ($p < 0.01$).

1.3.3 Lipids

Lipids are the only nutrient that can perpetuate a chronic imbalance between intake and oxidation and that can lead fat directly to the adipose tissue (Kendall, A., et al., 1991; Tremblay, A., 1993). Lipids' higher energy density and lack of metabolic pathway to regulate excess fat intake make it the weak link for energy and substrate balance (Trembaly, A., 1993; Tremblay A., & Alméras, N., 1995). Among the components implicated in the macronutrient balance, fat balance may appear as a problem for individuals liable to obesity who are characterized by an increase taste preference for fat and a reduced capacity to oxidize fat when their body weight is normal (Tremblay, A., 1995). In an average male adult, the fat stores of the body may be equivalent to some 140,000 kcal (590 MJ). Obese people embrace a greater amount of energy stored as fat (Wootton, S., 1988).

To achieve a steady state in which average fat oxidation matches fat intake, other factors become capable of influencing fat oxidation and/or the relative use of free fatty acids (FFA) and glucose for ATP regeneration. These factors include:

a) carbohydrate intake
b) the maintained glycogen levels
c) sustained exercise which leads to a gradual increase in the rate of fat relative to carbohydrate oxidation
d) exercise-induced buildup of the muscle mass, which at rest uses primarily FFA as fuel (Flatt, J.P., 1991)

1.3.4 Alcohol

Alcohol's energy density surpasses that of carbohydrates and proteins. It also conduces to lipid synthesis (Jéquier, E., et al. 1987).

1.4 Substrate oxidation

Tuominen, et al., (1996), appraised insulin sensitivity and lipid oxidation in 19 male runners after a 42-km marathon run. Muscle glycogen correlated positively with lipid oxidation ($r = 0.60$; $p < 0.05$) and maximal aerobic power peak ($r = 0.61$; $p < 0.05$). Maximal aerobic peak correlated positively with basal lipid oxidation ($r = 0.57$; $p < 0.05$). These authors concluded that the contribution of lipid oxidation in energy expenditure is increased in proportion to physical fitness.

Calles-Escandon et al., (1995) evaluated thirty-two women (18-73 y). The women were appraised for body composition, maximal aerobic capacity, and fat oxidation. Their results showed that fat oxidation was negatively correlated with age ($r = 0.41$ $p = 0.017$) and positively correlated with the fat-free mass ($r =$

0.69; $p < 0.0001$) and with the level of aerobic fitness ($r = 0.47$; $p = 0.007$). Fat oxidation had no relationship with fat mass ($r = 0.26$; $p = 0.136$). The author suggested interventions to increase or preserve the quantity of fat-free mass (e.g., exercise training) to increase fat oxidation and therefore diminish the age-associated adiposity in women.

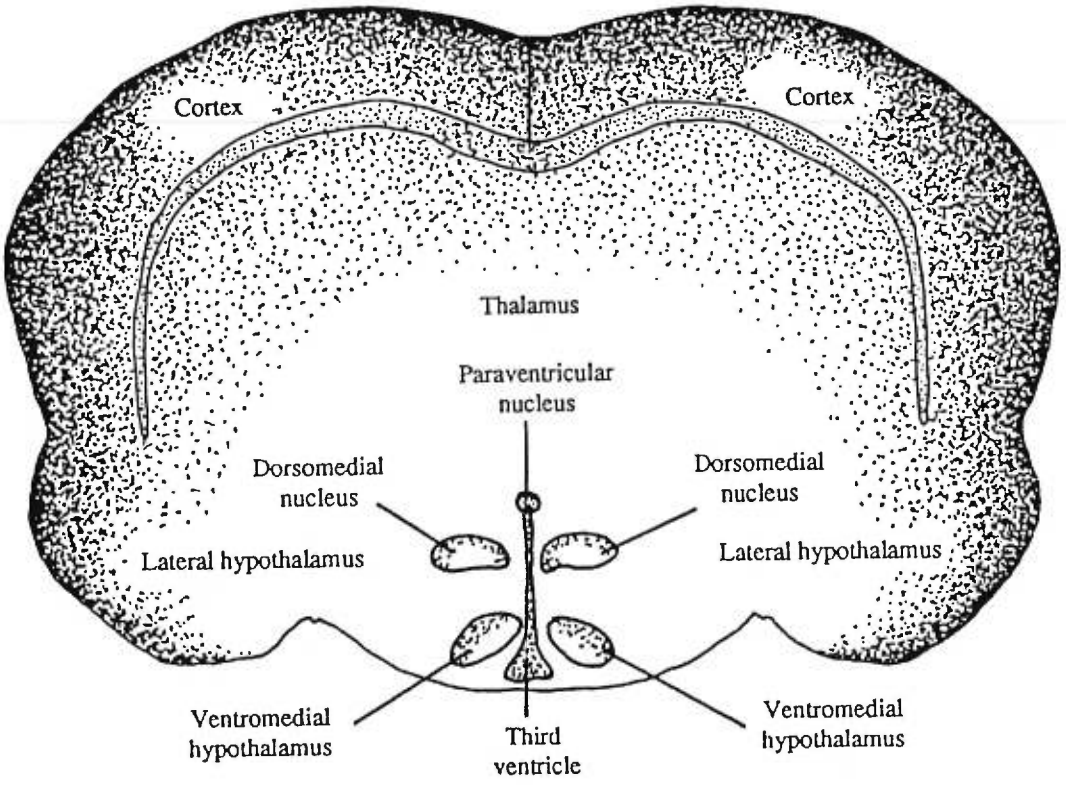
1.5 Physiological control of food intake

The brain mechanisms involved in food intake have been studied extensively and it has been proved that feeding behavior is controlled by activation or inhibition of the specific neurons of the hypothalamus by way of serotonin, noradrenalin and other neuropeptid mechanisms (Blundell, J.E., 1984; Morley, J.E., 1987; Shimazu, T. et al., 1986).

Shimazu et al. (1990), suggested that ventrometial hypothalamus-sympathetic nervous system (VMHSNS) is involved in the control of both energy expenditure and energy supply of the body by regulating lipid and carbohydrate metabolism in adipose tissue, liver and skeletal muscle (Figure 1).

Figure 1

Appetite and satiety regulatory areas of the brain



Adapted from Berdanier, C.D., (1995)

The sympathoadrenal system plays an important role in the regulation of both energy intake and energy expenditure. Differences in TRF can also be explained by different abilities to activate the sympathoadrenal system.

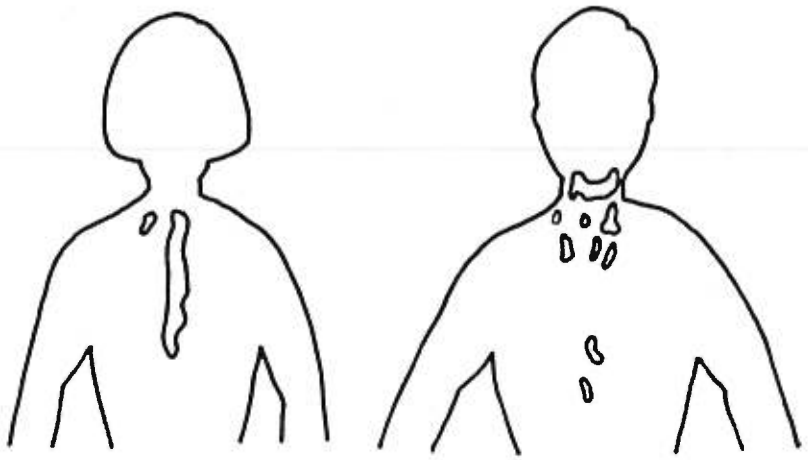
Pharmacological stimulation with sympathomimetic compounds suppresses appetite and increases energy expenditure through stimulation of beta1 and beta 2 receptor types (Astrup, A., 1995).

Brown adipose tissue (BAT) has been found as an important site of non-shivering thermogenesis during cold acclimation, hibernation in animals exposed to cold, in heat production in the newborn animal and diet-induced thermogenesis (DIT) in spontaneous hyperphagic animals. Thermogenesis in BAT comprises a significant component of energy expenditure in small mammals. Its mechanism involves the activation of lipid metabolism in this tissue (Nicholls, D.G., & Locke, R.M., 1984). BAT is characterized by a high content of mitochondria, cytochromes, as well as developed blood supply. Its metabolic importance is on the oxidative processes of high oxygen consumption with a large conversion of both glucose and fatty acids to carbon dioxide (Harper, H. A., 1977).

Although BAT is not a prominent tissue in humans, this tissue has been identified to be active in normal individuals. (Figure 2) It appears to be responsible for diet induced thermogenesis which may explain how some individuals have a disposition to obesity while others do not (Lean, M.E.J. & James W.P.T., 1986). It has been proposed that on days where intake energy exceeds need, the surplus energy is released as heat via BAT. It has been suggested that genetically obese individuals become obese because they are unable to increase their heat production when overfed (Himms-Hagen, J., 1989).

Figure 2

Location of brown fat depots



Female Male

Adapted from Berdanier, C.D., (1995)

1.6 Components of energy expenditure

Kleiber, M., (1975) suggested that "energy expenditure is the most representative parameter of the life process". Ultimately, all the energy produced in the body is dissipated as heat, therefore measurements of the vital heat production of an animal is a way to estimate its energy expenditure (Harper, H. A. Rodwell V. W., Mayers P. A., 1977).

The components implicated on the total energy expenditure are:

Resting Metabolic Rate (RMR) may be defined as the energy necessary to maintain the physiological systems at rest. This component accounts for 60-75% of the total energy expenditure and is associated to the fat-free body mass (Ravussin, E. S., et coll, 1986).

Thermogenic Response to food (TRF) is the energy dissipated in excess of RMR after the ingestion of a meal. This component explains 7-13 % of the total energy expenditure. The TRF component will be discussed in more details in Chapter 2.

Physical Activity (PA), includes minor movement and the residual effects of exercise. This component is the most variable component of daily human energy expenditure. It may account for 15-30% up to 75% of total energy expenditure (Poehlman, E. T., 1989; Elia, M., 1992).

The "other" thermogenic component includes drug induced thermogenesis (e.g., smoking and a thermoregulatory component (e.g., energy produced in response to cold). This constituent of RMR may account for 2-7% of the total energy expenditure (Elia, M., 1992).

1.6.1 Basal metabolic rate (BMR)

Basal Energy Expenditure (BEE) or Basal Metabolic Rate (BMR) is the supply of energy needed for the maintenance of the body's vital functions such as brain function, body temperature, turn over of body tissues, blood circulation, and maintenance of muscle tone at rest. Sleeping, meditation and sedatives may be lower than BMR. Measuring BMR at the laboratory is interpreted as the heat expended by an individual at least 10 hours after the last meal, resting in a lying position, awake at a normal body temperature, ambient temperature between (21-25 ° C) and without physical or psychological stress. Due to its difficult control, manipulation and practicality, (RMR) is commonly applied.

1.6.1.1 Resting metabolic rate (RMR)

This is the measurement of BMR without the standardized strict factors. It is sometimes hard to differentiate between the two (Bursztein, S. et al., 1989).

1.6.1.1.1 Factors influencing RMR:

RMR is influenced by different factors such as:

A. **Surface Area:** In 1883, Rubner proposed that the metabolic rate was somehow limited by body surface area and that oxygen comes in and heat goes out through surfaces that are proportional to the total body surface area (Kinney, J.M. , 1988). The RMR of different individuals when pointed in terms of surface area are constant. However, smaller individuals have a higher RMR per unit of surface area than larger individuals (Harper, H.A., et al., 1977).

B. **Age:** The age-related decrease in RMR has been described in normal men aged > 50 years in cross-sectional studies comprising a large number of subjects (Boothby, W.M., et al., 1936; Shock, N.W. and Yiengst, M.J.,

1955). Webb detected that the reduction in RMR assigned to aging *per se* is no more than 1-2% per decade in subjects aged 20-75. He suggested that the age effect on RMR is explained by a change in body composition (Webb, P., 1981). RMR per kg. body weight (or per kg. fat-free mass) in young children may be two times greater than in adults. Hypothetically, the decline with age could be explained by the reduction in the metabolic rate per gram of individual tissue or to a change in the proportion of different tissues (Elia, M., 1992). Endurance training has been found to increase basal levels or norepinephrine appearance into circulation, and this has been associated with an increase RMR and enhanced fat oxidation (Poehlman, E.T., et al., 1994).

C. **Sex:** Some investigators have established that women normally have a lower RMR than men. The RMR of females declines between the age of 5 and 17 more promptly than that of males (Harper, H.A., et al., 1977). The significance of this difference is a function of the amount of active cells in the body or body cell mass (BCM). Women have more adipose tissue and less muscle than men. The difference begins to appear at age 3, and increases at puberty, when there is an increase mainly in skeletal muscle in boys and in adipose tissue in girls (Malina, R.M. 1988). While other investigators concluded that RMR is lower in women and that is independent from body composition and aerobic fitness (Arciero, P.J., Goran, M.I., Poehlman, E.T., 1993).

D. **Racial Variations:** Most standards of RMR have been derived from studies assuming only Caucasians. The few studies including other races, indicate that race has an important effect on metabolic rate and that many non-Caucasians have a lower RMR than Caucasians of the same weight and height (Henri, C.J.K., Rees, D.G, 1988). The reason for the effect of race on metabolic rate is unknown, but it could, theoretically, be due to differences in genetics, body composition, diet, environmental temperature, hormonal differences, etc. (Elia, M., 1992).

E. **Climate:** The basal metabolic rate is lower in warm climates. When the ambient temperature decreases below the zone of thermal neutrality, there is an increase in energy expenditure, denominate cold-induced thermogenesis. This may occur in two forms; shivering and nonshivering thermogenesis. Nonshivering thermogenesis increases RMR reactions involving hydrolysis of ATP. In rodents, most of this occurs in a distinctive organ; the brown adipose tissue (BAT) (Himms-Hagen, J., 1980). In humans, the BAT is located in small amounts mainly on the scapular area. It is not known whether this tissue is responsible for nonshivering thermogenesis in men. Additional factors controlling body temperature regulation are: secretion of sweat glands, water evaporation from the skin, wetness of the skin and humidity of the air and water loss by evaporation from the respiratory tract (Bursztein S. et al., 1989).

F. **State of nutrition:** In cases of starvation and obesity, the basal metabolic rate is lowered.

G. **Disease:** Infections and febrile diseases raise the metabolic rate, usually in proportion to the elevation of temperature. There may be an estimated increase of 12% of the basal caloric requirement for each degree centigrade.

H. **Effects of Hormones:** The RMR is inferior in hypothyroidism and increased in hyperthyroidism. Another hormone that has a direct effect on the rate of heat production is epinephrine. In adrenal insufficiency, the RMR is subnormal, whereas adrenal tumors and Cushing's disease may produce a small increase in the metabolic rate. Alteration on the hormone levels of the pituitary gland may produce changes on RMR as well (Harper, H. A., et al., 1977).

I. **Genetics:** Heredity may account for as much as 40% of individual variation in RMR once the influences of age, sex and body composition have been considered (Bouchard, C. et al., 1989; Bogardus, C.S.,. et al., 1986). A low RMR for a given body composition is one manifestation of the genetically determined predisposition to obesity (Astrup, A., 1995). The genes involved

in body size and body composition also may have an effect on RMR. Additionally, a major gene affecting only the RMR has been identified (Rice T., et al., 1996).

J. **Body Composition:** There is a positive relationship between RMR and body weight among humans and other mammals with ample grade of body sizes (Kleiver, M., 1947; Schofield, W.N., 1985). The predictability of RMR may be enhanced when fat mass is considered in addition to fat free mass (Heshka, S. et al., 1990; Garby, L., et al, 1988).

K. **Levels of physical activity:** Energy expenditure due to physical activity depends on the intensity and duration of the muscular exercises worked out daily. In athletes, the energy expenditure coming from their physical training could reach 50-80% especially in runners and cyclists from total energy expenditure. The benefits of vigorous exercise, like negative energy and lipid balance, are higher than exercise of low to moderate intensity (Tremblay, A., 1994).

1.6.2 Thermogenic response to food (TRF)

1.6.2.1 Dietary factors that may modify TRF

A. Meal size.

The TRF correspond in amplitude and duration to the meal size ingested (Hill, J. O., et al., 1984; D'Alessio, D.A. et al, 1988; Kinabo, J.K., et al., 1990).

B. Meal composition (Nutrients)

Forbes, E.B. & Swift, R.W., (1944) and Jéquier, E., (1983). noticed the difference between the reaction to single nutrients and mixed nutrients in rats. They detected that protein alone increased 32.3% of the ingested energy,

carbohydrate by 20.2 % and lipid by 16%. Mixing the three of them in a fixed regime declined 22% from computing carbohydrates, protein and lipids separately.

Table I expresses the thermogenesis induced by nutrients in man and the cost of nutrient storage.

Nutrient	Thermogenesis	Nutrient storage
	%	%
Glucose	6-8	12
Lipid	3	4
Amino acids	25-40	25-40

Thermogenesis as a percentage of the energy content of the ingested nutrients.
Cost of nutrient storage expressed as a percentage of the energy content of the stored nutrient.
(Flatt, J. P., 1978; Jéquier, E., 1983).

The group of D'Alessio and the group of LeBlanc noticed a boost of TRF with enlarged amounts of carbohydrates and proteins (D'Alessio, D.A. et al., 1988; LeBlanc, J., et al., 1991).

A meal with a high carbohydrate/fat ratio is more thermogenic than a meal with a low ratio. Due to this rationale, Jéquier, E. (1990) suggested increasing this ratio for obese individuals, especially after a period of hypocaloric diet when establishing a weight maintenance diet.

C. Spices added in the food

Henry, C.J.K. & Emery, B., (1985) tested the same meal with and without spices (chili and mustard). They measured TRF for over 3 hours. The results of their study indicated that the chili and mustard powder increased the oxygen uptakes by 53 ± 8 % compared with a rise of 28 ± 7 % from the milder meal.

D. Coffee and TRF

Bracco D., et al, (1995) explored the influence of coffee ingestion on substrate oxidation in 10 lean and 10 obese women. In one experiment, the subjects consumed decaffeinated coffee and in other, they consumed coffee with caffeine. The extent of thermogenesis was smaller in the obese group (4.9 ± 2.0 %) than the lean group (7.6 ± 1.3 %). The thermogenic response to caffeine was perpetuated during the night in lean women only. The coffee-induced stimulation of EE was moderated by lipid and carbohydrate oxidation. The next day, in postabsorptive basal conditions, the thermogenic effect of coffee disappeared but a significant increase in lipid oxidation was observed (29 % in lean and 10 % in obese women). The investigators' conclusion was that regardless of high levels of urinary methylxanthine excretion in both groups, thermogenesis and lipid oxidation were less stimulated in obese than in lean subjects.

1.6.3 Physical activity (PA)

1.6.3.1 Factors influencing the response (PA)

A. **Heredity.** Bouchard, et al., (1989) studied the heritability of the energy cost of submaximal exercise on a cycle ergometer in 20 pairs of dizygotic (DZ) twins and 31 pairs of monozygotic (MZ) twins. Their results suggested a

significant genetic effect for the oxygen uptake during submaximal exercise of about 6 times the RMR and less, but it was not detectable above this level of intensity.

B. **Sex**. Docherty, D., and Gaul, C.A., (1991) appraised the aerobic, anaerobic and strength performance of 52 young girls and boys (mean age 11.1 and 10.8 years respectively). Anthropometrically the boys and girls were similar with the exception of the content of body fat. The boys showed greater values in maximal aerobic power, anaerobic performance and strength relative to lean body mass. The relationship between height and weight were strongly related to anaerobic performance in boys, whereas, weight and thigh volume were related to all performance measures for the girls.

C. **Body composition**. About one half of the decline in fitness with age can be attributed to the typical increase in body fat and/or diminished fat free mass (Sharkey, B.J., 1990).

D. **Age**. Aerobic fitness develops into the late teens or early twenties and then declines slowly with the years. The rate of decline for inactive individuals is about 8 to 10% per decade). Those who decide to remain active can cut the decline in half, and those who train may decrease that into about 2-2.5 % per decade (Sharkey, B.J., 1987).Endurance training in older individuals increases basal levels of norepinephrine appearance into circulation, and this has been associated with an increase RMR and increase fat oxidation (Poehlman, E.T., et al. 1994).

2.0 THERMOGENIC RESPONSE TO FOOD (TRF).

The term Thermogenic Response to Food (TRF) has been used to explain the heat (energy) rise following the ingestion of a meal (Hegsted, D.M., 1976).

The study of the (TRF), dates back to the age of Aristotle. He explained the concept of "vital heat" as a source of life and all of its powers "of nutrition, of sensation, of movement, and of thought". Galien wrote that food "is used up by our heat as oil is by a flame". He found an identity between the flame and animal heat where not only both were nourished by fuel, but utilized air in a process related to the respiration generating waste products alluded to as smoke. (Leicester, H.M., 1974).

The development of chemistry from 1750 to 1850 enhanced the study on energy metabolism (Kent, A., 1950).The rationale for understanding metabolism was not possible until the discovery of oxygen and carbon dioxide followed by the study of the role of oxidation in many branches of chemistry and biology described by Lavoisier. In some of his studies, Lavoisier suggested that, more oxygen was absorbed. when the temperature outside the body was lower, or after food ingestion or especially during the execution of physical work. (Holmes, F.L., 1985).

The rise in energy expenditures starts immediately after the ingestion of a meal, and returns to its resting value after approximately 4-8 hours (Flatt, J.P.,1978;

2.1 Components of TRF

As previously mentioned, the ingestion or administration of food generates an increase in energy expenditure in excess of RMR. This phenomenon has been denominated as the Thermogenic Response to Food (TRF).

TRF involves two components: The Obligatory TRF and the Facultative TRF
The obligatory TRF which results from the energy expended for the process of absorption, transport, processing and storage of the nutrients. According to Flatt, JP., (1978), the energy expended in the digestion, absorption and transportation of nutrients is negligible in comparison to the metabolic processing of the ingested nutrients. Evidence for this notion comes from a study by Vernet et al. (1986) who found no significant difference in TRF between enteral and parenteral feeding.

The facultative TRF represents the energy expenditure in excess of the obligatory thermogenesis. In their studies, Hill, JO et al.(1985) and Jéquier, E., (1990) suggested that this component is partially mediated by the activation of the SNS.

In rodents, the facultative component appears to have two phases: a short lasting cephalic phase which is related to the sensory stimulus associated with eating and a postprandial phase which is superimposed on the obligatory component of TRF. Both phases depend on the oral ingestion of food. (Himms-Hagen J., et al.,1989). In humans, de Jonge, L., et al., (1991) corroborated the existence of a facultative thermogenesis after food ingestion which depends on sensory stimulus associated to oral ingestion of food.

The facultative heat production is a fundamental process of the human body to respond to over-nutrition and under-nutrition to maintain a constant lean body mass. In this process, the SNS is an important regulator of metabolic processes. Thermogenesis is primarily mediated by both beta 1 and 2 -adrenoreceptors (Saris, W.H., 1995). In rodents,the facultative thermogenesis mediated by SNS does not take place in skeletal muscle. By contrast, in humans, a late post-prandial plasma adrenaline peak stimulates thermogenesis via B_2-receptors in skeletal muscle (Astrup, AV., et al., 1990).

2.2 Measurement of the TRF

Since the obligatory component of TRF does not depend on the route of food administration, one way to measure this component is to inject a blenderized meal into the stomach through a nasogastric tube thereby bypassing the oral cavity and the pharynx (Hill JO., et al., 1985).

The facultative component of TRF cannot be measured directly. Deducting the obligatory component of TRF from the total TRF will reveal its value.
Oral versus gastric feeding has shown inconsistent results with respect to the existence of a facultative component of TRF in man. LeBlanc, et al., (1985, 1989) found a higher TRF after oral than after intragastric feeding in healthy subjects. Hill, JO., et al., (1985), using a similar methodology, concluded that there was no difference between the two modes of feeding. De Jonge, L., et al., (1991), demonstrated that TRF was 40% lower after intragastric feeding in healthy subjects.

Previous studies assessing TRF have been inconsistent. Discrepancies in methodological factors need to be considered, such as:

2.2.1Technique used

The choice of a method for the assessment of energy expenditure will depend upon parameters such as experimental goals, laboratory vs. field studies, the budget available, cultural and social factors, and environmental and statistical factors (accuracy and precision tolerated, sample size, study duration etc.) (Schutz, Y., and Deurenberg, P., 1996).

2.2.1.1 Direct calorimetry:

Direct calorimetry is the direct measurement of the actual total heat production of an individual or an animal. It is performed in a specialized chamber in which the walls of the chamber are able to measure the heat dissipated (by evaporation, radiation, and conduction/convertion) (Ferranini, E., 1988; Kinney, J., 1988). This method is expensive and time consuming to operate and, further, is not accurate to measure TRF.

2.2.1.2 Indirect calorimetry:

Indirect calorimetry is a non-invasive, non-intrusive technique which can be employed in different scenarios such as fundamental research, clinical studies and may be combined with other non-caloric methods (e.g., heart rate monitoring, the double labeled water method and accelerometry) in field studies (Shutz Y., Deurenberg P., 1996). Presently, two methods of indirect calorimetry are used: The whole body method and the ventilated hood system method.

1) The whole-body method uses a respiration chamber. The chamber's volume may vary from 12,000 to 40,000 liters, depending on its dimensions and structure. This chamber is an open-system indirect calorimeter through which air is drawn by a fan. The flow, temperature, barometric pressure, and humidity of the air are measured. A sample is then dried and measured for O2 and CO2 concentrations by differential analyzers.(Ravussin, E., et al., 1986).

2)

The study by Tataranni, et al., (1995) compared different ways of computing TRF from daily energy expenditure measurements in a respiratory chamber. They studied 471 volunteers (258 Pima Indian and 213 whites). One hundred and fourteen available measurements of repeated TRF were analyzed. TRF was calculated using three different approaches: 1) 18 subjects studied twice in

which TRF was calculated as the difference in 24 h EE between the day in the fed state and in the fasted state. 2) In all subjects, TRF (15 h) was calculated from the intercept of the linear regression between EE and activity measured by radar. The difference between RMR and BMR expressed over time (08h00 to 23h00 indicated TRF. 3) In all subjects 24 h TRF was also calculated as the increase of energy expenditure above SMR (sleeping metabolic rate) correlated for SPA (spontaneous physical activity). Their results showed (only in women) a positive correlation between 24 h TRF with SPA and a negative correlation with fasting plasma glucose, body weight, % body fat, waist circumference. In males, a weak negative correlation between 24h TRF and age was found. They concluded that measurements in the respiratory chamber underestimated TRF and that this method is not ideal for assessing this component of EE. In a previous study, Ravussin, E. et al., (1986) concluded that the respiratory chamber is by far the least reproducible of all the components of 24 h EE.

2) The ventilated hood system permits the continuous measurement of whole oxygen consumption (VO_2) and carbon dioxide release (VCO_2). This method, in conjunction with the measurement of urinary nitrogen excretion, assesses heat production generated by the biochemical processes within the body. It also estimates the rate of substrate utilization (Jéquier, E., et al. 1987). The Weir formula is the most commonly used equation to calculate REE from oxygen consumption and carbon dioxide production measurements. It is described as follows:

$$EE = 3.941 \times VO_2 + 1.106 \times VCO_2 - 2.17 \times N$$

(Weir, J.B., 1949)

The energy released from the oxidation of substrates is a process linked to high energy molecule synthesis, mostly adenosine triphosphate; (ATP) This is the principal means of transfering metabolic energy. The most efficient way of using

oxygen to produce the energy available (ATP) is to oxidize glucose; fat and protein oxidation which are most costly in terms of oxygen currency. The oxidation of glucose leads to the following:

C6 H12 O6	+	6 O 2	=	6 CO 2	+	6 H2 O	+	heat
180 g		6 x 22.4 L		6 x 22.4 L		6 x 18 g		2.78 MJ

From this equation it can be computed that the combustion of 1 g of glucose is equivalent to 15.5. kJ, and that 1 L of oxygen used is equal to 20.8 kJ. Analogous equations exists to calculate lipid and protein heat combustion. (Ferranini, E., 1988). (Table II and III).

Table II

Calculations
Energy expenditure was derived from VO_2 , VCO_2 and urinary nitrogen according to the Weir formula: **$EE = 3.941 * VO_2 + 1.106 * VCO_2 - 2.17 * N$** EE = kcal/min kJ = kcal * 4.184 VO_2 , VCO_2 = ml/min N = g/min
Substrate oxidation was estimated according to the following formulas: **Protein oxidation**: **N* 6.25**
Glucose oxidation: **NPRQ > 1 =[(1.34*(VCO_2 - (4.88 *N)]** **NPRQ < 1 = [(4.55*VCO_2) - (3.21*VO_2)- (2.87*N)]**
Lipid oxidation: **[(1.67*VO_2) - (1.67*VCO_2) - (1.92* N)]** N = urinary nitrogen (g/min) NPRQ = Non protein respiratory quotien

Adapted from Ferranini, E., (1988)

Table III

Energy balance for the three main fuels

Utilized fuel (1 mol)	Glucose •	Palmitate	Amino acids Γ
ΔG°(kcal/mol)	- 673	- 2.398	- 475
O_2 Used (mol)	6	23	5.1
O_2 Used (L)	134	515	114
CO_2 Produced (mol)	6	16	4.1
CO_2 Produced (L)	134	358	92
RQ	1.000	0.695	0.807
Net ATP Yield (mol)	36	131	23
Net ATP Yield (kg)	18.3	66.4	11.7
Caloric Cost of ATP (kcal/mol)	18.7	18.3	20.7
Oxygen Cost of ATP (L/mol)	3.72	3.93	4.96
Caloric Equivalent of O_2 (kcal/L)	5.02	4.66	4.17
ATP Equivalent of O_2 (mol/mol)	3.00	2.85	2.85

• Complete oxidation of glucose yields 38 mol of ATP per mol of glucose, but 2 ATP mol are used up during glycolysis.

Γ Complete oxidation of amino acids yields 28.8 mol of ATP, but 5.8 mol are consumed in the process.

Adapted from (Ferranini, E., 1988)

The respiratory quotient (RQ) is a reflection of which fuels are being oxidized. The RQ is the ratio of carbon dioxide produced to oxygen consumed (VCO2 / VO2). Glucose oxidation has an RQ of 1.0, fat oxidation has an RQ of 0.7 and protein metabolism is about 0.8. By excluding protein, the non-protein RQ (npRQ) ranges from 0.70 when fatty acids are the preferred fuel to 1.0 when carbohydrate is the sole nutrient being oxidized. Values in excess of 1.0 indicate that the conversion of carbohydrate to fat is taking place (for example the case of lipogenesis). Reference is often obtained from the Lusk table, derived from measurements in animals to determine the ratio of carbohydrate to fat metabolism based on npRQ (Lusk, G.,1924). This table has been modified to incorporate human physiologic criteria (Peronnet, F., and Massicotte, D.,1991).

Indirect calorimetry has been attested to be a reliable method of measuring EE and substrate oxidation. Recently, Segal, et al., (1992) reported that the TRF measured by ventilated-hood systems has an intraindividual coefficient of variation (CV) of 5.7%. Westrate, JA., (1993) found that the variability of this method was small compared with the intraindividual variability in RMR and TRF. The variability in the method contributed < 10% of the total observed intraindividual variability in RMR.

In our laboratory, accuracy and precision of the system (Deltatrac Metabolic Monitor, DATEX Inc., Helsinki, Finland) measured in vitro is 99% and 2% respectively (Houde-Nadeau, M., et al., 1993).

2.2.2 Time spent for the measurement

Previous studies which measured TRF in trained subjects showed enormous discrepancies (Table IV).The disparity in their results may be explained by the lack of control of the time measurement. The time of measurement in these studies fluctuated between 1.5 h to 3 h.

Table IV

Previous studies in Athletes and TRF

Author	Post-exercise	Sex	Meal	Time	Results
Witt, 1993	40 hours	M	3632 kJ	2 hours	↑ 23%
Tremblay, 1988	84 hours	M	75 g glucose	3 hours	↑ 21%
Hill, 1984	36 hours	M	2092-6276 kJ	3 hours	↑ 37%
Davis, 1983	36 hours	M/F	4184 kJ	2.5 hours	↑ 110%
Thorne, 1989	36 hours	M	60% BMR	3 hours	=
Owen, 1986	?	F	46 kJ/kg	4 hours	=
Poehlman, 1988	24 hours	M	42 kJ/FFM	3 hours	↓ 30%
LeBlanc, 1984	24 hours	F	3422 kJ	2 hours	↓ 11%
LeBlanc, 1984	?	M	3159 kJ	1.5 hours	↓ 50%
Tremblay, 1983	?	M	6845 kJ	2 hours	↓ 48%

- **The controversy from these studies may be explained by methodological factors.**

- **New investigations are needed to clarify the influence of exercise on TEF.**

It has been proven that the rise in energy expenditure starts immediately after the ingestion of a meal and returns to its resting value approximately 4-8 hours later (Flatt, J.P.,1978; Garrel, D.R. & de Jonge, L., 1994; Reed, W.R. & Hill, J.O., 1996). Based on this concept and on the results from the studies of Houde-Nadeau et al., 1993 and Hill, J.O., 1996), it has been suggested that to asses the TRF, indirect calorimetry should be continued for 4-6 h to guarantee the registration of both components (Astrup, A.V., et al. 1990).

2.2.3 Control of the diet before the measurement

Few studies on TRF and exercise-trained subjects monitored the diet prior to the TRF measurement. Witt, K.A. et al., (1993) controlled a low, medium and high carbohydrate diet prior to the TRF measurement on trained and untrained subjects. On the test day, subjects ingested a liquid breakfast of 3632 kJ (74 % carbohydrate, 22 % protein and 4 % lipids). The results of this study showed a higher TRF on trained-subjects (23 % higher). The previous carbohydrate diet showed no influence on TRF.

2.2.4 Control of exercise prior to measurement

Question: How long does metabolic rate remain elevated?

Answer: McArdle, W.D, (1997). " The duration of the post-exercise metabolic rate is very dependent upon the intensity of the exercise bout. If steady-state exercise, which does not promote significant changes in body temperature, is performed, recovery is rapid and metabolic rate will return to resting levels quickly. With high intensity exercise, recovery is slower and metabolic rate may remain elevated for as long as 24 hours. It most be pointed our, however in the elevated post exercise metabolic rate will remain only slightly above resting levels for most of the recovery period, and will not amount to an appreciable caloric expenditure. Therefore it is incorrect to assume that you can burn a significant number of calories following exercise".

Studies that measured TRF at least 36 h after the last bout of exercise showed higher TRF in trained subjects vs. untrained subjects (Davis, J.R., et al., 1983; Hill, J.P., et al., 1984; Tremblay, A., et al., 1988; Witt, K.A., et al., 1993). Thörne, A. & Wahren, J., (1989) studied trained subjects vs untrained subjects. Their last bout of exercise before the TRF test was 36 h for the trained group. They found no difference in TRF. The TRF was lower when measured 24 h and less from the last bout of exercise (LeBlanc, J., et al., 1984; LeBlanc, J et al., 1984; Poehlman, ET., et al., 1988; Tremblay, A. et al., 1983) (Table IV).
There is still discrepancy concerning the last bout of exercise and TRF. In our study, we suggested measuring the trained subjects 24 h after the last bout of exercise to have a homogeneous group for the trained subjects. We did not let more than 24 h pass after the last bout of exercise because the objective of this study was to measure the effects of habitual exercise training on TRF. Trained subjects exercised on average 13.57 ± 2.8 h per week. It is difficult for an athlete to stop training even for just a day.

2.2.5 Definition of trained subject

It is difficult to compare studies dealing with physical adaptations because of the wide variation in training methods. Different results will be present as the method of exercise (e.g., running, stationary cycling, aerobic dance, weight training , etc.), training frequency, duration, intensity differ. In addition, there is an ample variation among studies in the initial fitness level, age, body type and body composition of the subjects.

2.3 Studies in animals

Reduced SNS activity in BAT is a significant factor in the development of obesity (Yoshida, T., 1990). Himms-Hagen, J., (1989) suggested a deficit in the hypothalamic control of facultative thermogenesis and or food intake in all obese

animals. The BAT thermogenesis is important in the control of meal size, relating it to thermoregulatory needs. When this function is lost, as in many obese animal models, the animal loses its ability to remain in energy balance by precisely adjusting its intake in relationship to environmental temperature and meal size increases (Himms-Hagen J., 1995).

Thurlby, PL., & Trayhurn, P., (1979) studied genetic obesity in homozygotic mice ob/ob.

They observed diverse deficits of thermogenesis due to:

a) Thermogenesis induced by overfeeding.

b) Exposiure to cold.

c) Thermogenic response to noradrenaline response.

d) BAT Atrophy.

e) Mitochondrial alterations of BAT.

f) Insulin resistance.

g) Obesity may be developed without overfeeding.

Hirata, K., (1982) directly measured thermogenesis of BAT in physically trained rats and control rats in response to infused norepinephrine (NE). His results demostrated that physical training per se, increases thermogenesis. The contribution of BAT to the NE response increased to 50% in trained rats compared to 36% in control rats.

Puerta, et al., (1996) inserted subcutaneos implants of progesterone in female rats. The rats were acclimated to thermoneutrality to avoid cold influences. The rats increased their food intake and body weight above the control group values. No sign of activation of the SNS was observed, judged by the unaltered noradrenaline content in BAT, pancreas and heart. The authors suggested that the central mechanisms in regulating energy balance are probably influenced by a different process by progesterone rather than by sensory properties of palatable diets.

2.4 Studies in humans

2.4.1. Obesity

Suppression of facultative components of RMR, thermic effect of cold and heat exposure, mental stress and exercise may contribute to a positive energy balance. Inhibition of facultative thermogenesis by B-blockers such as propanolol, diminishes the daily EE and promotes weight gain and obesity (Astrup, A.V., et al., 1990).

Whether or not TRF is reduced in obesity has been the aim of considerable research in humans. (Anton-Kuchly, B. et al., 1985; den Besten, C. et al., 1988; de Boer, J.O. et al., 1987; D'Alessio, D.A. et al., 1988; Segal, K.R. et al., 1990).

Low EE (relative to body size), high RQ and insulin sensitivity are indicators to predict body weight gain (Swinburn, B.A. & Ravussin E., 1994).

2.4.1.1 Exercise and obesity

As the most variable component of EE, exercise-induced thermogenesis delivers the greatest range of effects on the energy balance equation, and therefore has great potential in the prevention and treatment of obesity (King, A.C. & Tribble D.L., 1991).

Weinsier, R.L. et al., (1995) studied 24 moderately obese, postmenopausal, non-diabetic women with a familial predisposition to obesity. The obese women were assessed before and after weight loss (mean 12.9 kg). This group was compared to 24 women of comparable age and body composition who had no history of obesity. All parameters measured in the post-obese women were similar to the never-obese control group: REE, TRF, fasting and postprandial substrate

oxidation and insulin-glucose levels. Four years later, without intervention, post-obese women regained a mean of 10.9 kg while control subjects remained lean with a mean of ± 1.7 kg ($p < 0.001$). Self-reported physical inactivity was associated with greater weight regain in obese-prone women.

Racette, S.B., et al., (1995) tested the benefits of aerobic exercise and dietary carbohydrate during reduced-energy feeding. Twenty three obese women (44 ± 4 % fat) were randomly designated either for aerobic exercise (Ex) or non exercise (Nx) and either a low fat (LF) or a low carbohydrate reducing diet (LC) (5.00 ± 0.56 MJ/d) for 12 wk. RMR, TRF, and TDEE were measured by respiratory gas exchange and double labeled water (DLW). Their results showed a greater loss of fat mass with exercise vs. non-exercise (- 8.8 ± 2.1 vs. - 6.1 ± 2.3 kg). Total daily energy expenditure (TDEE) was higher with exercise (+ 0.07 ± 1.23 vs -1.46 ± 1.04 MJ/d, $p = 0.004$). Diet composition did not significantly influence body composition or energy expenditure changes (RMR and TRF) but weight loss was significantly lower after the LC diet than after the LF (- 10.6 ± 2.0 vs. - 8.1 ± 3.0 kg, $p = 0.037$). Contradictory, Segal, K.A., et al.,(1990) found that the percent of body fat was the best predictor of TRF ($r = -0.74$; $p = < 0.01$) between body composition variables that included percent of body fat, FFM, BW, and aerobic fitness in male obese subjects.

The addition of exercise to a weight loss regimen increases EE, preserves lean body mass and alters fat mobilization and utilization (Bjorntorp, P, 1989;Saris, WHM, 1989). Exercise appears to increase RMR and TRF in obese subjects (Frey-Hewitt, et al., 1990; Poehlman, E., 1989; Segal, K.R. and Pi-Sunyer F.X., 1989)

2.4.2. Exercise and eating disorders: anorexia nervosa and bulimia

The strong cultural fixation with thinness on women in many Western societies may have serious health effects in the form of eating disorders such as anorexia nervosa or bulimia nervosa.

2.4.2.1 Exercise and eating disorders

In the case of eating disorders, strenuous, excessive exercise may become part of the problem rather than the solution. Whether the exercise in such cases is a consequence of the anorexia or one of the contributing factors in its development is not evident (Yates, A., et al., 1983).

2.4.3 Effects of exercise on TRF in female athletes

There is a limited number of studies on female athletes and TRF. Although LeBlanc, J. et al., (1984) studied a group of competitive female athletes(VO_{2max} 57.5 ml* kg^{-1}*min^{-1}) and found a significantly lower TRF in this group compared with sedentary subjects (VO_{2max} 38.9 ml*kg^{-1}*min^{-1}). Measurements were taken 1 h after ingestion of a 800 kcal mixed meal. However, no significant differences were noted in TRF after the second hour following the meal. Myerson et al., (1991) did not find any significant difference in the TRF between amenorrheic runners, eumenorrheic runners and sedentary controls ($p > 0.05$) with measurements taken for 3 h after consumption of a 2704 kJ liquid test meal. However, the TRF for the sedentary subjects was approximately 200 kJ/d higher than the TRF value for the amenorrheic and eumenorrheic runners. The sample size was small and only 3 h measurement might not have been long enough. Owen et al., (1986) found TRF for trained and untrained women to be similar with measurements taken 4 h after consumption of 11 kcal* kg^{-1}.

2.4.3.1 Factors Influencing TRF and exercise in females

A. Genetics

Another reason that inconsistent results may be found in the literature regarding TRF and exercise training may be due to the selection of the subjects genetically predisposed to high or low TRF. Poehlman, E., et al., (1986) found poor responders and high responders when they studied TRF before and after exercise training. Their study included 6 pairs of male monozygotic twins. The researchers found no significant difference in TRF after a 22 d ergocycle exercise program which significantly increased VO_{2max} ($p < 0.01$). Some twin pairs manifested a decreased TRF while other twin pairs showed increased TRF. These two responses canceled each other out and therefore no significant difference in TRF was found before and after training. However, intrapair resemblance was observed for the absolute TRF, and a significant intra-cohort correlation ($r = 0.72$) was noticed. Consequently, genetic influence may partially explain the individual variation in TRF and the effect of exercise training. Additionally, Bouchard, C. et al., (1989) found that the genetic effect of RMR had 40% variation after adjustment for age, gender and body composition in 21 pairs of dizygotic (DZ) twins and 37 pairs of monozygotic (MZ) twins as well as in 31 parent-offspring pairs. Correlations of 0.35, 0.52, and 0.30 were found respectively for TRF. This was equivalent to at least 40-50% of the variation in energy dissipated 4 h after a 1000 kcal high carbohydrate meal. No significant genetic variance was observed for the glucose and insulin response to the carbohydrate meal.

B. Menstrual cycle

There is little information on the effects of the menstrual cycle on TRF and postprandial substrate oxidation. Melanson, K.J. at al., (1996) studied 8 young women to whom four different test meals (0, 1046, 2092 and 4148 kJ) were administered on non-consecutive days, once each during the follicular phase and

once during the luteal phase of their menstrual cycles. Postprandial energy expenditure was measured until it returned to the base line (test duration of the 0 kJ meal was matched to duration of 4184 kJ meal). No significant difference between menstrual cycle phases in the duration of the TRF to any meal size were found. No effect of the menstrual cycle phase was detected on postabsorbtive or postprandial carbohydrate, protein or fat oxidation.

Few studies have determined the stage of menstrual cycle and fuel oxidation. There may be evidence of enhanced lipid oxidation during physical activity observed in the luteal phase compared to the follicular phase of the menstrual cycle (Hackney, A.C., et al., 1994; Nicklas B.J., et al., 1989).

3. PRINCIPLES OF TRAINING

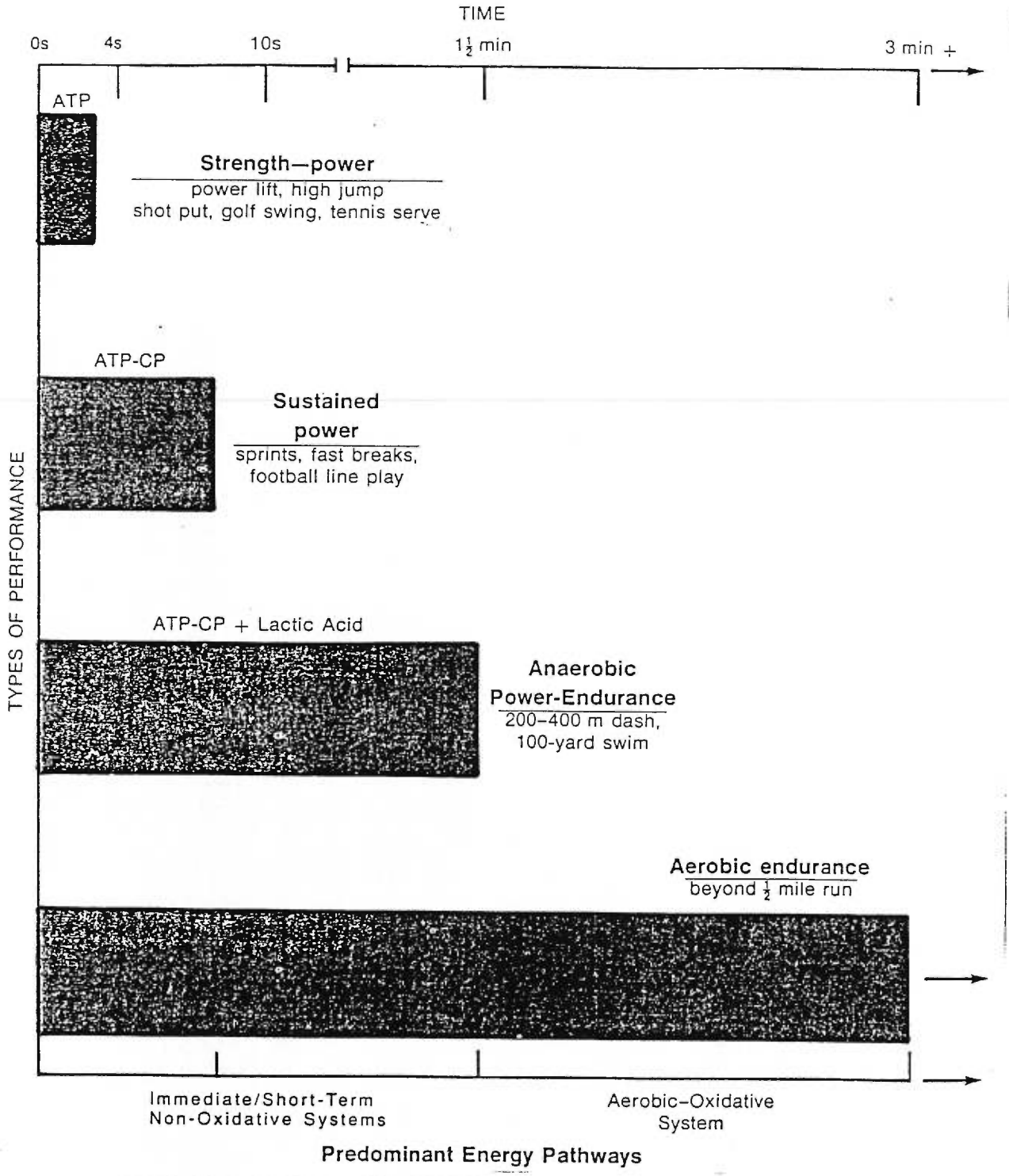
There are considerable physiological differences between the average male and female. These contrasts are reduced considerably when the analogy is estimated between highly trained male and female athletes who compete in the same event or sport. Highly trained male and female athletes are similar in lower body strength denoted per unit of body weight; cardiovascular endurance capacity; body composition and muscle fiber type. (Wilmore, J.H., 1979).

The major objective of training is to cause biological adaptation in order to improve achievement in a specific exercise. Many factors may affect performance, such as frequency and length of workouts, type of training, speed, intensity, duration and repetition of the activity. (Figure 3) illustrates the classification of activities based on duration of performance and the predominant intracellular energy pathways.

Aerobic training improves a variety of functional capacities related to oxygen transport and use. The most remarkable adaptation following aerobic training involves the increased number of mitochondria from trained skeletal muscle and consequently, a considerable increased production of ATP aerobically by oxidative phosphorylation (Holloszy, J.O., et al.,1984; Holloszy, J.O., 1973).

Figure 3

Predominant energy pathways



Adapted from Wilmore, J.H., (1977)

3.1 Body composition definition

A person with a good level of health-related fitness should show a body mass that is close to the actuarial ideal value (i.e. weight associated with the lowest incident of mortality), with a low percentage of body fat, an adequate muscle mass, strong but flexible tendons, and bones with an adequate mineral content (Metropolitan Life Insurance Company, 1983).

The minimum amount of body fat compatible with good health in men is 3 %, a lower percentage may impair normal physiologic function or capacity for exercise. World-class male marathon runners ranges from about 4-8 %. This low quantity of body fat endorse a more effective transfer of metabolic heat during high intensity exercise (Katch F., et al., 1988). For the average men 12-15 % is acceptable. More than 20 % may be consider obese (Lohman, T.G., 1992).

The lower limit of body weight for females 7-10 % body weight world-class runners. (Katch, V.L., et al., 1980;Wells, C., 1991). For the average women the values of 18- 24 are acceptable. More than 31 % may be consider obese (Lohman T.G., 1992).

Women over the age of 25 will be inclined to lose some body tissue. Body density will decrease and fat will be gained. Most women in their 30s and beyond need either to become more physically active or practice at least occasional dietary restriction. The "battle of the bulge" begins even earlier for physically inactive women (Wells, C., 1991)

Body composition refers to the internal constitution of the body. Most indirect methods estimating body composition depend on a two component model that divides the body into fat and lean masses. The following definitions are adapted from (Sady, S.P., and Freedson P.S., 1984)

Lean body mass (LBM) - The mass of a body less all but essential fat.

Fat-free mass (FFM) - The mass of the body less the ether-extractable fat (this still contains a small percentage of essential fat.

Adipose tissue - The tissues that contain fat (triglyceride), adipocytes and supportive connective tissue.

Essential fat - The fat (lipids such as lecithin and phospholipids) required for normal physiological functioning that is contained in bone marrow, heart, lung liver, spleen, kidney, intestinal muscle, and central nervous system tissues.

Storage fat - The major fat deposit that accumulates in adipose tissue (serves as a nutritional reserve and as protection for internal organs), that includes subcutaneous fat deposits.

Sex-specific fat - The fat that is specific to females (sometimes included in the essential fat classification) and thought to be associated with hormone synthesis for reproduction. (Table V).

3.1.1 Differences between men and women

There are very small differences in body fat and lean body mass (LBM) in early life (3 to 4 years of age). The gain in LBM and muscle mass occurs by late adolescence in girls and two thirds of the values of boys (Malina, R.M., 1988). The higher levels of estrogenic hormones in the adolescent and adult female are at least partially responsible for the higher percentage of fat tissue in the female body. The absolute level of storage fat tissue in woman may equal that of men, but because her total body weight is less, her relative fat is greater (Wells, C.L., 1991).

Table V

Body composition of average (reference) adult men and women

Characteristics	man [a]	women [a]	women [b]
Height (inches)	68.5	64.5	64.5
Weight (kg)	70.0	56.8	56.8
Muscle (kg)	31.4	20.5	21.0
(%)	44.8 %	36.0 %	37.0 %
Bone (kg)	10.5	6.8	8.0
(%)	14.9 %	12.0 %	14.0 %
Storage fat (kg)	8.4	8.5	8.5
(%)	12.0 %	15.0 %	15.0 %
Essential fat (kg)	2.1	6.8	2.3
(%)	3.0%	12.0%	4.0%
Sex-specific fat (kg)	-	-	2.8
%	-	-	5.0 %
Total fat (kg)	10.5	15.3	13.6
%	15.0 %	27.0 %	24.0 %
Remainder	17.7	14.2	14.2
%	25.3 %	25.0 %	25.0 %

Data from Behnke (1969)

Data from Katch, Campaigne, Freedson and Sady (1980)

3.1.2 Genetic factors

Research on heredity of body fat, fat free mass, and the ratio of fat mass to fat free mass has suggested that the noticeable level of genetic transmission across generations is moderate. After allowing for age and gender phenotypes, the differences may be about 25 %. This implies that most of the individual differences in body composition are independent of the genotype and result from individuality in the amount of food intake, food preference, level of habitual physical activity, and other life characteristics such as smoking, alcohol intake, sleeping and work habits, adaptability to stress, common climate conditions. Regardless of these low levels of heredity, genetic variation remains relatively important in detecting individual differences in the adjustment to positive or negative energy balance (Bouchard, C. and Tremblay, A., 1990).

Chitwood, L.F., et al., (1996) explored the metabolic predisposition towards the development of obesity in 11 black vs 11 white normal weight sedentary females with a family history of obesity. The study included the cross-sectional comparison of the responses during 30 min at rest, 30 min on a treadmill (65 % VO_{2max}) and 30 min of recovery. Their results in black women indicated lower VO_{2max} at rest ($p = 0.04$) and recovery ($p = 0.04$), higher respiratory exchange ratio (RER) during rest, exercise and recovery ($p = 0.003$) and higher insulin levels. The investigators proposed that lower rates of oxygen consumption, the higher metabolic dependency on carbohydrates, and higher insulin levels may gradually impact energy balance predisposing the black females toward obesity.

3.1.3 Evaluation of body composition (Methods used in this study)

The underwater-weighing method (hydrostatic weighing) of appraising body composition is considered by most investigators to be the criterion method from which other methods are derived. At present, the underwater-weighing is still considered one of the most reproducible techniques (coefficient of variation = 1.2%). It has been the base when comparing other methods' precision to assess

body composition in athletes (Hortobágyi, T., 1992). Underwater-weighing assessment of body composition is likely to be subject to error for children and older persons (e.g., the stress involved into submersion under water and extraction of the air from the lungs); post-menopausal women (e.g., accounting for possible osteoporosis); extra lean or extra fat persons; racial groups (e.g., blacks have a higher bone density than whites).

Hortobágyi, T. (1992) compared four methods for assessing body composition in black and white athletes. The percentage of body fat (%BF) was estimated by hydrostatic weighing at residual volume, corrected for race; seven-site skinfolds (7SF), bioelectrical impedance analysis (BIA); and near-infrared spectrophotometry (NIR). They concluded that compared to hydrostatic weighing, the NIR (futrex-5000, Futrex Inc., Gaithersburg, MD) and the BIA (RJL Spectrum II System, Detroit, MI) method did not accurately estimate body composition in athletes. When the cost and expertise of the method are considered with NIR and BIA, skinfold measurements may be a superior alternative for coaches and staff for rapid and accurate assessment of body composition in athletes, independent of race.

3.2 Maximal aerobic power (VO_{2max})

The conventional index of aerobic fitness is the maximal oxygen intake, or aerobic power (VO_{2max}). It may be defined as the plateau of oxygen intake observed when a subject performs a progressive, large-muscle exercise to exhaustion, such as uphill treadmill running, step climbing, or cycle ergometry. A plateau is commonly defined as an increase in oxygen intake of less than 150 ml/min or less than 2ml/(kg * min) in response to a further substantial increase in the rate of working. Aerobic power is not synonymous with health-related fitness, though this has sometimes been supposed. Still, large aerobic power is one of the most important physiological indicators of good physical condition (Shephard, R.J., 1994) (Table VI).

Table VI

Norms for VO_{2max} in women

(Maximal aerobic power in $L \bullet min^{-1}$ and $ml \bullet kg^{-1} \bullet min^{-1}$)

Age	Low	Fair	Average	Good	High
20-29 years					
$L \bullet min^{-1}$	1.69	1.70-1.99	2.00-2.49	2.50-2.79	2.80 +
$ml \bullet kg^{-1} \bullet min^{-1}$	28	29-34	35-43	44-48	49 +
30-39 years					
$L \bullet min^{-1}$	1.59	1.60-1.89	1.90-2.39	2.40-2.69	2.70 +
$ml \bullet kg^{-1} \bullet min^{-1}$	27	28-33	34-41	42-47	48 +
40-49 years					
$L \bullet min^{-1}$	1.49	1.50-1.79	1.80-2.29	2.30-2.59	2.60 +
$ml \bullet kg^{-1} \bullet min^{-1}$	25	26-31	32-40	41-45	46 +
50-65					
$L \bullet min^{-1}$	1.29	1.30-1.59	1.60-2.09	2.10-2.39	2.40 +
$ml \bullet kg^{-1} \bullet min^{-1}$	21	22-28	29-36	37-41	42 +

Adapted from Åstrand, I. (1960)

The highest level of work that can be accomplish without inducing a prolong metabolic acidosis is called the anaerobic threshold. It is marked by the sudden change in the measured variables mainly because of developing lactic acidosis as glycolysis takes over further of the muscle energy supply as the result of the failure of the body to supply oxygen to the muscles at the rates demanded by the level of exercise. When the exercise level is below anaerobic threshold, ventilation is linearly related to both carbon dioxide and oxygen consumption (Berne, R., Levy M., 1993).

3.2.1 Factors that affect (VO_{2max})

1. Body weight and body composition
   a. Active muscle mass.
   b. Fat weight.

The difference in body composition between genders is basically attributable to the additional 5-9% of body mass that is essential fat in females.

2. Maximal system oxygen transport
   a. Maximal cardiac output (maximal heart rate, maximal stroke volume).
   b. Oxygen-carrying capacity (hemoglobin concentration).

3. Skeletal muscle oxidative capacity

Resting muscle metabolism is positively associated with the differences in RMR observed among people and may represent a major determinant of the inter-individual variation in EE. Several factors may influence the variation in muscle EE, such as thyroid hormone, muscle fiber types, muscle tone, sympathetic innervation and/or cathecolamines levels (Zurlo, F., et al., 1990).

4. Muscle fibers

Two distinct types of fibers have been identified in human skeletal muscle:

Fast-twitch fiber- This muscle fiber, also called a type II fiber, gathers a high capacity for anaerobic production of ATP during glycolysis. The contraction speed of these fibers is rapid. This kind of fiber is activated in short-term, sprint activities that depend practically on anaerobic metabolism for energy. The metabolic capacities of fast-twitch fibers are also important in stop-and-go or quick change-of-pace sports such as basketball, soccer, field hockey and squash. At times, these sports require rapid energy that can only be supplied through anaerobic metabolism.

Slow-twitch fiber - This muscle fiber has a contraction speed about half as fast as fast-twitch fibers. Slow-twitch fibers hold numerous mitochondria and a high concentration of the enzymes required to maintain aerobic metabolism. Their capacity to generate ATP aerobically is much greater than that of fast-twitch fibers. Slow-twitch fibers are active in endurance activities that depend mostly on aerobic metabolism such as middle-distance running or swimming.

Successful athletes in most sports have mixed muscle fiber distributions similar to the average population. However there may be a relationship between performance in some sports and muscle fiber composition. Female athletes appear to acquire similar muscle fiber ratio patterns as males practicing the same sport (Wells, C., 1991).

5. Genetic factors

There may be a inherited potential considering low and high responders to training. The heredity of VO_{2max} could account between 20-60 % of the total phenotypic variation. The range to improve VO_{2max} by training may be between 20-40 %. Three mitochondrial DNA morphs, two in the NADH dehydrogenase gene and one in the tRNA for threonine have been detected. People carrying

these genes may endure higher VO_{2max} than non carriers, even if the subjects are untrained (Dionne, F.T. et al., 1991).

3.3 Effect of VO_{2max} on TRF

Tremblay, A., et al., (1983), measured 8 highly trained (VO_{2max} = 69.2 ± 2 ml/kg/min) and 8 untrained (VO_{2max} = 47.7 ± 3 ml/kg/min) male subjects. RMR was not detected to be different between groups. TRF was assessed after the ingestion of a mixed meal (1636 kcal). TRF was significantly smaller in the highly trained subject. The RQ was lower in the trained men indicating boosted fat oxidation.

LeBlanc, J., et al., (1984) studied a highly trained group of women (57.5 ml/kg/min), moderately active (49.9 ml/kg/min) and sedentary subjects (38.9 ml/kg/min). Results from this study disclosed that RMR was not different among groups, but the TRF after a mixed meal (800 kcal) was smaller in the highly trained women. In both studies, TRF was only measured for two hours. This may be too short of time as suggested by Houde-Nadeau, M., et al., (1993) and Reed, G.W., and Hill, J.O., (1996).

Davis, J.R., et al., (1983) observed RMR, TRF, aerobic capacity (VO_{2max}) and percentage of body fat (% BF). Sixteen women and 10 men participated in the study. Their results indicated that TRF was significantly positively correlated with VO_{2max} ($r = 0.658$; $p < 0.01$). The % BF was significantly negatively correlated with VO_{2max} ($r = -0.727$). In the same study, three men and three women participated in an endurance training program which significantly increased their VO_{2max}. TRF was increased in harmony with the enhanced VO_{2max} ($r = 0.924$; $p < 0.05$). Their conclusion was that endurance training enhanced the extent of TRF in proportion to the improvement in aerobic capacity.

Hill, J.O., et al., (1984) also detected that TRF was highly correlated with VO_{2max} ($r = 0.78$) following a 1500 kcal meal. However, Poehlman, E.T., el al., (1989) reported a curvilinear relationship between TRF and VO_{2max} in 28 male non-obese subjects where TRF was measured for three hours . They observed a higher TRF in the mid-range of VO_{2max} values whereas a lower TRF was observed in the extreme ranges (low and high of VO_{2max}). The correlation started to be curvilinear until their subjects reached 70 ml/kg/min.

Owen, O.E., et al., (1986) detected no difference in TRF for 8 Trained and 36 untrained women with measurements obtained four hours after consumption of an 11 kcal/kg meal. Even though their trained subjects showed high values of VO_{2max} (52-62 ml/kg/min), the ample range on age (18-65 y) and wide range on body weights (43-143 kg) of their subjects may have altered their results.

3.4 Relationship between body composition and VO_{2max}

The percentage of body fat is possibly reduced by an aerobic training regimen, provided that the exerciser does not counter the added energy expenditure with an increased intake of food (Pollock, M.L., 1973). Both the size and the triglyceride content of individual fat cells are reduced by aerobic training (Taylor, A.W., 1979).

VO_{2max} is largely dependent on body weight and active skeletal muscle mass. Aerobic power expressed per unit of body weight is significant because it is the best global indicator of physical working capacity in weight-carriage exercise such as walking, running, bench stepping, and stair or hill climbing. One factor observed to influence VO_{2max} during weight-bearing work was the level of relative body fat. Body fat adds to the load carried but not to the definitive VO_{2max}. Since the adult female has about 10 % more relative body fat than the adult male, part of the difference between men and women in relative VO_{2max}

between trained men and women is less than that in untrained subjects when expressed in liters per minute and milliliters per unit of body weight but not when expressed relative to FFM (Sparling, P.B., 1980).

4. OTHER CHRONIC ADAPTATIONS TO EXERCISE TRAINING

The degree of chronic adaptations to exercise training is related to the intensity of training, the athletes genotype and phenotype and also to other factors such as age, nutrition, lifestyle and environment. The morphological transformations, especially the adaptations of the heart and circulatory system increase blood flow to muscle cells leading to a higher energy supply and substrates and increasing the elimination of metabolic waste products (Keul, J., et al., 1996).

During prolonged physical activity, the SNS activity is increased and this results in higher RMR and metabolic adaptations taking place in the skeletal muscle favor the process of lipid oxidation (Tremblay, A., 1994, Saris, W.H., 1995).

4.1 Hormonal adaptations at rest

4.1.1 Catecholamines

Plasma concentrations of epinephrine at rest are increased in athletes who have been training for years in comparison with sedentary subjects (Kær, M. et al., 1988). In addition, (Poehlman, E.T., et al., 1994) considered that endurance training in older persons has been found to increase basal levels of norepinephrine appearance into circulation. This has been associated with an increased RMR and fat oxidation.

Trained subjects present bradycardia at rest and during exercise. This adaptation to exercise training may be due to a decreased activity of sympathetic cardiac nerves, and due and increased parasympathetic activity (Williams, R.S. and Wallace, A.G., 1988).

4.1.2 Insulin and lipoprotein lipase

Insulin decreases the action of lipoprotein lipase in muscle and since the training stage declines insulin in plasma concentrations, this may be important for the maintenance of high levels of lipoprotein lipase (LPL) in skeletal muscle. Simsolo, R.B., et al, (1993) examined the regulation of lipoprotein lipase (LPL) in adipose tissue and skeletal muscle before and after detraining. After detraining, LPL was augmented in adipose tissue and lowered in skeletal muscle. Therefore, they concluded that high levels of physical activity may lead dietary fat to muscle for oxidation, rather than to adipose tissue for storage.

4.1.3 Cortisol and Testosterone

Bell, G., et al., (1997), investigated the response to serum testosterone (T) urinary cholesterol, (UC) and strength improvement in 6 men and 5 women. The training protocol consisted in strength training (ST) 3 times a week for 16 weeks. Additionally, a combined training program (CT) was appraised on a group of 14 men and 8 females performing the same training program plus endurance training 3 times a week on alternate days. The investigators observed no significant changes in T with either program. UC showed to be significant elevated at 8 weeks for men and prevailed as well after CT, but decreased to baseline levels after ST. In women, UC declined with the ST at 8 weeks but increased with both types of training (ST and CT) after 16 weeks. The investigators suggested that there are differences in strength and hormonal adaptations between genders.

4.2 Other benefits from chronic physical training

Trained athletes are more efficient in their use of metabolic fuels than are untrained, sedentary individuals. One of the factors involved in training is to

increase muscle fatty acid oxidation and the increase of the glycogen-glucose reserve in the muscle. Training or adaptation to exercise or work is also characterized by a decrease RQ due to an increase in the oxidation of lipids and the uptake of less glucose per minute (Berdanier, C., 1995).

Physical activity can affect both total energy intake and total energy expenditure. Physical activity also affects fat balance, because imbalances in total energy are substantial imbalances in fat. High levels of physical activity may facilitate individuals to attain fat and energy balances and achieve lower levels of body fat in comparison with individuals at lower levels of physical activity (Hill, J.O., 1996). Exercise may play an important role to defeat the impaired lipid oxidation in muscles of obese individuals (Saris, W.H., 1995).

4.3 Food Attitudes in exercisers and non-exercisers

Georgiou, C., et al., (1996) compared attitudes about food, recent dietary changes, and food choices of 319 young adults (18-24 y) who defined themselves as exercisers and non-exercisers. Exercisers considered is was more important to eat nutritious foods and ate more nutrient-dense, low fat foods (more grain and fruit intake) than non-exercisers.

Physically trained adults usually present faster movement speed than sedentary adults. Additionally, some studies in older adults involved in regular physical activities show better scores on psychomotor, cognitive and memory tests. (Bouchard, C. and Shepard, R., 1993).

There is a positive relationship between exercise habits and self-esteem for both children and adults. However, there is no attestation that in an over-trained state athletes hold a relationship between exercise and psychological well-being (Bouchard, C. and Shepard, R., 1993).

SUMMARY OF THE LITERATURE

In conclusion, the former literature review outlines:

1) The components involved in the energy balance equation.
2) Recalls previous studies on TRF in trained subjects which indicate disagreement.
3) Reviews the previous knowledge on TRF and its relationship with body composition and/or VO_{2max} which also disclose contradiction.
4) Evidences the insufficient documentation to arrive to any conclusion in female athletes.
5) Considers the possible methodology factors associated with the discrepant results in the past.
6) Contemplates the possible physiological adaptations to exercise training related with TRF.

AIM OF THE STUDY

1. The aim of this study was to measure the obligatory (OTRF) and facultative (FTRF) components of TRF in trained and untrained healthy women.

2. To correlate body composition and maximal aerobic power (VO_{2max}) with each component of TRF.

CHAPTER II: ARTICLE

Relationship of the Thermogenic Response to Food (TRF) with VO_{2max} and body composition in women

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ABSTRACT

The relation between TRF, VO_{2max} and body composition in women is poorly understood. These parameters were measured in 25 female volunteers (12 trained and 13 untrained subjects). Both components of the TRF: the obligatory component (OTRF) and the facultative component (FTRF) were explored by giving an oral test meal and an intragastric test meal on a second occasion. The FTRF was calculated as the difference between the oral meal and the intragastric meal. VO_{2max} was measured on a treadmill and body composition by underwater weighing. FTRF was not significantly different between trained and untrained subjects, but TRF (222.71 ± 63 vs 184.93 ± 50 kJ/6 h, $p = 0.03$) and OTRF (173.25 ± 37.66 vs 131.31 ± 36.46 kJ/6hrs $p = 0.004$) showed to be significantly higher in trained female subjects. Multiple regression analysis of both test meals, VO_{2max} and percentage of body fat including both groups (trained, untrained) was performed. OTRF was significantly related to VO_{2max} ($p = 0.04$; $r = 0.68$.) but not to the percentage of body fat. There was no relationship between TRF, VO_{2max} and percentage of body fat. These results suggest that trained subjects have a higher OTRF than untrained subjects and that the difference is related to their higher VO_{2max}.

Key words: Energy expenditure, thermic respond to food, runners. VO_{2max}, body composition.

INTRODUCTION

The thermogenic response to food (TRF) is the increase of the resting energy expenditure (REE) after food ingestion. In rodents, TRF has two components: an obligatory component which includes the process of digestion, absorption and storage of nutrients and a facultative component which is suggested to be related to oropharingeal stimulation (1,2,3). In humans, it has been shown that 30-40 % of facultative TRF was related to oropharyngeal stimulation (4) and that this component is under the control of the sympathetic nervous system (SNS) (5,6,7,8,9). These observations support the concept that a facultative component of TRF exists in humans (9,10) and that it is regulated in a similar manner as that of rodents.

The effects of exercise training on TRF are poorly understood. Some research suggests a higher TRF in trained subjects (11,12,13,14), while other studies have found a lower TRF (15,16,17,18) or no difference between the two groups (19,20). The disparity in the results could be explained by methodological differences in measuring TRF. To attain an acceptable reliability, it has been suggested a $\geq$ 5 h. measurement duration of the TRF (21, 22).

In addition, no study has considered both components of TRF and their relationship with VO_{2max} and body composition in women. The objective of this study was therefore to explore OTRF and FTRF in trained and untrained healthy women.

SUBJECTS AND METHODS

Twenty five (12 trained and 13 untrained) young (aged 18-35 y), healthy female subjects were recruited in this study. Trained subjects were distance runners or triathletes currently exercising ≥ 8 h/wk of aerobic exercise, had engaged in training for ≥ 2 y previously and had experienced at least provincial and national tournaments. This group was selected from 3 university sports centers in Montreal. The untrained subjects were involved for less than 1.5 h/week in aerobic exercise during the previous 2 years; their aerobic exercise, including walking and had never been enrolled in any athletic competition involving endurance activities. This group was selected from newspapers ads and local ads at the universities. Subjects were non-smokers, weight stable (± 2.5 kg) during the 6 months prior to the study, were not taking any medication known to affect metabolic rate, did not present any family history of diabetes or obesity and had a regular menstrual cycle (28-35 d). Trained and untrained subjects were matched for BMI (18-22 kg/m^2). Before the study, approval was obtained from the ethics committee of our institution.

Procedures for admission into the study

Before their engagement in the study, all subjects went through a preliminary session, during which they were informed of all the investigation procedures. During this session, a nasogastric tube (flexiflo no. 8 enteral feeding tube: Ross Laboratories, Columbus, OH) was inserted into the stomach without nasal anesthesia and left in place for about 10 minutes. The participants were also familiarized with the indirect calorimetry measurement and they went through a physical health exam as well. All subjects gave their written approval to participate in this study

Glucose tolerance test

Subjects arrived at the clinic after a 12 hour fast, a first blood sample was taken, then subjects ingested 75 g of glucose (Glucotrol, Rougier, Chambly, Canada), a second blood sample was taken at 120 minutes. The glucose level was measured by the glucose oxidase method. The insulin level was measured by radioimmunoassay (SERONO RIA kit, Immunocorp, Montreal, Canada).

Body composition

Body fat was assessed by densitometry and skinfold measurements. Residual lung volume was estimated using the close-circuit helium-dilution method (23). Body density was estimated by the underwater weighing method (24), a maximum of 7 trials were performed for each subject. Three trials with the least variability and providing the highest body density were averaged to obtain final body density. Skinfold-thickness was measured as the average of triplicate measurements made on the left side of the body with a Lange caliper (Cambridge Scientific Industries, Cambridge, MD) at 10 sites (chin, back, chest, side, waist, abdomen, arm, knee, calf, cheek). Fat free mass (FFM) and fat mass (FM) were computed according to the equation of Siri (25) and the equation of Allen (26).

Maximal treadmill exercise test

The maximal aerobic capacity was measured on a treadmill (Quinton Q65), The Léger-Boucher protocol was selected (27) using the modified formula to predict VO_{2max} on the treadmill from Léger-Mercier (28). Subjects were asked: to abstain from drinking alcohol or products containing caffeine 6 h prior to the test, not to eat a large meal before 3-4 h before the evaluation and trained subjects were asked to abstain from intense exercise 6 h before the test.

Subjects started to run at 8 km/hr during five minutes. Subsequently, the speed was increased by 1 km/hr every two minutes until exhaustion. Expired gases were measured by the breath by breath system (mass spectrometer, Marquette 1100 medical gas analyzer with a ventilation measurement module and the Marquette Calcul VO_2 software). HR was continuously monitored by using a portable chest strap monitor (Polar CIC Inc., Port Washington, NY).

Pre-experimental procedures before TRF tests

Subjects completed a 3-d dietary recall (2 week days and 1 weekend), to obtain mean total caloric intake and percentage of energy nutrients. Prior to each TRF test, subjects were asked to eat a high carbohydrate diet (60 % carbohydrate, 18 % protein and 22 % fat). To attain this objective, each subject was given a list of equivalents to follow during their meals.

Trained subjects were asked to train 24 hours before the REE and TRF tests and then avoid intense physical activity.
On two non-consecutive days from their follicular phase, subjects were asked to arrive at the laboratory with the minimum effort, fasted, at 8:00 am. They rested for 20 minutes before the procedures.

REE and TRF Tests

REE was measured for 30 min., subsequently they ingested one of the two randomly assigned tests (Table VII). Test 1 was the oral ingestion of a standard meal; test 2, the same blenderized meal was administered through a nasogastric tube, for a duration of 20 min. The tube was removed after the procedure.

Energy expenditure was measured by continuous indirect calorimetry with the use of a ventilated-hood system (Deltatrac Metabolic Monitor; SensorMedics,

Anaheim, CA). The calculation of oxygen consumption (VO_2), and carbon dioxide production (VCO_2) were made from continuous measurements of oxygen and CO_2 concentrations in inspired and expired air diluted in a constant flow generated by the analyzer. Accuracy and precision of the system measured in vitro in our laboratory is 99% and 2%, respectively (21). Energy expenditure was measured for 30 min. every hour, over a 7 hour period (including a 30 min. REE measurement). Calculations of energy expenditure were made from VO_2 and VCO_2 recorded during the last 20 min. of each 30 min period.

Urine collection was initiated following voiding at 8:20 am , and lasted until the end of the procedure. Total nitrogen concentrations were measured by chemiluminescence with an ANTEK analyzer (ANTEK, Houston).

Calculations and statistics

Energy expenditure was calculated from VCO_2 , VO_2 and urinary nitrogen using the Weir equation (29).

$$EE\ (kJ/24h) = (3.941*VO_2 + 1.106*VCO_2 - 2.17*N)*4.18$$

Data was analyzed by ANOVA, when the analysis was significant, we established a multiple comparison between groups (Tukey "B" method). Paired t tests were used to compare substrate oxidation between-meals (oral vs intragastric) within individuals. Unpaired t tests were used to compare substrate oxidation between groups (trained vs untrained) (30). Calculations were performed using the Statistical Package for Social Sciences (SPSS V4.01, SPSS Inc, Chicago, IL).

RESULTS

Average aerobic exercise in trained subjects was 13.57 ± 2.8 h/week, the time since they were involved in competitive activities was 4.3 ± 1.6 y. The physical characteristics of the trained and untrained subjects are shown in (Table VIII), There were no differences in age, weight, height and BMI between both groups. Body fat, triceps and fat mass and resting heart rate were lower in trained subjects. Fat free mass, and REE were higher in trained subjects. When REE was expressed in kJ x 24h /weight (kg), REE was higher in trained than untrained subjects (101.00 ± 12.34 vs 92.56 ± 6.86 $p = 0.04$), but when REE was expressed in kJ x 24 h/FFM (kg), the difference was not significant (116.97 ± 11.23 vs . 110.51 ± 6.64, $p = 0.09$). VO_{2max} was greater in trained subjects. There were no significant differences in plasma insulin and glucose concentrations (data not shown).

The 3 d dietary recall revealed no significant difference in the percentage of total energy from carbohydrates or protein consumption. Trained subjects consumption of total energy from lipids was lower (20.85 ± 4.72 vs 25.03 ± 4.17; $p = 0.05$). Total energy intake was higher in trained subjects (7459.78 ± 1389.09 vs 6663.02 ± 852.72 kJ/d, $p = 0.01$).

Figure 4, shows the substrate oxidation of glucose (Figure 4-A), fatty acids (Figure 4-B) and protein (Figure 4-C) from both test meals in trained and untrained subjects. The glucose oxidation at resting metabolic rate and with the intragastric meal showed no significant difference between groups. But, glucose oxidation was lower in the trained group with the oral meal in comparison with the untrained group (2.81 ± 0.66 vs 3.13 ± 0.54 mg/kg/min, $p < 0.05$). For the fatty acid oxidation, the trained group showed higher oxidation at resting metabolic rate (0.52 ± 0.3 vs 0.41 ± 0.2 mg/kg/min, $p = 0.05$) and with the intragastric meal in comparison with the untrained group (0.65 ± 0.2 vs .51 ±

0.2 mg/kg/min, $p < 0.05$). The fatty acid oxidation showed to be almost doubled in trained subjects with the oral meal in comparison with the untrained group (0.73 ± 0.3 vs 0.38 ± 0.1 mg/kg/min, $p < 0.01$). Protein oxidation was higher in trained subjects with the oral meal (0.76 ± 0.1 vs 0.63 ± 0.3 mg/kg/min, $p < 0.05$) and was even higher with the intragastric meal in this group compared with untrained subjects (0.81 ± 0.2 vs 0.59 ± 0.3 mg/kg/min, $p< 0.01$). In trained subjects, the correlations for fatty acid oxidation were as follows: oral diet: $r = -0.73$; tube feeding: $r = -0.66$. For the untrained subjects these correlations were: oral diet: $r = -0.90$; tube feeding $r = -0.86$. Concerning protein oxidation, OTRF was 6.6% higher than TRF in trained subjects. In untrained subjects, OTRF was decreased 6.3 % from TRF. When comparing protein oxidation between groups, trained subjects exhibited higher oxidation with TRF and OTRF (+ 20 and + 37 %, respectively) in comparison with untrained subjects.

Trained subjects showed higher TRF and OTRF in comparison with untrained subjects [TRF trained subjects: 222.71 ± 63; untrained subjects: 184.93 ± 50 kJ/6 h, $p = 0.03$]. [OTRF trained subjects: 173.2 ± 38; untrained subjects: 131.30 ± 36 kJ/6 h, $p = 004$)]. The variance was smaller in OTRF than in FTRF (Figure 5-A-B-C).

Figure 6-, shows the response of each group: trained (figure 6-A) and untrained (figure 6-B) to oral and tube feeding. Both groups exhibited significant lower energy expenditure after tube feeding (-22.2 % and -29.0 % of the ingested meal respectively, $p = 0.01$).

A multiple regression analysis showed no relationship between TRF, VO_{2max} and percentage of body fat. But, OTRF was significantly correlated to VO_{2max} ($p = 0.04$; $r = 0.68$) (Figure 7).

DISCUSSION

Previous surveys measuring TRF in trained and untrained subjects at rest manifested discrepancies. Methodological issues such as heterogenecity of the group, small number of subjects studied, "mixed" gender, lack of criterion to define trained subject, various time lapse between testing and the last bout of exercise, inconsistent duration of TRF measurement, various amount and type of meal, may explain this discrepancies (11,12,13,14,15,16,17,18). The literature is limited and controversial concerning TRF in female athletes as well (14, 17, 20).

In this study, the trained and untrained groups were matched for age, weight, height and BMI. Our results revealed a slightly significant difference of TRF and no difference in FTRF, but there was a significant difference with the OTRF.

Since there variance of the OTRF was very small between groups, and the variance for the FTRF was higher, our findings of a correlation only between VO_{2max} and OTRF suggests that the facultative part increases the variance of TRF. Some authors had proposed that FTRF may be partially influenced by the SNS in animal and man (31,32,33,34,35). Astrup et al., suggested that the sympathetic component may stimulate thermogenesis in the heart, liver and white adipose tissue (35) and that approximately 60 % of the thermic effect of carbohydrate was situated in muscle. Its suggested distribution was 45% for obligatory thermogenesis and 15 % for facultative thermogenesis (36).

In this study, FTRF was calculated from the difference between an oral and intragastric meal. FTRF showed to be almost equal in trained and untrained subjects. The results of FTRF are in concordance with previous studies in our laboratory on lean subjects (9,10,39). The average decrease when the meal was

administered by intragastric tube was 22.2% in trained subjects and 29% in untrained subjects. In a previous study, de Jonge and Garrel suggested a role for SNS in FTRF in normal weight men and women (9). Our hypothesis was that the trained group would present lower TRF and that this decrease would be due to the facultative component. Surprisingly, our results indicated a slightly higher TRF with an average increase of 20 % over untrained subjects. However, the explanation to this may be the significantly higher OTRF observed in trained subjects, possibly related to a higher parasympathetic nervous system tone (PNS) (37,38). The fact that trained subjects disclosed a significant decreased heart rate at rest, confirms this fact. There may be a balance between SNS and PNS of the autonomic nervous system in resting conditions in trained subjects (34,37).

We found a high correlation between % fat and VO_{2max}, (-0.686, $p = 0.005$) However the correlation between OTRF and VO_{2max} is analogous whether or not we take into account the % of body fat. In the past decade, some studies suggested a positive association between VO_{2max} and higher TRF in trained subjects (13, 14). Others suggested a lower TRF in relation to VO_{2max} (17, 40). From these studies, LeBlanc et al., measured female subjects in the post-ovulatory state and TRF was measured only for a short period of time (2 h) (17). Poehlman et al., studied 28 young males, and he proposed a curvilinear relationship between VO_{2max} and TRF in which highly trained subjects showed a lower TRF only after 70 ml/kg/min (15). This statement may be controversial due to the short time used in measuring TRF in this study. Toth et al., suggested that the loss of FFM and the increase in adiposity may be conducive to a decrease in peak VO_{2max} with age in men and women (41). Poehlman appraised the influence of eight wk endurance training on basal levels of fat oxidation and its analogy with changes in norepinephrine (NE) kinetics in 8 senior men and 10 senior women. Endurance training increased their peak VO_{2max} by 11 % and REE by 7 %, both parameters with a $p < 0.01$ (42).

Bouchard et al., observed that the genetic effect for TRF was correspondent to at least 40-50 % of the variation of energy dissipated after a 4 h test meal (43). The fact that the untrained group displayed a correlation between OTRF and VO_{2max} ($r = 0.617$, $p = 0.01$) suggests that due to their genetic background, untrained subjects with a higher VO_{2max} may have less difficulty controlling their body weight.

At present, the literature concerning substrate oxidation has been mostly measured during exercise periods. Hagerman, measured elite runners during marathon training and competition. He suggested that the power of type I and IIA skeletal muscle fibers significantly develop storage capacity for intramuscular lipids and increase mitochondrial density to increase fat oxidation. He also proposed that proteins may contribute as much as 5-10 % of the total energy to marathon achievement (44). Tarnopolsky et al. studied the effects of gender on substrate utilization during prolonged submaximal exercise in 6 males and 6 females with a maximal aerobic capacity of 63.5 ± 1.1 and 57.8 ± 1.8 mg/kg/min respectively. When expressing VO_{2max} in ml/kg FFM/min the difference was nil (74.9 ± 0.9 vs 74.7 ± 1.7). They found lower insulin concentration and higher epinephrine concentration in males. Also they concluded that during moderate-intensity duration exercise, females manifested greater lipid utilization and less carbohydrate and protein metabolism than matched trained athletes (45). In another study, Tarnopolsky et al. studied 7 males and 8 females endurance athletes in response to an increased dietary carbohydrate from 55-60 % to 75 % of energy intake for a period of 4 days. Their results showed that men increased 41% muscle glycogen in response to the dietary manipulation whereas, in contrast, women did not increase glycogen concentration (0%). In addition, these authors confirmed their previous findings regarding greater lipid and lower carbohydrate and protein oxidation in women vs men (46).

Substrate oxidation of OTRF in trained subjects,. indicated a decrease of 10.3 % for the glucose oxidation and a decrease of 10.9 % for the lipid oxidation in comparison with TRF. The untrained group showed a decrease of 12.9 % for glucose oxidation with OTRF in comparison with TRF. For lipid oxidation. TRF exhibited a reduction of 25 % in comparison with OTRF. Untrained subjects presented higher RQ through all measurements with both test meals in comparison with trained subjects ($p < 0.05$). These results suggest that FTRF uses more glucose.

When comparing each group and their response to both test meals, their RQ increased significantly for the first hour of measurement in both groups, ($p = 0.001$). Correlation between glucose oxidation with each test meal, suggested a higher correlation with tube feeding in trained and untrained subjects ($r = 0.98$ and $r = 1.0$ respectively). The glucose oxidation with the oral test meal indicated a lower correlation in trained subjects compared with untrained subjects ($r = 0.78$ and $r = 0.93$ respectively).

When lipid oxidation was analyzed, trained subjects increased their substrate oxidation to 92 % in comparison with the untrained group for TRF. As with OTRF, this substrate increased only 27 %.

Trained subjects seem to be less efficient in conserving energy. The fact that they oxidize more lipids and protein than untrained subjects costs them more energy to process these nutrients.

In conclusion we have found that VO_{2max} is correlated to OTRF and also that metabolic cost of nutrient utilization may be predisposed by genetic factors.

RECOMMENDATIONS

Chronic exercise training may provide different benefits related to energy balance in women, such as development of a higher VO_{2max} which may increase PNS and consequently OTRF and TRF. Another advantage of exercise training is the increase in lipid oxidation. Maintaining chronic exercise training may attenuate the decreased VO_{2max} experienced with aging.

For future studies, it is recommended that standards be developed and used in order to benefit from these well controlled studies. Some factors, such as assessment of TRF and its both components, by indirect calorimetry should be measured for at least 6 h. TRF. More studies in female athletes may be beneficial to understand the benefits of chronic exercise training and TRF.

TABLES AND FIGUES
(ARTICLE)

(Table VII)

Meal composition

		Oral	Liquid
Milk 2%	(ml)	200	200
Orange juice	(ml)	125	125
Cream 10%	(ml)	20	20
Butter	(g)	2	4
Eggs		3	3
White bread	(g)	60	-
Cream of wheat	(g)	-	265
Sugar	(g)	5	10
carbohydrate	(g)	60.1	60.5
(% of energy)		40.4	40.5
protein	(g)	29.9	29
(% of energy)		20.1	19.5
fat	(g)	26.1	26.5
(% of energy)		39.5	40.0
Energy	(kJ)	2490	2497

(Table VIII)

Subjects characteristics

	Trained	Untrained	p =
Age (y)	25.64 ± 4.30	23.85 ± 4.38	N/S
Weight (Kg)	56.45 ± 5.76	55.68 ± 5.27	N/S
Height (cm)	164.86 ± 6.26	163.86 ± 5.54	N/S
BMI (kg/m2)	21.00 ± 2.23	20.81 ± 1.65	N/S
Hydrodensity (% BF)	12.51 ± 3.24	16.82 ± 5.02	0.0026
10 skinfolds (% BF)	13.08 ± 3.76	16.35 ± 3.43	0.0089
Triceps (mm)	10.21 ± 2.3	13.25 ± 3.63	0.0050
Fat mass (kg)	7.07 ± 1.97	9.47 ± 2.89	0.0012
Fat Free Mass (kg)	50.06 ± 5.33	45.87 ± 4.15	0.05
VO2max [ml (kg *min)]	53.63 ± 5.18	42.45 ± 4.78	0.000
Resting HR (beats/min)	59.35 ± 7.22	69.71 ± 8.83	0.005
REE kJ/h	234.87 ± 23.75	212.69 ± 21.62	0.02

Substrate oxidation in trained and untrained females

Figure 4-A Glucose oxidation

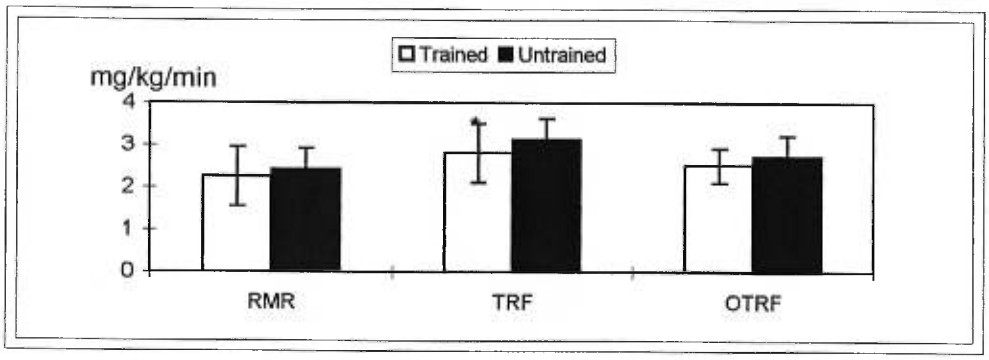


Figure 4-B Fatty acids oxidation

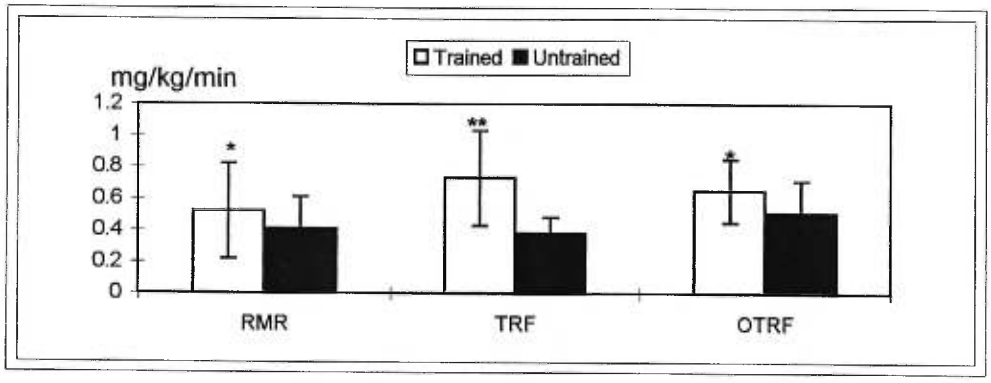
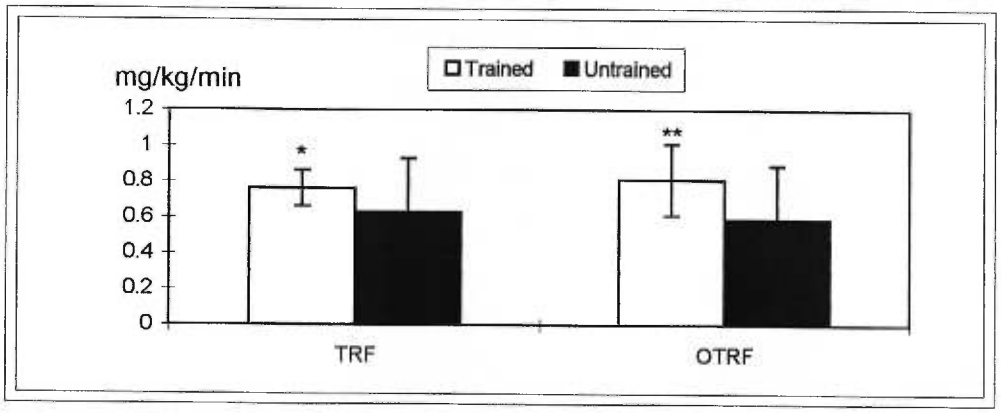


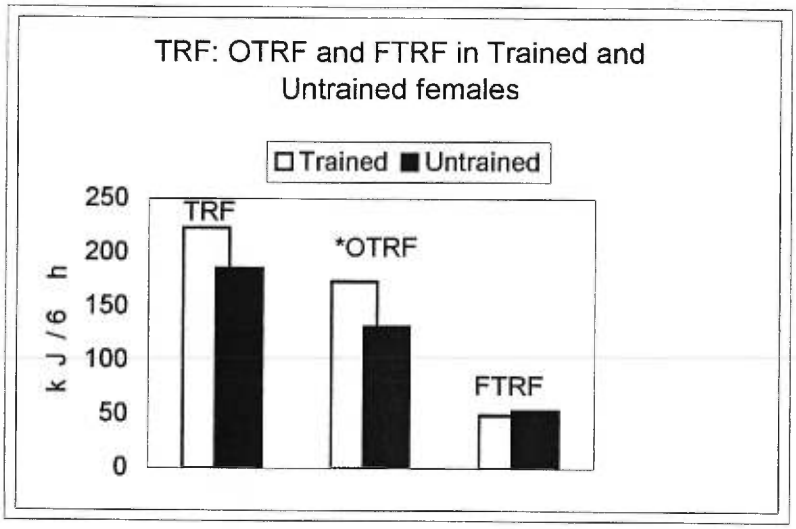
Figure 4-C Protein oxidation



* $p < 0.05$ mg = miligrams kg = kilogram min = minute
** $p < 0.01$
RMR = Resting metabolic rate
TRF = Thermogenic response to food
OTRF = Obligatory thermogenic response to food

Figure 5-A

TRF:OTRF and FTRF in trained and untrained females



TRF = Total thermogenic response to food
OTRF = Obligatory thermogenic response to food
FTRF = Facultative thermogenic response to food
kJ/h = Kilojoules per hour

* $p < 0.05$
** $p < 0.01$

Figure 5-B

TRF in trained vs untrained females

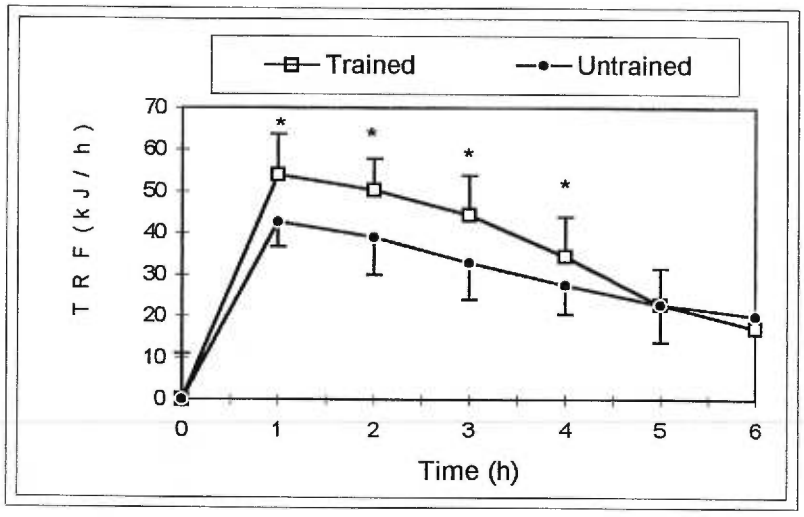
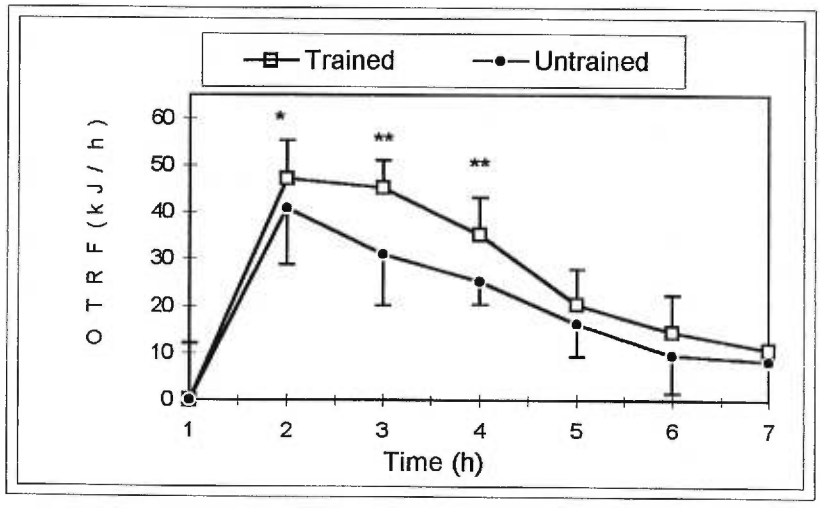


Figure 5-C

OTRF in trained vs untrained females



* $p < 0.05$
** $p < 0.01$

TRF = Total thermogenic response to food
OTRF = Obligatory thermogenic response to food
kJ/ h = Kilojoules per hour
h = hour

Figure 6-A

TRF vs OTRF in trained females

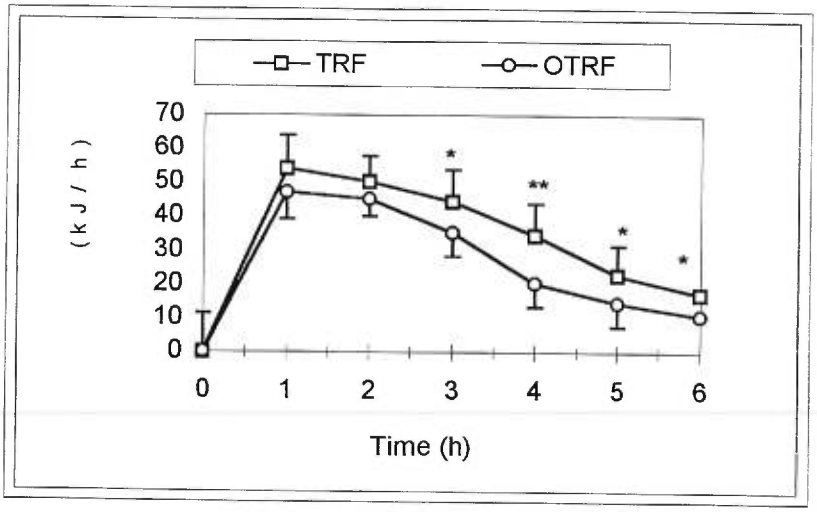
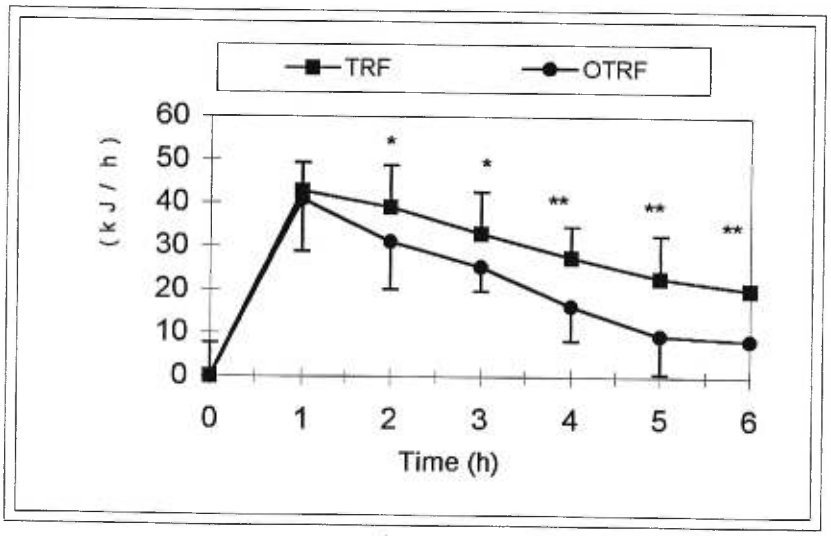


Figure 6-B

TRF vs OTRF in untrained females

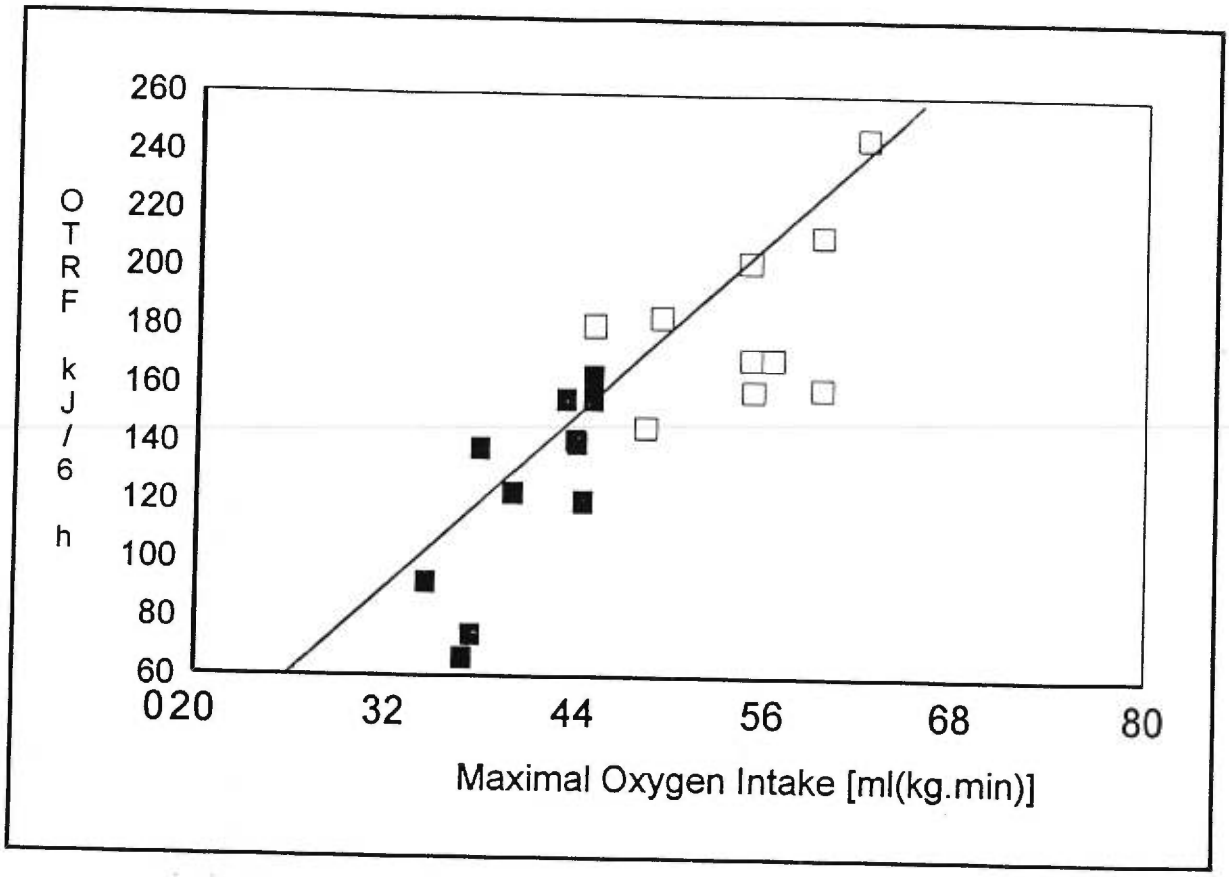


* p < 0.05
** p < 0.01

TRF = Total thermogenic response to food
OTRF = Obligatory thermogenic response to food
kJ / h = kilojoules per hour
h = hour

Figure 7

Correlation of OTRF and VO_{2max} in females



Correlation of OTRF and VO_{2max}	r = 0.68	p = 0.04
Correlation runners	r = 0.45	p = 0.06
Correlation untrained	r = 0.62	p = 0.01

OTRF	=	Obligatory thermogenic response to food
kJ/6 h	=	Kilojoules per 6 hours
VO_{2max}	=	Maximal oxygen intake
ml(kg • min)	=	Milliliters per kilogram per minute

□ Trained (runners) ■ untrained (sedentary)

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CHAPTER III: GENERAL DISCUSSION

Introduction

Our study is the first study to appraise both components of TRF (obligatory and facultative) in trained and untrained healthy females and to correlate both components of TRF with body composition and VO_{2max}.

Past surveys focusing on measuring only TRF in trained subjects at rest revealed discrepancies. The controversy from these studies may be explained by methodological factors such as homogenous groups, number of subjects studied, age, gender, body composition, criteria to define trained subjects, TRF measurement after the last bout of exercise, time spent measuring TRF, amount and type of meal, genetic factors, measure of both components of TRF.

1. Homogenous groups, definition of trained subject and type of sport.

It may be recommended to gather subjects, according to age, gender, type of sport, duration and intensity of training and food habits to create more homogenous samples. Different types of sports acquire different average body composition, average VO_{2max}'s and metabolic adaptations to training . According to factors such as type of exercise training, adaptability to the training environment, swimmers may present higher levels of body fat than runners and lower levels of VO_{2max}. (Table IX and X).

Table IX

Average body fat of male and female athletes

Sport	Females % Body Fat	Males % Body Fat
Ballet dancing	13-20	8-14
Basketball	20-27	7-11
Body building	9-13	6-9
Cycling	15	8-10
Gymnastics	10-17	5-10
Racquetball	14	8-9
Rock climbing	10-15	5-10
Alpine skiing	21	7-14
Cross-country skiing	16-22	7-12
Speed skating	15-24	11
Swimming	**14-24**	9-12
Tennis	20	15-16
Long distance runners	**10-19**	6-13
Middle distance runners	10-14	7-12
Volleyball	16-25	11-12
Rowing	14-18	8-15
Triathlon	7-17	5-11

Adapted from Heyward, V.H. and Stolarczyk , L. (1996)

Table X

Comparative VO_{2max} values for athletes in various sports

Sport	
Women	**VO_2 (ml • kg^{-1} • min^{-1})**
Cross-country skiers	56-74
Average marathon runners	**50-55**
Olympic distance runners	55-62
Pentathletes	43-52
Rowers	41-58
Distance swimmers	**45-60**
Speed skaters	40-52
Sprinters	38-52
Basketball players	35-45
Men	**VO_2 (ml • kg^{-1} • min^{-1})**
Cross-country skiers	70-94
Average runners	55-62
Rowers	58-75
Bicyclists	55-70
Long-distance swimmers	48-68
Speed skaters	50-75
Ice hockey players	50-60
Tennis players	42-56

Adapted from Wilmore, J.H., (1977)

1.1 Sub-study

In a sub-study, 12 swimmers were also assessed for body composition, VO_{2max}'s and TRF (Table XI). Body percentage of fat was significantly different in runners (12.51 ± 3.2 %, p = 0.003) than swimmers and control group (18.79 ± 4.8 and 16.82 ± 5.0 %) respectively. Runners demonstrated significantly higher VO_{2max} values than swimmers and control (53.62 ± 5.2 ml/kg/min, p = 0.0000). Swimmers presented significantly higher VO_{2max} values than untrained subjects (48.38 ± 4.4 vs 42.44 ± 4.7, p = 0.05).

TRF in swimmers resulted to be alike from the control (untrained) group (183 ± 43 vs 184.93 ± 50 kJ/6 hours) respectively, while TRF showed a slight difference in runners in comparison with swimmers and untrained subjects (222.71 ± 63 kJ/6h, p = 0.07). Even though swimmers held higher FFM in comparison with runners and untrained subjects (51.54 ± 3.8, p = 0.02 vs 48.7 ± 4.4 and 46.49 ± 4.3 kg FFM) respectively, they also accounted for significantly higher of FM in comparison with runners and untrained subjects (11.58 ± 3.5 vs 7.07 and 9.47 ± 2.9 kg of FM, p = 0.001). Body composition and VO_{2max} values from our female swimmers, may be comparable with the reference values for female athletes of this specific type of sport (Table IX and X) .

Annex XI

Results

	Swimmers	Runners	Control
Age (years)	24.75 ± 4.07	25.64 ± 4.3	23.85 ± 4.38
Weight (kg)	61.12 ± 4.79 [a]	56.45 ± 5.8	55.68 ± 5.3
Height (cm)	167.83 ± 4.2	164.86 ± 6.3	163.86 ± 5.5
BMI (kg/m^2)	21.84 ± 1.4	21.00 ± 2.2	20.8 ± 1.6
% Body Fat	18.79 ± 4.8	12.51 ± 3.2 [c]	16.82 ± 5.0
FFM (kg)	49.55 ± 3.8	50.06 ± 5.3	45.87 ± 4.1 [a]
FM (kg)	11.58 ± 3.5 [c]	7.07 ± 2.0	9.47 ± 2.9
Triceps (mm)	14.16 ± 3.1	10.21 ± 2.2 [c]	13.25 ± 3.6
VO_{2max} ($ml \bullet kg^{-1} \bullet min^{-1}$)	48.38 ± 4.4 [a]	53.62 ± 5.2 [d]	42.44 ± 4.7
HR (beats/min)	64.5 ± 7	59.35 ± 7	69.7 ± 9 [c]
BMR (kJ/h)	229.0 ± 83	234.87 ± 23 [a]	212.69 ± 22
TRF(kJ/6h)	183 ± 43	222.71 ± 63	184.93 ± 50
% from ingested meal	7.25 ± 1.5	8.93 ± 2.5	7.28 ± 1.9
OTRF (kJ/6 h)	-	173.25 ± 38 [b]	131.31 ± 36
% from ingested meal	-	6.91 ± 1.4 [b]	5.23 ± 1.3
Insulin (0min)	67.80 ± 22	78.07 ± 20	78.38 ± 17
Insulin Time (120 min)	180.30 ± 40	193.28 ± 91	229.77 ± 96
Habitual kJ/day	8199.26 ± 1397[c]	7459.78 ± 1389	6660.93 ± 870
% from carbohydrate	56.58 ± 8.6	61.43 ± 5.6	57.78 ± 4.17
% from protein	17.50 ± 1.9	17.71 ± 2.0	17.07 ± 2.7
% lipids	26.00 ± 7.7 [a]	20.86 ± 4.7	25.00 ± 4.17

Insulin time 0 min and time 120 min = mmol/L

[a] $p < 0.05$
[b] $p < 0.01$
[c] $p < 0.005$
[d] $p = 0.0000$

2. Gender

It is important to differentiate that women present lower RMR than men of similar age. The diminished RMR is independent of differences in body composition and aerobic fitness (Arciero, P., et al., 1993). We also must consider the gender differences when appraising substrate oxidation at rest and during exercise in trained and untrained subjects (Tarnopolsky, L.J., et al., 1990, Tarnopolsky, M. A., et al, 1992, Phillips, S.M., et al, 1993, Ruby, B.C., 1994; Tarnopolsky, M.A., 1995).

The majority of the literature information is based on studies in men. Only three studies may have considered to study female athletes. Results of these studies, however, have only created controversy because of methodological discrepancies as discussed below.

Leblanc, J., et al (1984), studied 30 female subjects (10 competition athletes, 10 moderate active and 10 sedentary subjects). Subjects were measured on the post-ovulatory phase of their menstrual cycle. This study does not consider the length of time after the last bout of exercise for the TRF measurement and TRF was determined only after 2 hours of measurement. Their results suggested a diminished TRF in the competition female athletes correlated with a lower carbohydrate oxidation.

Owen, E.O., et al, (1986), studied 44 healthy women with a wide range of ages (18-65 years) and body weights (43-143 kg). Only eight athletes were included in the study. Their study do not mention time after the last bout of exercise for the trained subjects. The menstrual phase in which subjects were measured is not mentioned as well. The study concluded that TRF was the same in trained and untrained women.

The study from Davis, J.R., et al (1983), studied three males and three females after 12-16 weeks of training. The TRF measurement took place after 36 hours post-exercise, the duration of the measurement for the TRF lasted only 2.5 hours. The study did not mention in what phase of their menstrual cycle women were measured. They concluded that endurance training increased the magnitude of TRF in proportion to improvement in aerobic capacity. The increased TRF was associated with diminished body fat in males but not in females. Past studies exploring TRF and its relationship with body composition and/or VO_{2max} in women were unclear and needed further research.

Our results indicated that trained subjects showed higher TRF than untrained subjects (222.71 ± 63 vs 184.93 ± 50 kJ/6 hours, p = 0.03), respectively. OTRF showed also to be significantly higher in trained subjects compared with untrained subjects (173.25 ± 38 vs 131.30 ± 36 kJ/6 hours, p = 0.004), respectively. Multiple regression analysis showed no relationship between TRF, VO_{2max} and percentage of body fat. Only OTRF was significantly related to VO_{2max} (Trained: r = 0.454, p = 0.06; untrained: r = 0.617, p = 0.01; mixed r =0.68, p = 0.04). Both groups showed significant lower energy expenditure after tube feeding (trained: -22.2 %, untrained - 29.0 %) of the ingested meal. Lipid oxidation was almost double in trained subjects than untrained subjects with the oral meal (0.73 ± 0.35 vs 0.38 ± 0.14 mg/kg/min, p = 0.005).

3. Age-related decreased FFM

Changes in stroke volume, maximal heart rate, and arteriovenous oxygen difference are important factors in age-related diminution of VO_{2max}. Toth, M.J., et al., (1994), found that maximal heart rate may be identified as the strongest predictor of the decline VO_{2max} with age in men (r = 0.07) and in women (0.62). Their main finding was that the loss of FFM and the increase in body fat (identified with aging) are strongly and independently associated with

the decline VO_{2max}. Calles-Escandón, J., et al., (1995), investigating basal fat oxidation in aging women, indicated that the decline in FFM was associated with decline in basal fat oxidation. The investigators acknowledged that their data may not be applicable to male subjects.

4. Body composition

Poehlman et al (1988) pointed out that body composition alone, may not register the differences observed between well-trained subjects and untrained subjects in response to food. In disagreement, Segal et al., (1990), trained lean and obese subjects and concluded that the percentage of body fat was an excellent predictor of TRF ($r = -0.74$, $p < 0.01$) between body composition variables involving body weight, percentage of body fat, FFM and aerobic fitness. Both studies did not analyzed both components of TRF to acknowledge which component was related to TRF, for this reason, this controversy needed further examination.

5. Time after the last bout of exercise

When studying exercise-trained individuals after 12-16 hours post exercise, the magnitude of TRF may be significantly lower (Table IV). Studies in which TRF was evaluated thirty six hours or more after the last training session displayed an increase TRF (Table IV). Thörne, A. and Wahren, J., (1989) studied 14 male subjects (7 trained and 7 sedentary). Trained subjects were measured 36 hours after their last training session. TRF was measured after a liquid test meal (17 % kJ protein, 28 % kJ lipids and 55 % kJ carbohydrates). The TRF procedure lasted only 3 h. Their results indicated that there was no difference between the well-trained subjects' and sedentary subjects' response to TRF. In other words, their study did not support TRF as proportional to aerobic capacity in healthy young men. Their results can be questioned due to the small sample size, time of

measurement for the TRF after the last bout of exercise, the time spent on the measure of TRF, lack of measurement of both components of TRF to detect which one may be involved related to aerobic capacity.

Davis, J.R., et al., (1983) measured 16 women and 10 men with a range of ages from 21-42 years. The authors describe the subjects studied as in good physical health; one third of them exercised regularly (not specific), only one of them was a marathon runner in training. Afterwards they choose 3 men and 3 women and trained them for 12- 16 weeks. Subjects were measured 36 h after the last bout of exercise and TRF was measured only for 2.5 hours. Their results suggested a positive correlation with aerobic capacity (VO_{2max}) and TRF and that body percentage of fat was negatively correlated with aerobic capacity. Probably the genetic background of these individuals may have promoted these results. Our study agrees with their results but furthermore, showed which of the TRF component is related to VO_{2max}.

6.Time for measurement of TRF

Past studies investing TRF in athletes did not take enough time to accomplish a valid appraisal of TRF (Table IV). The incomplete measurement duration of TRF may lead to errors. To acquire an acceptable reliability, we measured TRF for 6 hours as suggested by (Houde-Nadeau, M., et al, 1993; Reed, G.W. and Hill., J.O., 1996). Also, to ensure the registration of both components of TRF as suggested by (Astrup, A.V., et al, 1990).

7. Meal composition and meal size

The composition of the test meal may influence the magnitude and duration of TRF. Welle, S. et al., (1981) documented that fat and protein produce a later and more gradual metabolic rise than carbohydrates. Additionally, they

suggested that protein and fat ingestion did not modify SNS activity, as estimated by plasma norepinephrine (NE) levels, but glucose intake increased NE levels. Schwartz, R.S., et al, (1987), concluded that the increased SNS activity, was significantly correlated ($r = 0.57$, $p < 0.01$) to the increment in energy expenditure after a meal.

Hill, O.J., et al., (1984), suggested an outspread reaction from non-obese men in response to any given meal. They also observed widely inconsistent increments of TRF in relation to an increase in meal size. Additionally, this study recognized VO_{2max} as one factor that may account for some of the individual variations in TRF.

8. Appraisal of both components of TRF.

No previous studies had considered both components of TRF in trained and untrained female subjects. Consequently, at present, there may be no point of reference for this factor.

9. Genetic factors

Bouchard, C., et al, (1989), concluded that the genetic effect on TRF may correspond to at least 40-50 %.. Even though, our study do not measured the genetic factor itself, it may be suggested that the individual variation detected in TRF due to exercise training may be explained by genetic factors. When correlating OTRF with VO_{2max}, the total correlation including both groups (trained and untrained) was ($r = 0.68$, $p = 0.04$; the correlation for the group of runners was $r = 0.454$, $p = 0.06$ and the correlation for the untrained group computed $r = 0.617$, $p = 0.01$). In other words, in spite of the highly training qualities of runners, specifically, their significantly higher VO_{2max}, the possible

genetic background in untrained subjects may account for the individual variation in response to OTRF.

SUMMARY OF THE METHODOLOGY USED IN THIS STUDY

It may be suggested that our study presented a stricter control in methodology in comparison with past studies on TRF. The different factors may be explain as follows:

1. The results from our study, suggested homogenous groups according with age, weight, height and BMI.
2. Trained subjects were engaged in competitive activities for at least the past 4 years. Their aerobic training was about 14 hours/ week.
3. Trained subjects were measured 24 hours after the last training session.
4. Untrained subjects did not exceeded 1.5 hours of aerobic exercise/ week, including walking.
5. A preliminary session was previewed to avoid the effects of stress in our results.
6. Three days before TRF test, subjects followed a high carbohydrate diet.
7. All subjects received the same amount and type of diet.
8. The tube feeding contained the same amount of nutrients as the oral meal.
9. TRF (oral and tube feeding tests) were measured during the follicular phase of the menstrual cycle in a randomized order.
10. TRF measurements lasted for 6 hours after RMR measurement on each test.

GENERAL CONCLUSION

These results suggest that the metabolic cost of nutrient utilization is related to VO_{2max} regardless body composition. Metabolic adaptations and/or cardiovascular adaptations due to training may influence the digestion, absorption, and storage of nutrients and consequently, may result in increased TRF.

The group of swimmers was not assessed for both components of TRF in this study. This factor, may have assisted us to corroborate the correlation of OTRF with VO_{2max}.

The topic of aerobic fitness and the thermogenic effect of food offers some inspiring and motivating opportunities for studies to answer important and practical questions of considerable relevance to human health.

An increase of habitual activity may be the most effective preventive approach for North Americans to decrease obesity and its related causes of morbidity and mortality.

ANNEX I

HOTEL - DIEU DE MONTRÉAL

FORMULE DE CONSENTEMENT

FEUILLET D'INFORMATIONS DESTINÉ AUX SUJETS

TITRE DE L'ÉTUDE
Une étude comparative de la thermogénèse induite par l'alimentation chez des femmes athlètes et chez des femmes sédentaires.

INVESTIGATEURS
Dr. Dominique Garrel, M.D. et Marielle Ledoux, Ph.D.

BUT DE L'ÉTUDE
Explorer la phase obligatoire et facultative de la thermogénèse induite par l'alimentation après un repas hypoglucidique et après un repas hyperglucidique.

DESCRIPTION DE L'ÉTUDE
La séquence des activités à faire avec chaque sujet sera la suivante:

1. Séance d'entraînement pour familiariser les sujets avec le masque et le tube nasogastrique.

2. Un examen médical complet sera effectué avant l'entrée dans le protocole.

3. Mesure de la composition corporelle. Cette mesure sera faite par la méthode de pesée sous l'eau. Votre poids et votre pourcentage de graisse seront mesurés pendant 3 immersions totales dans l'eau. Cette mesure aura lieu au Département d'Éducation Physique de l'Université de Montréal.

4. Mesure de la capacité aérobique maximale (VO_{2max}). Cette mesure sera faite sur un tapis roulant en mesurant votre consommation d'oxygène. La vitesse du tapis roulant sera augmentée progressivement jusqu'à épuisement. Cette mesure aura lieu au Département d'Éducation Physique de l'Université de Montréal.

5. Mesure de votre métabolisme de repos. Cette séance sera prise en position allongée à l'aide d'un masque transparent dans lequel vous pouvez respirer normalement.

6. Mesure de la tolérance au glucose. Au début de cette mesure, un échantillon sanguin sera pris à jeun. Par la suite, vous devez ingérer une boisson contenant 75 g. de glucose; 120 min après l'ingestion du glucose une deuxième prise de sang sera effectuée.

7. L'étude comporte 2 séances de sept heures chacune. Au cours de chacune de ces séances, la thermogénèse induite par l'alimentation sera mesurée à partir de la consommation d'oxygène, de la production de gaz carbonique, et de l'élimination urinaire d'azote. L'ordre des séances sera choisi au hasard. Vous ne saurez pas le type de repas (oral ou liquide) prévu pour la séance.

Séances:
A. Repas oral.
B. Repas liquide.

TEMPS APPROXIMATIF POUR CHAQUE ACTIVITÉ AVEC LES SUJETS

JOUR	ACTIVITÉ	TEMPS APPROXIMATIF
1	1 et 2	45 minutes
2	3 et 4	2 heures
3	5 et 6	3 heures
4	7A	7 heures
5	7B	7 heures

Remarque: S'il est nécessaire, l'ordre des activités pourrait être modifié.

RENSEIGNEMENTS PERTINENTS

1. Pour les athlètes: il est important de ne pas vous entraîner 24 heures avant chaque séance.

2. Vous devrez être à jeun depuis 20:00 h (8:00 p.m.) la veille de la séance. Vous recevrez un repas le matin. Ce repas vous sera donné par ingestion normale ou directement dans l'estomac. Ceci sera effectué à l'aide d'un tube souple placé dans l'estomac par voie nasale. Cette manoeuvre n'est pas douloureuse.

La composition du repas utilisé pour cette étude est de:

Glucides	40%
protéines	20%
lipides	40%

3. Pendant les 3 jours avant chaque mesure de thermogénèse, vous devrez manger une diète composée d'environ 50 % de glucides; 20 % de protéines et 30 % de lipides. (diète équilibrée). Des informations vous seront donneés à cet effet pour tenir compte de votre alimentation avant l'étude.

4. Vous aurez à écrire un journal alimentaire de trois jours (2 jours de la semaine, et 1 jour du fin de semaine) pour que nous puissions avoir un aperçu de votre alimentation habituelle.

5. A chaque séance, nous vous questionnerons sur votre diète de la veille (rappel de 24 heures).

6. Pendant chaque séance vous aurez 7 tests de calorimétrie indirecte de 30 minutes chacun qui nous permettront de mesurer les échanges gazeux.

7. La mesure de la thermogénèse induite par l'alimentation sera faite pendant la phase préovulatoire (Les 10 premiers jours après votre premier jour de menstruation).

8. En tant qu'individu, le bénéfice que vous retirerez sera de connaître votre dépense énergétique et par conséquent la quantité d'énergie alimentaire qui est nécessaire au maintien de votre poids. De plus vous connaîtrez votre tolérance au glucose, qui est liée au risque de diabète, votre composition corporelle et votre capacité aérobique maximale.

RISQUES RELIES A L'ÉTUDE

- Apparition d'une petite ecchymose aux sites de ponction sanguine.

Je ne dois pas participer à cette étude si:

- Je suis fumeuse.
- Ma consommation d'alcool est plus que 30 g par jour.
- Je suis enceinte ou allaitante.
- Mon cycle menstruel est irrégulier ou n'existe pas.
- Je fais des exercices du type aérobique pendant plus d'une heure par semaine (femmes sédentaires).
- Mon poids a changé de plus de 10 % pendant les derniers 6 mois.
- J'ai une histoire personnelle de diabète ou d'intolérance au glucose.

- J'ai une histoire personnelle de maladie gastro-intestinale.
- J'ai une histoire familiale de diabète, et/ou d'obésité.
- Je prends des médicaments qui pourront influencer le métabolisme énergétique (hormones, α-ou ß-bloqueurs, médicaments du système nerveux central).

ANNEX II

HOTEL-DIEU DE MONTRÉAL

FORMULE DE CONSENTEMENT

TITRE DE L'ÉTUDE: Une étude comparative de la thermogénèse induite par l'alimentation chez des femmes athlétiques et chez des femmes sédentaires.

INVESTIGATEURS: Dr. Dominique Garrel, M.D. et Marielle Ledoux, Ph.D.

1. J'ai pris connaissance du feuillet d'informations destiné au sujet.

2. Je reconnais avoir été bien informée et avoir eu suffisamment de temps pour considérer ces informations et pour demander conseil.

3. Je reconnais que le langage scientifique et technique utilisé a été expliqué à ma satisfaction, et que j'ai reçu des réponses satisfaisantes à mes questions.

4. Toutes les informations recueillies seront traitées de façon confidentielle et les résultats ne seront utilisés qu'à des fins scientifiques.

5. Je consens à la publication des résultats de cette étude en autant que les informations demeurent anonymes et/ou déguisées et qu'aucune identification ne puisse être faite.

6. J'ai été informée que ma participation à l'étude est volontaire et que je suis libre de refuser d'y participer ou de me retirer de l'étude en tout temps.

7. J'ai également été informée que les coordonnateurs de la recherche et le comité d'éthique de la recherche de l'hôpital ont approuvé le protocole de l'étude.

8. J'ai lu la présente formule et je consens volontairement à participer à cette étude. Je m'engage également à respecter la procédure de façon précise et à aviser les investigateurs de la moindre dérogation.

ANNEX III

Une étude comparative de la thermogénèse induite par l'alimentation chez des femmes athlètes et chez des femmes sédentaires.

RECOMMANDATIONS

PENDANT LE PROTOCOLE VOUS:

- ne devrez pas modifier vos habitudes de vie.
- ne devrez pas modifier vos habitudes alimentaires.
- devrez noter tout événement ' indésirable '
  (peu importe la cause). ex.
  → maux de tête
  → nausées, vomissement
  → diarrhée
  → infection
  → rhume etc....
- devrez noter les moyens utilisés pour contraindre ces malaises ex.
  → aspirines etc...

LA VEILLE DES TEST VOUS:

- ne devrez pas prendre d'alcool.
- devrez éviter les activités physiques intensives.
- devrez remplire le journal alimentaire.
- ne devrez pas modifier vos habitudes alimentaires.
- être à jeun à partir de 20:00 H.
- pouvez boire de l'eau jusqu'à minuit.

Vous devez vous assurer de manger suffisamment:
i.e. au courant de la jourée vous devriez manger au minimum l'equivalent de
→ 2 produits laitiers
→ 7 féculents
→ 5 fruits et légumes (sous forme de jus ou nature)

LE JOUR DU TEST VOUS:

- ne devrez faire beaucoup d'effort pour arriver au laboratoire (marcher beaucoup, arriver en velo, courrir, etc.)
- devrez rien manger et rien boire (pas du café, du thé, de l'eau).

Si vous avez des questions, n'hésitez pas à communiquer avec moi.
Pilar Lopez res. 636-8419 bur. 843-2611 # 4079 ou 4779.

Merci beaucoup de votre collaboration.

Aliments riches en protéines

1 choix (mesurez après la cuisson)

urre d'arachides	15 mL (1 c. à tab.)
avettes	10 moyennes
mage, ex. cheddar, brick	1 morceau (5 cm x 2 cm x 2 cm) (2" x ¾" x ¾")
mage cottage	50 mL (¼ tasse)
mage fondu, en tranches	1 tranche
uf	1 moyen
toncles, palourdes, huîtres	3 moyens
isson en conserve, égoutté	50 mL (¼ tasse)
isson, filet de	1 morceau (6 cm x 2 cm x 2 cm) (2½" x ¾" x ¾")
rdines, égouttées	3 petites
ucisse fumée	½
inde et volaille: bacon de dos, boeuf, poulet, jambon, agneau, rc, dinde, veau.	
côtelette, avec os	1 petite ou ½ moyenne
coupé en cubes	1 cube (25 mm carré) (1" carré)
naché ou émincé	30 mL (2 c. à tab.)
rlande pressee	1 tranche
ranché	1 morceau (10 cm x 5 cm x 5 mm) (4" x 2" x ¼")
oifteck	1 morceau (5 cm x 5 cm x 1 cm) (2" x 2" x ½")

Féculents

1 choix (mesurez après la cuisson)

iscuits secs	2
é d'Inde en épi	½ moyen
é filamenté	1 biscuit
éréale (cuite ou sèche)	125 mL (½ tasse)
squelins	8 petits
res et pois secs	125 mL (½ tasse)
acaroni	125 mL (½ tasse)
iis à grains entiers	125 mL (½ tasse)
ffin (moufflet) nature	1 petit
uilles	125 mL (½ tasse)
in	1 tranche
in à hot dog ou à hamburger	½
tit pain	1 petit
mme de terre	½
mme de terre en purée	125 mL (½ tasse)
ou autres grains	125 mL (½ tasse)
oupe	250 mL (1 tasse)
ghetti	125 mL (½ tasse)
st melba	4 tranches

ous pouvez obtenir des mesures plus précises des quantités e céréales, de biscuits et de soupes, en vous procurant le vret intitulé "Food Choices in the Marketplace" publié par Association canadienne du diabète (livret disponible en nglais seulement)

Lait

1 choix

	125 mL (½ tasse)
en poudre	30 mL (2 c. à tab.)
en conserve, évaporé	50 mL (¼ tasse)
mage	1 petite pointe + 3 craquelins
ourt nature	125 mL (½ tasse)

Vous pouvez obtenir plus d'informations en vous procurant le livret "Vive la santé! Vive la bonne alimentation!" publié par l'Association canadienne du diabète.

Fruits & légumes

1 choix (frais, en conserve dans l'eau, en conserve ou congelé non additionné de sucre. Voir **Planification de vos menus** au verso, pour fruits mis en conserve dans du jus de fruits.)

abricots	2
ananas	125 mL (½ tasse)
baies	125 mL (½ tasse)
banane	½ petite
cantaloup	¼
cerises	125 mL (½ tasse)
dattes	2
fraises	250 mL (1 tasse)
macédoine de fruits coupés	125 mL (½ tasse)
melon d'eau	250 mL (1 tasse)
orange	1 petite
pamplemousse	½
pêche	1
poire	½
pomme	½ moyenne
pommes, compote de	125 mL (½ tasse)
pruneaux	2
prunes	2
raisins	125 mL (½ tasse)
raisins secs	30 mL (2 c. à tab.)
tangerine	1
betteraves	125 mL (½ tasse)
carottes	125 mL (½ tasse)
courge	125 mL (½ tasse)
macédoine de légumes	125 mL (½ tasse)
pois verts	125 mL (½ tasse)
rutabags ou navet	125 mL (½ tasse)
tomates en conserve	250 mL (1 tasse)

Jus non sucrés:

pamplemousse, orange	125 mL	(½ tasse)
pomme, carotte, ananas	75 mL	(1/3 tasse)
raisin, pruneaux	50 mL	(¼ tasse)
tomate, légumes	250 mL	(1 tasse)

Matières grasses

1 choix

bacon	1 tranche
beurre	5 mL (1 c. à t.)
creme 10%	30 mL (2 c. à tab.)
huile	5 mL (1 c. à t.)
margarine	5 mL (1 c. à t.)
mayonnaise	5 mL (1 c. à t.)
Saindoux	5 mL (1 c. à t.)
vinaigrette commerciale, genre française ou italienne	10 mL (2 c. à t.)
noix et graines, décortiquées:	
amandes	8
arachides	10
avelines ou noisettes	5
graines de tournesol	15 mL (1 c. à tab.)
moitiés de noix de Grenoble	4
moitiés de pacanes	5
noix d'acajou	5

Autres légumes

asperges	concombre	germes de haricots mungo (fèves germées)	oignons
brocoli	courge à la moelle	haricots jaunes ou verts	persil
céleri	courgette	laitue	poivron rouge ou vert
champignons	epinards		radis
chou	feuilles de betteraves, de navet, etc.		rhubarbe
chou-fleur			tomate
choux de Bruxelles			

N.B. Les aliments suivants doivent être calculés dans votre régime alimentaire lorsqu'ils sont consommés en grande quantité:

2 tomates moyennes	= 1 choix du groupe fruits & légumes
250 mL (1 tasse) oignon cuit	= 1 choix du groupe fruits & légumes
250 mL (1 tasse) rhubarbe (cuite sans sucre)	= 1 choix du groupe fruits & légumes

ANNEX IV

ACTIVITÉ PHYSIQUE

1. RÉPARTITION DES ACTIVITÉS QUOTIDIENNES

S.V.P., donner une approximation du temps passé pour chacune des activités que vous pratiquez (en heures ou en minutes). Le total de vos activités doit être égal à 24 heures ou 1440 miutes.

Niveau d'activités		Heures	Minutes
A.	repos: (sommeil)	_____	_____
B.	très léger: (assis, debout, conduire une automobile, regarder la télévision, manger)	_____	_____
C.	léger: [marcher, bicyclette (12 Km/hre]	_____	_____
D.	modéré: [volleyball, gymnastique, golf (marcher avec sac), patinage (18-25 km/hre), bicyclette (15 km/hre), natation (2 km/hre), ski de fond (4 km/hre)]	_____	_____
E.	élevé: [squash, balle au mur, kayak, plongée sous-marine, basketball, marche (7-8 km/hre), course (12 km/hre), patinage (28 km/hre), byciclette (20-25 km/hre), natation (2.5-3.5 km/hre), ski de fond (6-8 km/hre)]	_____	_____
F.	très élevé: [kayak (15 km/hre), patinage artistique, sauts à la corde, course (10 km/hre, pente > 4.5 degrés), patinage (30-35 km/hre), bicyclette (25-35 km/hre), natation (3.5-4.5 km/hre), ski de fond (8-13 km/hre)].	_____	_____
G.	maximum: course (15 km/hre)	_____	_____
	TOTAL	_____	_____

2. ENTRAINEMENT

Indiquer le type, la fréquence et la durée de l'entraînement fait régulièrement.

TYPE	FRÉQUENCE	DURÉE	INTENSITÉ	REMARQUES

3. SPORTS DE COMPÉTITION

Nommer les activités sportives pratiquées en compétition et depuis quand?

1. _____ _____
2. _____ _____
3. _____ _____
4. _____ _____

A quel niveau compétitionnez-vous?

1. municipal 2. régional 3. provincial
4. national 5. international

Types d'activités et dernières performances enregistrées

1. _____ _____
2. _____ _____
3. _____ _____

ANNEX V

Questionnaire sur l'aptitude à l'activité physique - Q-AAP (version révisée en 1994)

Q - AAP et VOUS

(Un questionnaire pour les gens de 15 à 69 ans)

L'exercice physique pratiqué d'une façon régulière constitue une occupation de loisir saine et agréable. D'ailleurs, de plus en plus de gens pratiquent une activité physique de façon régulière. Règle générale, augmenter la pratique sportive n'entraîne pas de risques de santé majeurs. Dans certains cas, il est cependant conseillé de passer un examen médical avant d'entreprendre un programme régulier d'activités physiques. Le Q-AAP (questionnaire sur l'aptitude à l'activité physique) vise à mieux cerner les personnes pour qui un examen médical est recommandé.

Si vous prévoyez modifier vos habitudes de vie pour devenir un peu plus actif(ve), commencez par répondre aux 7 questions qui suivent. Si vous êtes âgé(e) de 15 à 69 ans, le Q-AAP vous indiquera si vous devez ou non consulter un médecin avant d'entreprendre votre nouveau programme d'activités. Si vous avez plus de 69 ans et ne participez pas d'une façon régulière à des activités physiques exigeantes, vous devriez consulter votre médecin avant d'entreprendre ces activités.

Lisez attentivement et répondez honnêtement à chacune des questions suivantes. Le simple bon sens sera votre meilleur guide pour répondre correctement à ces questions. Cochez OUI ou NON.

OUI	NON		
☐	☐	1.	Votre médecin vous a-t-il déjà dit que vous souffriez d'un problème cardiaque et que vous ne deviez participer qu'aux activités physiques prescrites et approuvées par un médecin?
☐	☐	2.	Ressentez-vous une douleur à la poitrine lorsque vous faites de l'activité physique?
☐	☐	3.	Au cours du dernier mois, avez-vous ressenti des douleurs à la poitrine lors de périodes autres que celles où vous participiez à une activité physique?
☐	☐	4.	Éprouvez-vous des problèmes d'équilibre reliés à un étourdissement ou vous arrive-t-il de perdre connaissance?
☐	☐	5.	Avez-vous des problèmes osseux ou articulaires qui pourraient s'aggraver par une modification de votre niveau de participation à une activité physique?
☐	☐	6.	Des médicaments vous sont-ils actuellement prescrits pour contrôler votre tension artérielle ou un problème cardiaque (par exemple, des diurétiques)?
☐	☐	7.	Connaissez-vous une autre raison pour laquelle vous ne devriez pas faire de l'activité physique?

Si vous avez répondu

"OUI" à une ou plusieurs questions

Consultez votre médecin AVANT d'augmenter votre niveau de participation à une activité physique et AVANT de faire évaluer votre condition physique. Dites à votre médecin que vous avez complété le questionnaire sur l'aptitude à l'activité physique et expliquez-lui précisément à quelles questions vous avez répondu "oui".

- Il se peut que vous n'ayez aucune contre-indication à l'activité physique dans la mesure où vous y allez lentement et progressivement. Par ailleurs, il est possible que vous ne puissiez faire que certains types d'efforts adaptés à votre état de santé. Indiquez à votre médecin le type d'activité physique que vous comptiez faire et suivez ses recommandations.
- Informez-vous quant aux programmes d'activités spécialisés les mieux adaptés à vos besoins, offerts dans votre localité.

"NON" à toutes ces questions

Si, en toute honnêteté, vous avez répondu "NON" à toutes les questions du Q-AAP, vous êtes dans une certaine mesure, assuré(e) que:

- vous pouvez augmenter votre pratique régulière d'activités physiques en commençant lentement et en augmentant progressivement l'intensité des activités pratiquées. C'est le moyen le plus simple et le plus sécuritaire d'y arriver.
- vous pouvez faire évaluer votre condition physique. C'est le meilleur moyen de connaître votre niveau de condition physique de base afin de mieux planifier votre participation à un programme d'activités physiques.

REMETTRE À PLUS TARD L'AUGMENTATION DE VOTRE PARTICIPATION ACTIVE:

- si vous souffrez présentement de fièvre, d'une grippe ou d'une autre affection passagère, attendez d'être remis(e); ou
- si vous êtes enceinte ou croyez l'être, consultez votre médecin avant de modifier votre niveau de pratique sportive régulière.

Veuillez noter que si votre état de santé se trouve modifié de sorte que vous deviez répondre OUI à l'une ou l'autre des questions précédentes, consultez un professionnel de la santé ou de la condition physique, afin de déterminer s'il vous faut modifier votre programme d'activités.

Formule de consentement du Q-AAP: La Société canadienne de physiologie de l'exercice, Santé Canada et ses représentants n'assument aucune responsabilité vis-à-vis des accidents qui pourraient survenir lors de l'activité physique. Si, après avoir complété le questionnaire ci-dessus, un doute persiste quant à votre aptitude à faire une activité physique, consultez votre médecin avant de vous y engager.

Nous vous encourageons à copier le Q-AAP dans sa totalité

Dans la mesure où le Q-AAP est administré avant que la personne ne s'engage dans un programme d'activités ou qu'elle fasse évaluer sa condition physique, la section suivante constitue un document ayant une valeur légale et administrative.

Je sous-signé(e) affirme avoir lu, compris et complété le questionnaire et avoir reçu une réponse satisfaisante à chacune de mes questions.

NOM _____

SIGNATURE _____ DATE _____

SIGNATURE D'UN PARENT _____ TÉMOIN _____
ou TUTEUR (pour les mineurs)

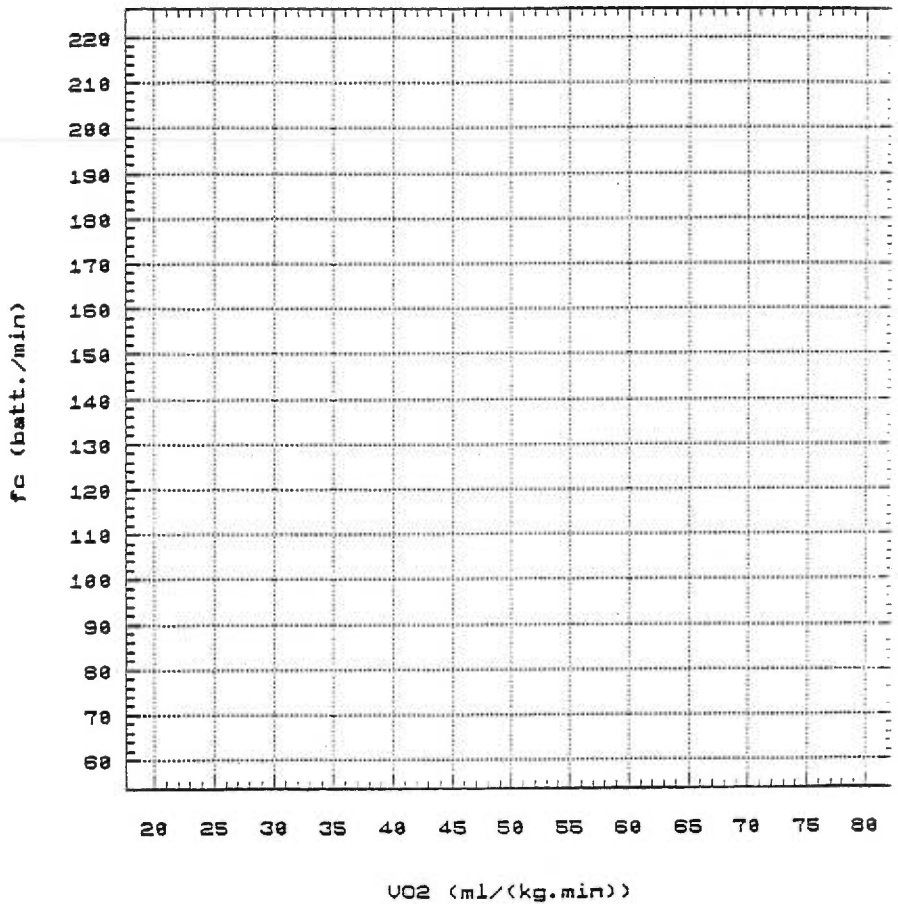
Avec l'appui de: Santé Canada Health Canada

suite au verso...

PROTOCOLE SUR TAPIS ROULANT

VITESSE INITIALE 8 KM H^{-1} (5 MIN)
ACCELERATION DE 1 KM H^{-1} PAR PALIER DE 2 MIN

VITESSE KM H^{-1}	TEMPS MIN	VO2 ML KG MIN	FC BATT.MIN^{-1}
8	5	___	___
9	7	___	___
10	9	___	___
11	11	___	___
12	13	___	___
13	15	___	___
14	17	___	___
15	19	___	___
16	21	___	___
17	23	___	___
18	25	___	___
19	27	___	___
20	29	___	___
21	31	___	___
22	33	___	___
23	35	___	___



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