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The effect of prior exercise on the thermic effect of carbohydrate versus fat feeding in man

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THE EFFECT OF PRIOR EXERCISE ON THE THERMIC EFFECT OF CARBOHYDRATE VERSUS FAT FEEDING IN MAN

by

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ABSTRACT

Prior exercise potentiates the thermic effect of a carbohydrate meal. The purpose of this study was to determine if the potentiating effect of exercise is unique to carbohydrate meals or whether prior exercise also potentiates the thermic effect of a fat meal. Subjects were 15 healthy females (24 years, 60 KG, 20% body fat). Each subject completed an exercise and a control trial for each meal, each trial on a separate day. Meal size was 2510 KJ (600 kcal) and consisted of 100% carbohydrate, compared with 90% fat combined with 10% carbohydrate. Subjects exercised for 45 minutes at 70% of VO_2 max on a cycle ergometer. Once VO_2 had returned to baseline following exercise, subjects ingested a test meal. As a control, subjects ingested a test meal without prior exercise. The thermic effect of food (TEF) was determined by indirect calorimetry over a 2 hour period following meal ingestion. The thermic effect of the carbohydrate meal was 50% greater than that of the fat meal ($p<0.05$). Thermic effect of food was increased by 59% over control when the carbohydrate meal was ingested after exercise (E = 124+/- 19 vs C = 78 +/- 17 kJ/2hr, $P<0.01$). However, TEF was not increased significantly over control with the fat meal after exercise (E = 75 +/- vs C = 61 +/- 10 kJ/2hr). These results suggest that the potentiation of the thermic effect of a meal by prior exercise depends on the composition of the meal, essentially excluding meals consisting primarily of fat.

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ACKNOWLEDGMENTS

"Most teachers are knowledgeable,
Good teachers are intelligent,
Great teachers are patient,
Exceptional teachers are student themselves"
Dale Dubin, M.D.

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CHAPTER 1

INTRODUCTION

There are three major components of daily energy expenditure: resting metabolic rate, (RMR); thermic effect of feeding, (TEF); and, thermic effect of activity, (TEA) (Figure 1). Resting metabolic rate, which comprises approximately 60-75% of the body's total daily energy expenditure, has been shown to be influenced by age, gender, body composition, weight, climate, circadian rhythms, hormonal status, pregnancy, genetic predisposition and exercise. The thermic effect of feeding contributes approximately 10% of the total daily energy expenditure. The thermic effect of food combined with the resting metabolic rate together constitute over 70-85% of the energy expenditure of humans (Figure 2).

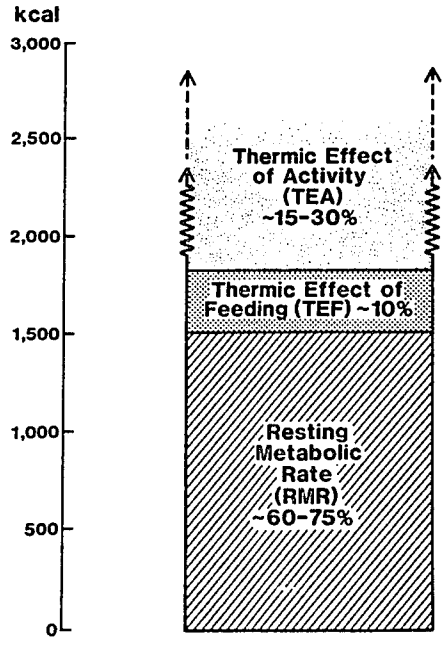


Figure 1 -The three major components of daily energy expenditure: RMR, resting metabolic rate; TEF, thermic effect of feeding; TEA, thermic effect of activity From Poehlman, E.T.: A Review: Exercise and it's influence on resting energy metabolism in man. Med. Sci. Sports Exerc., 21:515-525, 1989).

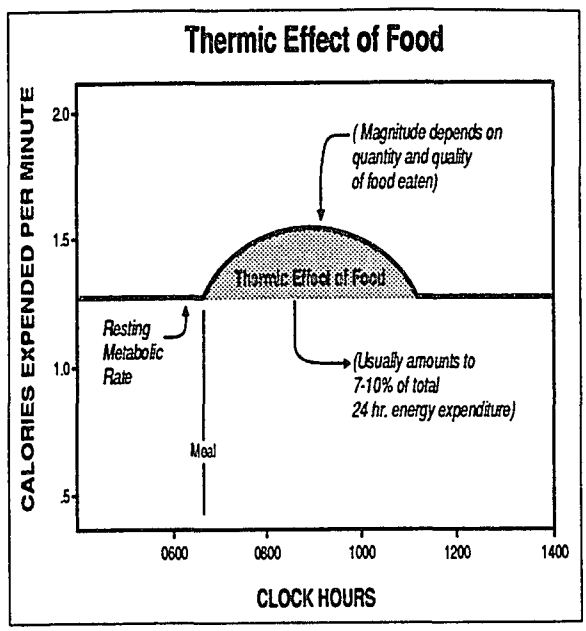


Figure 2 The thermic effect of food (TEF) is the energy expended for the digestion, absorption, transport, metabolism, and storage of food. From Horton, E.S. Introduction: An overview of the assessment and regulation of energy balance in humans. (Am J. Clin. Nutr. 38:972-977, 1983).

The thermic effect of activity constitutes approximately 15-30% of the total daily energy expenditure. This is the most variable component of the total daily energy expenditure and reflects the additional energy expended above resting metabolism and the thermic effect of feeding due to physical activity. Additionally, any variations in resting metabolic rate, combined with the thermic effect of a meal that could be augmented by purposeful physical activity could prove to be significant in the relationship to the overall energy balance in humans, inclusive of weight loss as well as sustaining optimal weight maintenance.

The interrelated effects of physical fitness, resting metabolic rate, and the thermogenic relationship of macronutrients, have not been clearly established. Recent research has yielded inconsistent findings (Tremblay, Coye, & Leblanc, 1983; Hill, 1984; Gilbert, Misner, Boileau, Ji, & Slaughter, 1991). It has been reported that aerobic exercise that precedes meal consumption may further alter the thermic effect of the meal (Young, Treadway, & Balon, 1986; Balon & Welk, 1991). Aerobic exercise following meal consumption may also enhance the thermic effect of a meal. (Segal & Gutin 1983; Welle,1984). However, in those studies failing to show a potentiation of the thermic effect of a meal by physical activity, the exercise tended to be of short duration and /or low intensity (Dalloso and James, 1984, Jones et.al 1963, Pacy et.al, 1985 Swindells 1972).

Need for the study

A better understanding of resting metabolic rate in humans respective to the manipulation of macronutrients, as well as the effect of physical activity is needed and could benefit athletes in pursuit of the optimal body composition for maximal human performance and competitive edge, as well as individuals trying to achieve a healthy body weight in compliance with the 1988 Surgeon General's report. Presently, it remains unclear whether exercise can increase an individual's metabolic activity during periods of inactivity or non-exercise times, or effect the contribution of the thermic effect of food by specific macronutrients.

A greater understanding of specific relationships among macronutrients, the thermic effect of feeding, and resting metabolic rate, coupled with exercise intensity and duration, could contribute toward the advancement of the knowledge of energy metabolism. Further knowledge could facilitate the development of more precise nutritional and exercise recommendations in both the exercising and non-exercising populations.

Purpose of the study

The purpose of this study was to compare the thermic effect of a 2510 KJ (600 kcal) meal composed of 100% carbohydrate with an equivalent caloric meal consisting of 90% fat and 10% carbohydrate after a prior bout of exercise, to determine whether the increase in dietary - induced thermogenesis after exercise was dependent on the type of food ingested.

Limitations of the study

1. Subjects were instructed to maintain their normal living patterns 36 hours prior to testing with the exclusion of exercise and to maintain their normal dietary intake.
2. Subjects were asked to refrain from eating 12-15 hours prior to reporting to the lab for testing.
3. It is assumed that all subjects performed maximally to attain max VO2 values for the establishment of a 70% workload.
4. Subjects were asked to report to the laboratory soon after rising in the morning and to be as conservative as possible with their energy expenditure on the way to the laboratory.
5. The subjects tested in this study were atypical of the average American demographics. They were comprised of young, lean, physically fit women.

Definitions and Explanation of Terms

Basal Metabolic Rate (BMR) Through the years, basal metabolic rate has sometimes been used synonymously with resting metabolic rate, this is technically incorrect. To clarify this issue it is customary to measure basal metabolic rate under standard conditions. The subject is usually housed overnight in a metabolic sleep lab and metabolic measurements are taken just after the subject has awakened and prior to arising from bed. Basal metabolism is usually slightly lower than resting metabolic rate and is remarkable constant within individuals from day to day. However, due to the limitation of constraints required to achieve a true basal metabolic rate, resting metabolic rate is frequently used for more practical purposes. Both terms are reflective of the sum total of all the involuntary activities that are necessary to support life, including respiration, circulation, and new tissue synthesis, excluding digestion, and is usually expressed in calories per day. Basal metabolic rate excludes minimal voluntary activities, and therefore accounts for a slightly lower energy expenditure than that represented by resting metabolic measurements.

Resting Metabolic Rate (RMR) This represents the metabolic cost of energy expended by the body to maintain life and normal integrated body functions, such as respiration and circulation, in an environment of thermal neutrality. It is inclusive of minimal voluntary activity, and is therefore, slightly higher than the caloric cost of basal metabolic rate. It comprises the largest component of daily energy expenditure Resting metabolic rate accounts for 60-75% of the of the daily total energy expenditure in sedentary populations.

Thermic Effect of Feeding (TEF) The thermic effect of feeding is also synonymously referred to as the thermic effect of food (TEF), or diet induced thermogenesis (DIT), and in earlier metabolic literature is referred to as the specific dynamic action of food (SDA). The (TEF) accounts for approximately 10% of the daily energy expenditure, and reflects the energy cost associated with the ingestion of food. It refers to the cumulative energy expenditure incurred with the ingestion, digestion, absorption and storage of food. It is energy expended above resting metabolic rate. Variations in (TEF) are dependent on the amount and the composition of the diet. High carbohydrate diets elevate the (TEF) more than high fat diets.

Thermic Effect of a Meal (TEM) Most researchers utilize the thermic response to a single meal test TEM, due to the limitations associated with the assessment of the total energy cost of total food intake. TEM is representative of the energy cost of food absorption, metabolism, and storage. Additionally, TEM is further delineated into two subcategories: obligatory and facultative. The obligatory thermogenesis of TEM defines the factor for energy cost associated with absorption and transport of nutrients and the synthesis of protein, fat and carbohydrate. In contrast, facultative thermogenesis represents energy expended that is in excess of obligatory thermogenesis. This is a phenomena that has been observed in brown adipose tissue in animals, but still remains to be confirmed in humans.

Thermic Effect of Activity (TEA) Thermic effect of activity is the additional energy

expended above RMR and TEF. It is representative of all physical activity, shivering, fidgeting and purposeful physical exercise. It is the most variable component of energy expenditure in humans and has been found to constitute 15-30% of total daily energy expenditure.

CHAPTER 2

REVIEW OF THE LITERATURE

Food must be consumed for survival, work, and health in quantities that will satisfy the needs of the body for energy and essential nutrients. Archeological data have recorded early human remains back nearly 3 million years. However, nutrition consisted merely of the instinctive biological drive to acquire nourishment, which is in total contrast to how nutrition and exercise are viewed as we approach a new millennia. Many of the achievements in nutrition and exercise physiology have arisen from vigorous application of scientific methods with an interdisciplinary approach. However, throughout most of human development, pragmatic and mythical thinking have been the predominant way of acquiring knowledge.

Origins of Nutrition and Exercise Physiology

In historical times, 2500 years ago, Greek philosophers concluded that direct observation and logical reasoning were the only reliable ways to learn about the natural world. Historians are in agreement that the concept of a close connection between diet,

exercise, and medicine dates back to three ancient physicians: Herodicus (400 B.C.), Hippocrates (460-370 B.C.), and Claudius Galenus or Galen (129-210 A.D.) . Hippocrates has been universally honored as "the father of scientific medicine". Many of his writings emphasized the importance of eating and exercise. Hippocratic physicians expressed impatience with the idea that disease was punishment sent by the gods. Diet and exercise or "gymnastic medicine" were an important part of the Hippocratic physician's diagnosis, and emphasis was placed upon how a patient lived, what he ate and drank, and his daily activities. Food was perceived to be a single source of nutriment or an ailment, and inhibited a true understanding of nutrition. Therefore, subjective observations provided the basis for treatment due to the lack of scientific methods (Todhunter, 1976).

Galen, about 600 years later, collected and transmitted the most extensive record of medicine and nutrition of that period. Galen considered exercise as a branch of hygiene, and hygiene to be a part of the science of medicine. Galen developed a crude concept of metabolism which dominated medicine and ideas about food into the 17th century. He concluded from his anatomical observations that food, after absorption, underwent sequential stages of transformation, with tissues and organs changing it so it was suitable for incorporation into their own substance. In addition, surpluses and wastes were subsequently excreted, and that during starvation material from other tissues flowed back to nourish the liver. Despite Galen's insightful observations, the prevailing thinking

of Hippocratic physicians was dominated by mysticism which inhibited the understanding of physiological processes and the relationships between foods and body function (Hutchins, 1952).

Nearly 2000 years of scholarly quiescence passed during the religious and political reign of the Roman and Christian empires before the intellectual ferment of the Renaissance in the 16th century spawned the resurgence of the scientific attitude that had risen briefly in ancient Greece. In 1543, Versalius began making direct observations on the anatomy of the human body. Paracelsus struggled in the 16th century for the independence of thought and began grasping for concepts of nutrition and metabolism, but did not have the knowledge of biology or science needed to formulate them clearly.

By the 17th century Galileo, Descartes, and Francis Bacon began to reject the dependence on mystical thinking as a way of explaining natural phenomena and concluded that the only way to obtain information about nature was by investigating it objectively, utilizing the senses, experience, and reason which gave rise to the scientific attitude (Todhunter, 1976).

Nutritional concepts arose from the establishment of both chemistry and physiology during late 1700's and early 1800's. 1770 brought the discovery of oxygen by Joseph Priestley and his confirmation that oxygen was consumed by animals. Antione Lavoisier first measured heat production by animals and provided evidence that animal catabolism was similar to combustion. He believed, though, that all animal heat was

produced in the lungs. Lavoisier related oxygen consumption to heat production by measuring the heat produced by a guinea pig in an ice-jacketed calorimeter. He later documented experiments relating gas exchange to heat production, and progressed to measuring human oxygen consumption in humans using a metal facepiece apparatus. In retrospect, his system has been referred to as a closed circuit, indirect calorimeter (Kinney, 1983).

Friedrich Wohler, a German chemist was the first to synthesize urea from inorganic substances in 1828, which showed that compounds produced by living things were within the realm of ordinary chemistry. 1837 brought the discovery by Heinrich Gustav Magnus that blood took oxygen from the lungs to tissue and then returned with carbon dioxide. Up to that point in history, heat was thought to be generated by both the blood and the lungs. By 1842, Justice von Liebig had classified foods as carbohydrates, lipids, and proteins, and had measured their caloric values. The pioneering investigators of calorimetry focused on developing the technology and establishing the agreement between direct and indirect calorimetric measurements. During the middle to late nineteenth century, closed and open circuit indirect calorimeters were used to study the effects of various diseases on human metabolism (Kinney, 1983).

Nutrition became and independent science somewhere between 1840 and 1860 when investigations with specific nutritional objectives became more common. Julius Robert von Mayer and James Joule developed the concept that heat, motion, and

electricity were all forms of one thing, energy.

Mayer clearly conceptualized the nature of animal energy transformations. Pettenkofer and Voit advanced the understanding of metabolism in the 1860's. They constructed a respiration apparatus and made caloric measurements on human subjects, as well has observations on a diabetic patient. Hermann von Helmholts and Edward Pfluger showed that animal tissues such as muscle produced heat and consumed oxygen. This finding confirmed that catabolism occurred in all tissues, not just blood and the lungs. In 1892, Haldane first used an open-circuit, indirect calorimeter in which an animal breathed room air, and the expired air analyzed for carbon dioxide content. Rudolf von Lolliker is credited with the discovery of mitochondria. Zuntz and Geppert developed an open-circuit indirect calorimeter for human studies and noted the importance of absolute skeletal muscle rest during measurements of resting energy expenditure. Magnus and Levy used the Zuntz and Geppert apparatus to study normal humans and observed the effects of food, age, gender and pregnancy on metabolic rate. In 1895, Magnus and Levy demonstrated increased metabolic rate with Graves' disease (Todhunter, 1976).

The latter half of the 19th century yielded studies by Claude Bernard on carbohydrate metabolism and by Pasteur and Buchner on fermentation and anaerobic bacteria, which gave rise to physiological chemistry. Bernard's development of the concept of homeostasis was a stimulus for studies of regulation of metabolism in the biological sciences. During this time, the concepts of hormones and enzymes were also evolving.

The term "vitamin" was coined in 1912 by Asimir Funk. In 1922, Fredrick Banting, J. Macleod and C.H. Best isolated insulin and showed it was able to reduce blood sugar levels. An English biologist David Keilin initiated the first study of electron transport.

By the turn century, a German named Max Rubner and his associates established that basal heat production of different species varied with their body surface area. Rubner was also credited with the discovery of the energy metabolism that was stimulated by the ingestion of food which was termed the specific dynamic action of foods, and which is now termed the thermogenic effect of foods.

The early 1900s saw the establishment of several major centers for the study of human metabolism. Notable laboratories included the Nutrition Laboratory of the Carnegie Institute at Boston, Massachusetts, which was under the direction of F.G. Benedict. Benedict performed extensive studies with humans using a variety of techniques and developed the first instrument suitable for clinical use. Grahm Lusk studied the effects of foods and intermediary metabolism at Bellevue Hospital in New York, New York. D.B. Dill presided over the Harvard Fatigue Laboratory and W.M. Boothby published a detailed description of the measurement technique and standards for normal resting energy expenditure based upon large population samples at the laboratory at Mayo Clinic in Rochester, Minnesota (Kinney, 1983).

1933 brought the discovery of German physiologists Gustav Embden and Otto Meyerhof who made fundamental discoveries concerning the sequence of chemical

reactions in glycolysis. Hans Krebs presented the first model of the citric acid cycle in 1937. Fritz Lipman postulated in 1941 the central role of ATP as an energy carrier. American biochemist Albert Lehninger and E.P. Kennedy established the function of mitochondria. Subdivisions of chemistry and biology were being recognized by the end of the 19th century (Berryman & Park, 1993).

Comprehensive research projects conducted at these sites produced a wealth of information that described the techniques of direct and indirect calorimetry. Investigations on the influence of disease on metabolism and the establishment of the standards for obtaining resting energy expenditure in large populations of normal subjects continue to be referenced as the "gold standard" by current investigators.

The last 100 years since 1890 have been a period of rapid development of the science of nutrition and exercise physiology that is unprecedented. The multidisciplinary integration of related areas such as exercise physiology, nutrition, physics, chemistry, biochemistry, physiology and computer science has allowed for the fruition of physiological science. The origins of the scientific disciplines of nutrition and exercise physiology have tightly parallelled the chronological record of time, yet both disciplines have achieved scholastic independence. However, when combined, they contribute valuable information about relationships between diet and exercise to provide solutions for important medical problems as well as practical applications for improved overall health for humans.

Measurements of Energy and Metabolic Rate

The metabolic rate of any organism reflects the rate at which it consumes energy and converts chemical energy into heat and physiological work. Heat is made in every pathway of energy utilization in the body. The metabolic rate reflects the intensity at which the body carries out maintenance functions, growth, and exercise and indicates the overall rate of functioning. The metabolic rate is depictive of the body's need for nutritive energy.

The traditional measure of energy in biology is the calorie, defined as the amount of heat required to raise the temperature of a gram of water by 1 degree Celsius under standard temperature and pressure conditions. Chemical and mechanical energy can be converted to heat and may be expressed in units of heat called a Calorie or kilocalories (kcals). The energy in carbohydrate, fat and protein can be measured in calories. Specifically, a kilocalorie or kcal is the energy unit familiar to the general population, however, technically it is a unit of 1000 calories. An internationally recognized unit of measurement is a Joule, (1 joule = 4.184 cal). The metabolic rate is expressed in units such as calories per hour or kilocalories per day.

The energy value of food, also called the caloric value of food, is determined by

burning a sample of food in oxygen in a device termed a bomb calorimeter (Figure 3) which measures all the heat liberated by the combustion of a food source. Representative mixtures of fat, protein and carbohydrate have been estimated for human metabolism: 4.1 kilocalories per gram of carbohydrates, 9.3 for lipids, and 4.5 for proteins.

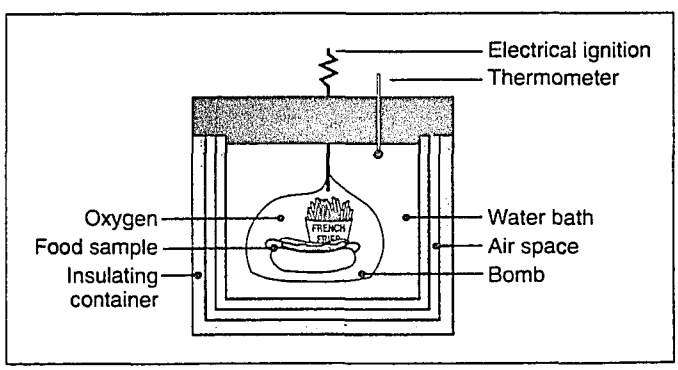


Figure 3 Bomb Calorimeter used to directly measure the energy value of food. From Essentials of Exercise Physiology, McCardle, Katch and Katch, page 181, 1994.

The two fold greater amount of energy per unit of weight for lipids is undoubtedly one of the reasons that animals have developed mechanisms to convert excess nutrients to lipids for storage in the body.

Methods of Measuring the Body's Heat Production

Energy expenditure in humans can be measured in a variety of ways (McLean, and Tobin, 1988). Direct calorimetry is the most accurate method for measuring heat production, however, this technique is limited. In the classic procedure of direct calorimetry, a calorimeter is used to measure heat production. The heat produced is measured as temperature changes through thermal gradients or as heat added to the ambient environment in an insulated chamber. In comparison, indirect calorimetry can be used to determine energy expenditure by measuring oxygen consumption and carbon dioxide production and then relating these measurements to the energy value of the oxygen consumed and the carbon dioxide produced.

Direct Calorimetry. Direct calorimetric measurements are still performed in research and clinical settings. Direct calorimetry is much more cumbersome, costly, labor intensive, and time consuming than the alternative indirect determination of heat production and is less practical clinically for measuring energy expenditure on a routine basis. Figure 4 depicts an airtight, thermally insulated living chamber (McArdle, 1991).

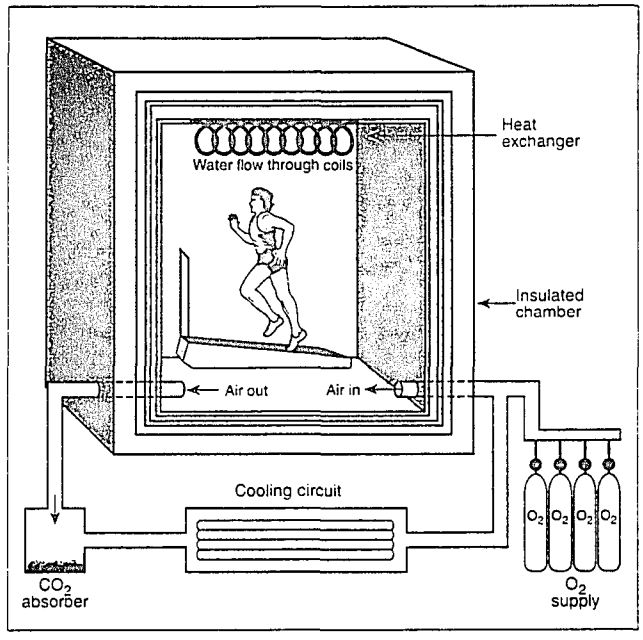


Figure 4 Direct Calorimetry. The body's heat production is measured directly in the human calorimeter. From McCardle, Katch and Katch, Essentials in Exercise Physiology, page 82, 1994.

The heat produced and radiated by the subject is removed by a stream of cold water that flows through tubes coiled near the ceiling of the chamber. The difference in the temperature of the water that entered the chamber and then leaves the chamber is reflective of the heat produced by the test subject. While direct calorimetry yields highly accurate results that are of significant importance, the physical constraints make it

impractical for routine studies of human energy expenditure.

Indirect Calorimetry. Indirect calorimetry can be used to determine energy expenditure by measuring oxygen consumption and carbon dioxide production and then relating these measurements to the energy value of the oxygen consumed and the carbon dioxide produced. Gas exchange studies can be accomplished by either closed-circuit or open-circuit methods and a strong correlation of $r = .84$ was reported between the two techniques (Fowler, Blackburn, and Helmholz, 1957). Both systems require the ability to capture all expired gas, and an accurate measurement of the volume of an expired gas. During a closed-circuit measurement, the subject breathes from a spirometer containing either oxygen or room air. Carbon dioxide is absorbed upon exhalation and the remaining expired air is directed back to the spirometer by means of a one-way valve. The decreased volume of gas in the spirometer represents the portion of oxygen that was assimilated over a precise time period.

The open-circuit technique is the most frequently employed method. The subject inhales ambient air that has a constant composition of 20.93% oxygen, 0.03% carbon dioxide, and 79.04% nitrogen. This method requires precise measurements of exhaled volume over a specific time and accurate determinations of the composition of the mixed expired gas. Analysis of the difference in composition between the exhaled air and the ambient air reflects the body's rate of energy utilization. Open-circuit spirometry provides a means to measure oxygen consumption and indirectly calculate energy

metabolism (McArdle, 1991).

Two traditional methods of open-circuit spirometry include the "Douglas bag" or "balloon method",(Figure 5) and the Kofranyi-Michaelis meter, more commonly known as the K-M meter. The K-M meter is a portable spirometer that was developed during the 1940's in Europe and was designed to provide an equitable basis for food rationing among soldiers.

(Figure 5) K-M Meter. Portable spirometer used to measure oxygen uptake by the open-circuit method. From Essential in Exercise Physiology, 1994, McCardle, Katch and Katch, page 83.

In the Douglas bag method, the subject breathes room air and exhales into a collection bag

via a two-way valve (Figure 6). After a specific period of time, the volume and composition of the gas in the bag are determined and the volume of oxygen consumed and volume of carbon dioxide produced are calculated from the concentration differences between the inspired and expired gas mixtures, with volumes corrected for standard temperature, pressure and water vapor saturation.

(Figure 6) Douglas Bag From Essentials in Exercise Physiology, McCardle, Katch and Katch, 1994, Page 84.

Ambient air is inspired and the expired air passes through a gas meter that measures the volume and also collects a small gas sample. The expired gas samples were analyzed

using either manual Haldane, or Micro Scholander gas analyzers. The Haldane procedure, developed in 1935 by Haldane and Priestly, measured the gas volume from a calibrated burette, and determined oxygen and carbon dioxide content by separating the chemical absorption of oxygen and carbon dioxide. Later, in 1947 Scholander utilized the same principles as Haldane using a calibrated micrometer as a measuring device (Consolazio, 1963). Both Haldane and Scholander analyzers remain standard instruments for the analysis of calibrating gases, irrespective of the escalation of computerized systems.

Current Clinical Techniques

Present-day indirect calorimetry employ "state of the art" methods of gas analysis, volume or flow measurement, and data management. Data acquisition and the balance between input and output flow rates are controlled by a computer. Expired gases are directed past a desiccant, pressure regulator, infrared carbon dioxide analyzer, and a paramagnetic oxygen analyzer and gas exchange is then calculated via material balance equations (Wilmore, 1977). This modern day technology allows investigators the capability of making long term measurements under controlled conditions.

Components of Energy Expenditure

Energy expenditure can be subdivided into resting metabolic rate (RMR). thermic effects of food, physical activity, and growth. RMR is the quantity of energy needed to maintain body temperature, repair internal organs, support cardiac function, maintain ionic gradients across cells, and support respiration. This constitutes approximately two-thirds of total energy expenditure. The second largest component of energy expenditure is required for physical work. The energy expenditure required to move the body is related directly to body weight, to the distance that weight is moved, and to the physical fitness status.

The heat produced following ingestion of a meal is termed the thermic effect of food (TEF) or diet-induced thermogenesis (DIT), and formally termed the specific dynamic action of food. This effect can be produced by any food, but the consumption of protein or carbohydrates results in larger thermic effects than does the consumption of fat (Forbes, 1987).

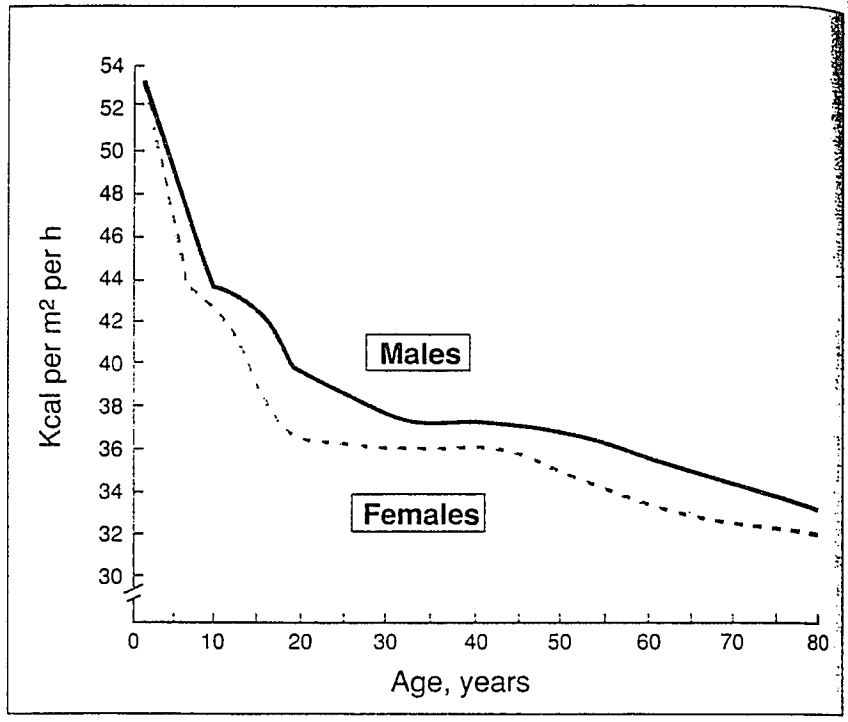
Factors Influencing Resting Energy Expenditure

Resting energy expenditure is influenced by many factors, including age, sex, body weight, dietary intake, hormonal status, pregnancy, lactation, psychogenic effects, environmental temperatures, activity level, circadian rhythms and disease states. Usually the two most potent influences on metabolic rate are environmental temperature and level

of exercise. Infancy and childhood are representative of the highest rates of energy expenditure per unit of body weight. In adulthood, the decline continues at approximately 2% per decade.

Gender Control of Energy Metabolism

Males have a higher energy expenditure per unit of body weight than females, probably due to the lower proportion of body fat (Figure 7). Resting energy metabolism has been shown to be 5 - 10% lower in females than males (Altman, and Dittmer, 1968). However, when energy expenditure is expressed on the basis of fat-free mass, the differences between males and females and younger and older adults disappear.



(Figure 7) Gender Differences. Basal metabolic rate as a function of age and gender.

Hormonal Control of Energy Metabolism

Hormones including thyroxin, catecholamines, and insulin, also influence energy expenditure. The thyroid gland secretes two hormones, thyroxin (T4) and triiodothyronine (T3). Approximately 90% of secretion is in the less biologically active form of thyroxin which has intrinsic hormonal properties of its own, but its primary function is to serve as a prohormone. The remaining 10% is the active functional form of triiodothyronine. (Guyton, 1986). The effects of thyroid hormones are multiple, and while this is a claim that can be made for many other hormones, none truly has the breadth of action of thyroid hormones. The action of thyroid hormone can be delineated into two categories. The first, which is relevant to this study is the metabolic effects including thermogenesis, regulation of water and ion transport, and the regulation of intermediary metabolism The second category is the effect of thyroid hormone on growth promoting and developmental properties which are not of relevance for the purpose of this study, but nevertheless, of great physiological significance.

Resting Metabolic Rate and Genetic Variation

A significant genetic component has been shown to influence resting metabolic rate. In a group of 130 nondiabetic adult southwestern American Indians from 54 families were investigated to further substantiate the existence of genetic susceptibility for human obesity is known to be a familial trait that is independent of individual differences in fat-

free mass, age, and sex (Bogardus, Lillioja, Ravussin, Abbott, Young, Knowler, Jacobowitz, and Mall, 1986). Earlier studies support a significant genetic component determining individual variation in RMR after accounting for the influences of age, gender and body composition (Fontaine, E., Savard, R., Tremblay, J., Despres, J, Poehlman, E. and Bouchard, C. , 1985). More recently, it has been demonstrated that 40% of the individual differences in RMR are genotype dependent (Bouchard,C., Tremblay, A., Tremblay, A. and Nadeau, 1989).

Additional Potential Factors Impacting Metabolic Rate

The efficiency of energy transfer from foods to adenosine triphosphate (ATP) and of ATP utilization may also influence energy needs. In some forms of obesity, the obese may be more efficient than lean subjects in utilizing nutrients for resting metabolic requirements and, thus, may have more energy to store as fat. A number of mechanisms have been suggested for understanding metabolic efficiency, including reduced sodium pump activity, lower protein turnover, altered nutrient partitioning, faulty thermogenic mediation by the sympathetic nervous system, impaired function of brown adipose tissue and reduced efficiency of muscular contraction (Bray, 1983) .

Thermic Effect of Meal and Metabolism

Max Rubner in 1902 identified the significant influence of the calorigenic effect of food on the metabolic rate in humans. It was Rubner who identified the term "specific dynamic action" of food when he noticed that the increase in energy expenditure was greater after the ingestion of protein than after fat or sugar. Following the ingestion of nutrients, the metabolic rate rises for a number of hours even though no other conditions are altered. The current terminology, diet induced thermogenesis (DIT) or the thermic effect of food (TEF) have replaced Rubner's original term, "specific dynamic action". The last decade has brought about renewed interest in DIT due to the implications that thermogenesis or energy expenditure might play an important role in the regulation of body fatness. Additionally, animal studies conducted in the early 1960's by Miller and Payne suggested that a defective regulation of adaptive thermogenesis in humans might also be responsible for the development or maintenance of obesity, triggering a cascade of studies addressing energy expenditure, resting metabolic rate and diet-induced thermogenesis. (Westrate, 1993)

The thermic effect of a meal can be explained largely by the energy necessary for digestion, absorption, transport, metabolism, and storage of ingested food. The thermogenic effect of a meal can vary between individuals, however, the thermogenic effect of the food usually amounts to approximately 10% of the caloric value of the ingested food (Jequier, 1984, Danforth, 1985).

Thermic Effect of Isocaloric Meals

The thermic effect of carbohydrate and protein is higher than that of fat (Forbes, 1987, Lean and James, 1988). The thermogenic effect of dietary protein, carbohydrate, and fat has been estimated to be 25, 10, and 3 percent of calories ingested, respectively (James and Trayhurn, 1981). The thermic effect of a meal has been shown to be highly correlated to the caloric content of the meal, and not correlated with body weight, fat-free mass, or fat mass (D'Alessio, 1988). The postprandial time lag accompanying an increase in metabolic rate above control levels associated with meals of carbohydrate (glucose) is different from that for fat meals, which differs for protein meals. A protein meal elicits a peak response at 1.5 to 2.0 hours after ingestion, and metabolic rate does not return to baseline for several hours. After a glucose meal, metabolic rate rises to a peak at about 45 minutes, declining to resting levels again by two hours post-prandial. The resultant metabolic rate after a fat meal is small and gradual, with no clearly defined peak (Garrow, 1986). The average person's TEF is about 7 to 10 percent of total ingested calories (Ravussin, Lillioja, Anderson, Christin, Bogardus, 1986). Increased postprandial thermogenesis after simple compared with complex carbohydrates in two carbohydrate-rich isoenergetic meals was significantly higher after the simple than after the complex carbohydrate meal (Raben, 1994).

Thermic Effect of Feeding and Fiber

The resting metabolic rate and the thermic effect of feeding and varying dietary fiber content in obese and non-obese males, both delayed and attenuated the thermic effect of feeding (Dudani, 1986). High fiber diets have been shown to decrease the postprandial rise of glucose and insulin levels and may also be beneficial in reducing total and LDL cholesterol levels. However, data on high fiber diets as part of a hypocaloric treatment and obesity are not conclusive (Scalfi, 1987).

Thermic Effect of Feeding in Vegetarians and Non-Vegetarians

Limited studies have evaluated the significance of the effect of TEM on vegetarian meal consumption yet current national nutrition goals were established with little information regarding the impact of variations in ingested macronutrients on resting energy expenditure. This study concluded that vegetarians showed a similar RMR to and a lower TEM than that for non-vegetarians (Poehlman, Arciero, Melby, Badylak, 1988).

Thermic Effect of Feeding and Body Composition

Segal and others (1985) concluded that body composition was a significant determinant of postprandial thermogenesis in male subjects matched for similar total body

weight and Body Mass Index. Additionally, Segal (1985) showed that the thermic responses of obese subjects were significantly reduced compared with lean subjects. The relationship between energy expenditure and body composition, in terms of fat and fat-free masses has been described by a variety of predictive regression equations. The relationship between resting metabolic rate and body weight have been addressed, in addition to the difference in energy expenditure between men and women with the same body weight (Garby, Garrow, Jorgensen, Lannert, Madsen, Sorensen and Webster, 1987).

Thermic Effect of Feeding and Meal Size

The TEF has been found to be higher with larger meal size (Hill, 1984). The duration of measurements of postprandial thermogenesis varies greatly among reported studies, but is generally limited to a few hours. Most studies on thermogenesis in man are discontinued before the energy expenditure has been allowed to return to the pre-meal base-line and therefore a part of the thermogenic response is not measured.

The alteration of meal frequency on TEM and intermediary metabolism of normal, obese, hyperlipidemic, and diabetic subjects remains to be of interest among researchers directed towards the debate of metabolic advantages of nibbling versus gorging (Jenkins, Wolever, Vuksan, Brighenti, Cunnane, Venketeshwer, Jenkins, Buckley, Patten, Singer, Corey and Josse, 1989).

It has also been demonstrated that the thermic effects of both constant and relative

caloric loads were significantly blunted for obese compared with lean men when the thermic effect of food is expressed either in absolute form or as a percentage of the calories ingested (Segal, Edano, Blando, Pi-Sunyer, 1990).

The effects of oropharyngeal stimulation on the thermogenic response to feeding in obese and healthy individuals has been observed in the response to the incongruity of findings between lean and obese subjects. This study suggested that the oropharyngeal stimulation elicits a greater thermic response to feeding in normal weight subjects than obese subjects (Garrel and deJonge, 1994). The role of dietary palatability in relationship to meal-induced dietary thermogenesis has also been reviewed. It has been suggested that a part of meal thermogenesis is due to food palatability and that the concomitant activation of the sympathetic system may be related to its action (Leblanc, 1985).

Recently, another study has investigated the impact of the metabolic effects of a change from the usual diet to the currently recommended low-fat diet (eg, dietary fat of 25-30%) on weight and body composition over a one month period of time. in lean and obese women (Roust, Hammel and Jensen, 1994). This study suggested that although body-fat distribution in obesity is an important predictor of many of the metabolic abnormalities associated with obesity, it does not play a significant role in the energy metabolic responses to the generally recommended changes in diet composition.

Thermic Effect of Feeding With Aerobic Exercise Before and After Consumption of Meal

Micronutrient utilization and efficiency continues to be an area of growing interest in the study of human energy balance and weight regulation. The advantage of an increased TEM is thought to be of benefit in the long-term regulation of body weight. Variations in the magnitude, direction and residual impact changes in TEM and the overall relationship with RMR have been inconsistent among reported findings. The source of the inconsistencies is due to the breadth of the impact of the TEM based upon the variations of the following: the physiological spectrum of lean, obese and normal weight subjects; parameters of the conditioning range of sedentary, active, athlete or endurance athlete; the exercise intensity protocol; the meal composition including caloric content, fiber content and food temperature; and the timing of ingestion either pre or post exercise.

There have been numerous investigations over the past decade examining the effects of meals varying in caloric content (D'Alessio, Kavie, Mozzoli, Smalley, Polansky, Kendrick, Owen Bushman, Boden and Owen, 1988) and type of micronutrient composition on postprandial thermogenesis (Swaminathan, HolmFeld, Sowel, Baker and Wales, 1985). Several groups have investigated the effect of an acute bout of exercise on the thermic effect of food (TEF). Some investigators have demonstrated an increase in

the TEM (Bradfield et al., 1968; Miller et al. 1967; Segal and Gutin, 1983; Nichols et al., 1988; Segal et al., 1984, 1985; Zahorska-Markiewicz, 1980). However, other investigations failed to demonstrate such an effect. (Dallosso and James, 1984; Jones et al. 1963; Pacy, 1985; Samueloff et al. 1982; Swindells, 1972 and Welle 1984). One important difference between these two groups of studies is in the exercise protocol employed. In most studies, a large test meal was administered prior to exercise and TEF was measured either during or following exercise suggesting that aerobic exercise, before eating may alter the thermic effect of food. This experimental approach has yielded conflicting results and there remains to be considerable conflict regarding the thermic responses to various combinations of meal and exercise. Many of the beneficial effects of exercise are dependent on both the intensity an duration of the work performed (Pollock, 1973). In those studies in which TEF was not potentiated by exercise, the exercise tended to be of mild intensity (35% VO2max) and relatively short duration (15-20 min.) Thus, the failure to demonstrate an exercise effect on TEF may be attributed to an insufficient exercise stimulus. However, not all studies support the notion that exercise potentiates the thermic effect of a meal. Studies failing to show a potentiation of TEF by exercise have used exercise of short duration and/or low intensity (Belko, Barbieri and Wong, 1986) and (Zahorska-Markiewicz, 1980). However, when exercise is sufficiently intense, greater than 60% VO_2 max, and prolonged to deplete muscle glycogen stores, a potentiation of TEF by exercise has been demonstrated (Treadway, Young, 1990). The

body of experimental evidence favors an exercise-induced augmentation of the thermic effect of food in the nonobese individual; however, the nature of this effect remains unclear.

Other studies have suggested that aerobic exercise after administering a meal may also alter the thermic effect of a meal. (Segal, Chun, Alexander, Coronel and Valdez, 1992), (McDonald, Wickler, Horowitz, Stern, 1988), and (Segal, Gutin, Nyman and Pi-Sunyer, 1985) This information is relevant to a diverse cross-section of the population interested in the fine control of body weight, such as individuals on weight control programs or athletes trying to meet a weight limitation.

There also appears to be a dichotomy between lean and obese subjects (Bray 1974), (Segal, 1983), (Segal, 1984), (Segal, 1987), (Nichols, 1988) and (Segal, Chun, Coronel, and Valdez 1992).

Currently, it remains unknown how long the synergistic effect of eating and exercise upon metabolism endures. Recently, Two studies demonstrated that in normal subjects the thermic effect of a meal was greater after exercise than at rest (Maehlum, Grandmontagne, Newsholme and Segerstead, 1986) and it has been observed that a bout of prior exercise potentiates the thermic effect of an oral glucose load (Young, Treadway, Balon, Gavras, and Ruderman, 1986). The effects of prior exercise on the thermic effect of differing carbohydrates, glucose and fructose has been demonstrated to further ascertain the role of insulin (Balon and Welk, 1991). Variations in exercise intensity may

have profound effects on metabolic and hemodynamic responses to exercise(Dagenais, Oril, and McGregor, 1965) and (Kelbaek, Munck, Christensen, and Godtfredsen, 1987). However, discrepancies have been attributed to the differences in experimental methodology. Furthermore, previous investigations that focused on the differential response of lean and obese individuals to eating and exercise and this emphasis has resulted in overlooking the true exercise effect make metabolic measurements for only a short period of time postexercise, or require exercise performance of insufficient intensity. The impact of work intensity on the postexercise thermic response to a mixed meal has been compared in lean verses obese subjects (Segal, Chun, Coronel and Valdex, 1992). Other factors in the variation of work intensity can generate difficulty in the delineation of the true thermic effect of the meal. Factors such as the total energy cost of the exercise, degree of glycogen depletion, and the body temperature are also affected by variations in the work intensity protocol (Gaesser, 1984).

Divergence among investigators may be due to: 1) the timing of indirect calorimetry measurement protocol; 2) differences in intensity and duration of the exercise training protocols; 3) mode of exercise; 4)genetic predisposition or endowment; 5) gender; 6) adaptations in body composition. The central question remains, despite the integration of new information linking the variation in the thermic effect of activity with the variations in RMR due to manipulations in TEF.

Thermic Effect of Feeding and Effect of Exercise Training

The relationship between the thermic effect of feeding and resting metabolic rate in terms of direction and magnitude of impact in response to chronic exercise training have been inconsistent (Poehlman, 1988). Some researchers have found TEM to be elevated with increasing fitness levels (Hill, 1984, Davis, 1983). In contrast, other investigations have reported an inverse relationship (Poehlman, 1988, Leblanc, 1984, Tremblay, 1983). A significant curvilinear relationship was identified by Poehlman (1989) between VO_2 max and TEM in a group of 28 young males.

Strenuous exercise has been shown to dramatically affect resting metabolic rate. Calories expended per kilogram of body weight steadily increase with an increase in physical work (Neiman, Butler, Pollet, Dietrich, 1989). Prolonged bouts of exercise can increase the overall heat production of the body to twenty times in trained athletes (Guyton, 1986). A higher resting metabolic rate and lower TEM persisted between trained and untrained subjects when groups were matched for fat free weight and body fat thus supporting the evidence of a higher resting energy rate in endurance athletes (Poehlman, Melby, Badylak, 1988).

Poehlman, (1989) stated that the lack of concordant results among studies could be due to several methodological factors including the following:

1. Failure to examine a wide range of fitness levels; most studies have classified subjects into two discrete fitness groups (i.e. trained and

untrained).

2. The use of different criteria to define trained and untrained individuals.

3. Insufficient sample size to detect differences in RMR and TEM among individuals varying in aerobic fitness.

Thermic Effect of Feeding in Lean versus Obese

Others have examined the thermic effect of food and exercise in relationship to lean versus obese women and found that when food is ingested before a bout of exercise, the thermic response to the exercise is increased over the response to the exercise alone. This effect was shown to be considerably less in obese females as opposed to lean females. (Segal, Gutin, 1983). Comparison of the exercise response with or without an antecedent meal in men matched for age, weight, and height but differing in percent body fat, showed the increase in thermic activity in the subjects having 10 percent body fat to be higher than the thermic response in men at the higher end of the body fat spectrum or 30% in this study (Segal, 1985). It has been suggested that the sustained increase in thermogenesis and the thermic response to food could have limited significance in the overall impact on obesity (Nieman, 1990). However, clinical significance in the relationship of thermogenic augmentation remains debatable due to the fact that a full explanation of the physiological parameters for why people gain and lose weight has not been provided.

Post Exercise Oxygen Consumption

A sustained elevation of oxygen consumption after exercise remains controversial and uncertain as to the degree and duration of the increase in resting metabolic rate following acute exercise. It has been suggested frequently that exercise promotes weight loss by increasing caloric expenditure at rest over and above the caloric cost of the exercise itself due to the sustained effect on VO_2 during recovery. Elevated oxygen consumption or Exercise Post Oxygen Consumption also known as (EPOC) has been estimated in the literature to be elevated from 7 to 48 hours (deVries, Gray, 1983). However, others have reported a lack of sustained increase in VO_2 following exercise in fit and unfit subjects (Freedman-Akabas, Colt, Kissileff, Pi-Sunyer, 1985.) The exact biochemical basis for EPOC still remains a mystery. However, it is speculated that an increased rate of substrate cycling in muscle tissue. Furthermore, it is also hypothesized that catecholamines play a vital role in the increased rate of the triglyceride/fatty acid cycle leading to the stimulation of the rate of substrate cycling (Maehlum, Grandmontagne, Newsholme 1986).

Temperature and Energy Metabolism

Environmental temperature is one of the two most potent influences on metabolic rate. The effect of environmental temperature depends strongly on whether the body

temperature is permitted to vary with the environmental temperature. Humans are identified as homeotherms which means their body temperatures are physiologically regulated to stay close to a certain core temperature. Humans are able to maintain a core temperature of approximately 98.6 degrees Fahrenheit or 37 degrees Celsius, while the ambient air temperature vary over wide extremes. In humans, metabolic rate increases during exposure to low temperatures. For a given duration and intensity of exercise, body temperature increases are higher in untrained men than in trained men (Morehouse and Miller, 1963).

Psychogenic Effects on Metabolic Rate

Oxygen uptake has been shown to increase with excitement and decrease with relaxation. Cognitive thought processes can independently influence metabolism (Morgan, 1983).

Sleep effects on Metabolic Rate

The metabolic rate has been shown to decrease 10-15% during sleep. This phenomena has been credited to a decreased tone of skeletal or postural muscles, in addition to the decreased activity of the sympathetic nervous system (Guyton, 1986).

Disease Status and Effect on Metabolic Rate

Severe pathological stresses such as burns, infections, fever, cancer and surgery can alter metabolic homeostasis (Kern, 1988).

Review of Energy Metabolism

Living organisms neither consume nor create energy but rather transform energy from one form to another form. This is consistent with the first law of thermodynamics which in essence states that energy can be neither created nor destroyed. Adenosine triphosphate commonly abbreviated as ATP is the major carrier of biologically usable energy in all living matter. The interrelationships of energy yielding and energy-requiring metabolic reactions may be considered largely as processes that couple the formation of ATP with its breakdown. In humans, the synthesis of ATP occurs in the mitochondria.

Whenever the heart contracts, or the brain generates nerve impulses, or the bone marrow generates a new molecule of hemoglobin, the energy required is derived from ATP molecules, in the process breaking them down to yield ADP again. ADP and ATP serve as key energy carriers, picking up energy from food molecules and then releasing it to perform various forms of work.

Fragmentation of Complex Food Molecules

Food must undergo oxidation in order to yield biologically useful energy. Oxidation does not necessarily involve oxygen, although it must involve the transfer of electrons from a donor molecule to a suitable acceptor molecule. Catabolism is the breakdown of larger food fragments into the representative constituent components (figure 8).

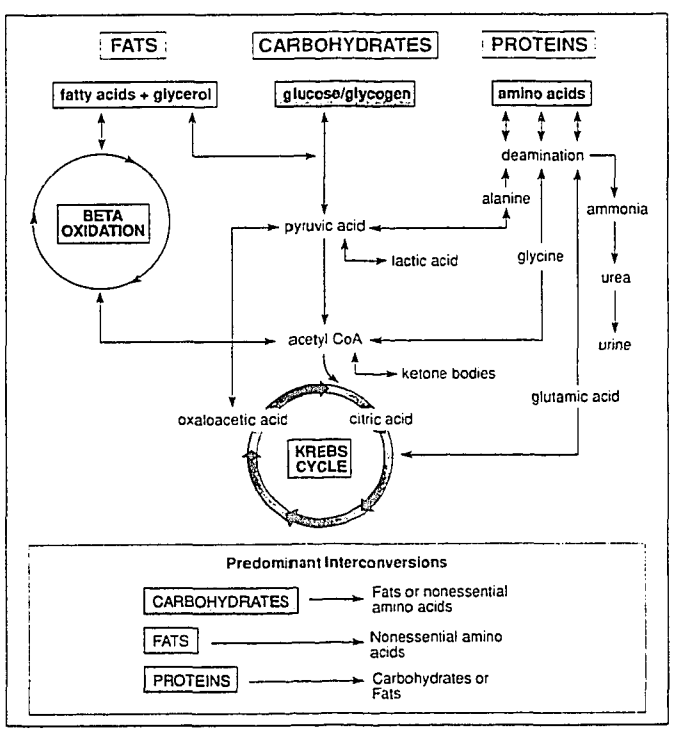


Figure 8 The "metabolic mill" defines the interrelationship of fat, protein and carb.

The immediate source of energy for most types of physiological work is the splitting of the chemical bonds of ATP. The major processes by which most animals transfer energy from the chemical bonds of nutrient molecules to the bonds of ATP is an intricate series of biochemical reactions known as cellular respiration or aerobic catabolism. Each cell of the body is equipped with the enzymes and cell components to carry out aerobic catabolism and to make its own ATP (Figure 9).

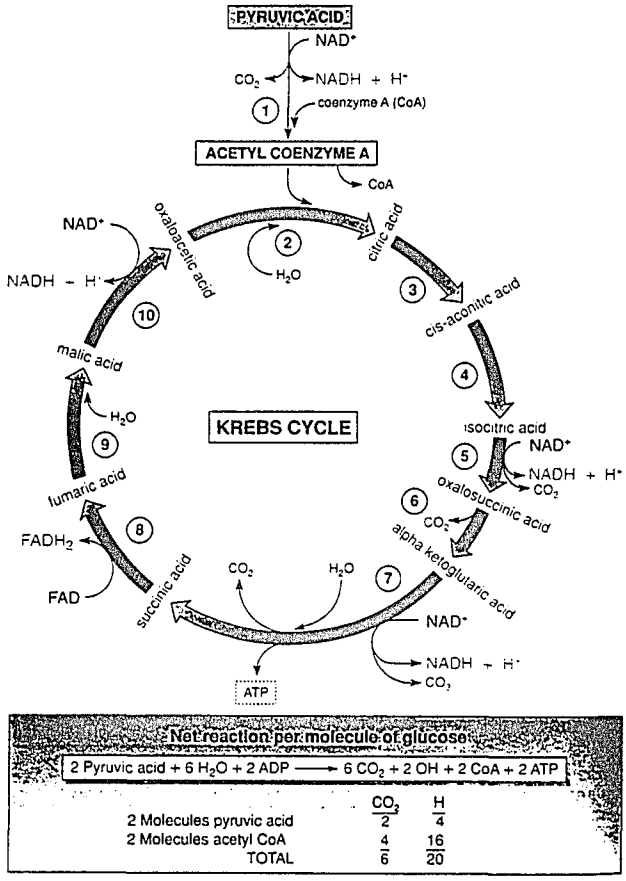


Figure 9 Krebs Cycle

All of the macronutrients can be oxidized by aerobic catabolism. There is division of labor among tissues in the handling of different nutrients, however some tissues are limited in the types of nutrients they use. In humans, the liver catabolizes lipids, carbohydrates, and amino acids. Muscle tissue utilizes lipid and carbohydrate almost exclusively, and the brain ordinarily depends exclusively on glucose as a source of energy. Adipose tissue and liver tissue both play a pivotal role in managing delivery of fuel molecules to the rest of the body in humans and other mammals.

After a meal, products of lipid digestion and glucose from digestion are taken up from the body by adipose tissue and deposited as fat. Until the next meal, adipose tissue then meters out fatty acids into the blood to meet the catabolic demands of all other tissues. Between meals, glucose is metered into the blood by the liver to meet the body's glucose needs. Amino acids are used preferentially to make proteins throughout the body. But, when amino acids are available in excess they are converted to carbohydrates or lipids which can be utilized for energy (Stryer, 1988).

The release of chemical energy from food materials essentially occurs in three phases. During Phase I, the large molecules that make up the bulk of the food materials are broken down into smaller constituents units: proteins are converted to the 20 or so different amino acids; carbohydrates such as glycogen are degraded to sugars such as glucose; and fats are broken down into fatty acids and glycerol. The amount of energy liberated in phase I are relatively small: approximately, 0.6 percent of the free, or useful

energy, of protein and carbohydrates and about 0.1 percent of that of fats, is released during this phase. Because this energy is liberated largely as heat, it cannot be utilized by the cell. The purpose of the reactions of phase I, which can be grouped under the term digestion is to prepare the macronutrients for the energy-releasing processes.

The second phase of the release of macronutrients into energy is termed incomplete oxidation. The smaller constituent nutrients derived in phase I, sugars, glycerol, fatty acids, and the variety of amino acids are incompletely oxidized by the removal of electrons or hydrogen atoms, with the end products being carbon dioxide and water, the remaining compound being predominantly acetyl coenzyme A with remnants of oxaloacetate and a-oxoglutarate.

Phase III is comprised of the total oxidation of the relatively few products of phase II and occurs in a cyclic sequence of chemical reactions known as the tricarboxylic acid or the citric acid cycle (TCA), or the Krebs Cycle. The TCA cycle is initiated by the 4-carbon oxaloacetate and acetyl coenzyme A, generated from phase II. Subsequent reaction in the TCA cycle result in the reformation of oxaloacetate and the formation of two molecules of carbon dioxide. The carbon atoms that go into the formation of the carbon dioxide are no longer available to the cell. The progressive stepwise oxidations in which hydrogen atoms or electrons are removed from intermediated compounds formed during the cycle and, via a system of carriers, transferred ultimately to oxygen to form water and are quantitatively the most important means of generating ATP from ADP and

inorganic phosphate. These events have been termed as cellular respiration and oxidative phosphorylation.

Hormonal Control of Macronutrients

Major hormones that are involved in control of the human body's use of carbohydrates, lipids, and proteins include insulin and glucagon from the pancreas, cortisol and other glucocorticoids from the adrenal cortex, epinephrine and norepinephrine from the adrenal medulla and sympathetic nervous system, thyroxin and tri-iodothyronine from the thyroid gland, and growth hormone from the pituitary gland.

Several hormones control use of nutrients over the daily cycle of eating and fasting. Following the ingestion of a typical meal, insulin secretion increases and glucagon secretion decreases. The result is that the breakdown of body fat is inhibited and synthesis of fat in adipose tissue is enhanced, the liver is stimulated to store rather than release carbohydrates, and entry of blood glucose from the meal into the cells of many tissues is enhanced which promotes the catabolism of carbohydrate as the source of energy in those tissues. A few hours after the consumption of a meal, when the blood's supply of nutrients from the meal is depleted, fuels are provided to the body by two processes, both promoted in part by the increased secretion of glucagon and by the decreased secretion of insulin. First, liver glycogen is broken down for release of glucose into the blood, and

second, fasting adipose tissues are broken down to release fatty acids.

Growth hormone is a pivotal controlling agent in both the growth of young individuals and the maintenance of structures in adults. It increases protein synthesis and decreases the use of amino acids merely as sources of energy. It also mobilizes fat and inhibits insulin action. There are numerous hormonal influences on metabolism, and the interrelation among them are often complex feedback systems. (Figure 10) reflects a diagram of catecholamines and cortisol act to promote fuel mobilization.

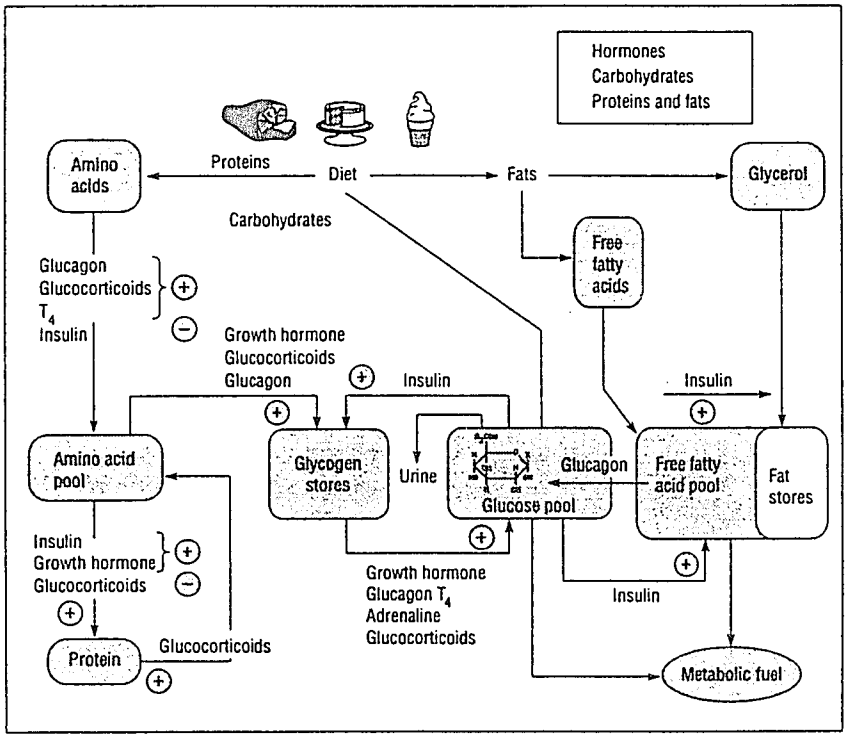


Figure 10 Hormonal Control of Fuel Mobilization

Aerobic Support of Human Exercise and Energy Metabolism

The level of physical activity is a powerful influence on the rate of energy utilization in all animals. Increased muscular activity requires an increased rate of supply of chemical energy in the form of ATP. In most forms of human exercise, the aerobic catabolic machinery centered in the mitochondria of the muscle cells is the main factor in meeting the demand for ATP. Exercise that can be sustained entirely by aerobic ATP production is called aerobic exercise. Glycogen, glucose, and fatty acids can serve as fuels for aerobic catabolism in the muscles.

Oxygen consumption is also essential. Therefore, so is the rate at which the lungs and circulatory system supply oxygen to the muscles. An individual's maximal rate of oxygen consumption is a measure of the highest rate at which aerobic catabolism can supply energy in support of exercise. It is a useful index of physical condition. The maximal rate of oxygen consumption increases with physical training. The strenuousness of a aerobic activity for an individual can be assessed approximately by determining how large a percentage of the individual's maximum rate of oxygen consumption is required by the exercise. An activity requiring 25-35% of his or her maximum rate of oxygen could be continued all day, but activity requiring 75-85% of maximum oxygen uptake would likely be totally exhausting in one to two hours.

Anaerobic Support of Human Exercise and Metabolism

Anaerobic glycolysis provides the muscles with the ability to make ATP without oxygen. The glycolytic mechanism uses glucose and glycogen as its exclusive fuels and produces lactic acid as well as ATP. Lactic acid is not excreted, and once it has accumulated to a critical level in a person's body, the individual succumbs to fatigue. Because of this, anaerobic glycolysis is limited in the total number of ATP molecules it can generate during a particular bout of exercise. By contrast, the products of aerobic catabolism, carbon dioxide and water do not pose limits on the duration or intensity of aerobic ATP production.

Despite the limited capacity of anaerobic glycolysis to supply ATP to working muscles, it can be a vital adjunct to aerobic ATP production in two ways. First, when the aerobic mechanisms are producing ATP at their maximal rate, the anaerobic mechanism can be brought into play for a limited length of time to raise the rate of ATP production even further and thus permit a greater intensity of muscular effort than the aerobic limitations alone would allow. Secondly, unlike aerobic mechanisms the anaerobic mechanism can raise its rate of ATP production to a high level almost instantaneously. The rate of aerobic ATP production in the muscles is strictly dependent on the rate at which oxygen is delivered by the circulation, and oxygen delivery increases only gradually after the onset of exertion with increased respiration. Anaerobic glycolysis is not tied to the performance of the circulatory system in this way. Sudden intense exercise is

supported initially by anaerobic glycolysis until aerobic ATP production has a chance to catch up.

If anaerobic glycolysis has been used during a bout of exercise, the lactic acid accumulated must be metabolically eliminated after the exercise has stopped.

Energetics of Exercise

Two types of exercise are supported primarily by aerobic ATP production. The first, is exercise requiring substantially less than an individual's maximal rate of oxygen consumption, such as walking for nearly everyone, and even rapid jogging for well conditioned individuals is nearly entirely aerobic. There will be some demand for oxygen deficit mechanisms at the start of such exercise while the rate of circulatory oxygen delivery is increasing but thereafter, the ATP demand will be met aerobically because it falls well within the body's capability for aerobic ATP production (Figure 11).

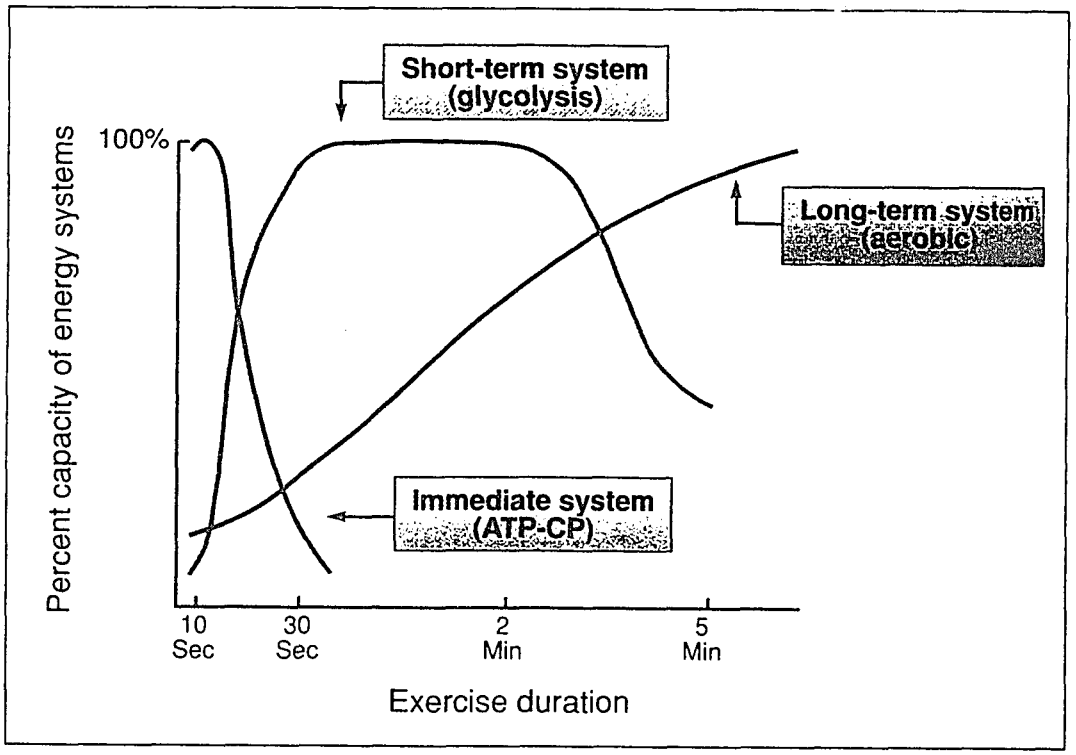


Figure 11 Various energy systems and their involvement during exercise of different durations.

The second environment is in the case of long-duration aerobic exercise. Events such as marathons or competitive cross country skiing require an elevated rate of ATP production over a long duration. This energy must be supplied aerobically because the total cost of ATP is so great it would quickly exhaust the ATP productive capacity of anaerobic glycolysis. Anaerobic glycolysis assumes a large role in the supply of ATP in quick, high intensity exertion such as events lasting about 1 minute because the time is too short for an appreciable increase in the oxygen supply to the muscles. Even if the time is adequate for the rate of ATP production to reach maximal levels, exercises that demands ATP at a greater rate than can be provided aerobically will require a continuing anaerobic contribution of ATP.

Summary

Historians agree that a close connection between diet, exercise and medicine was evident to three ancient physicians, 500 years prior to the birth of Christ. Yet, a crude concept of metabolism did not appear until Galen's speculation in the 17th century. By 1842, foods had begun to be identified as protein, carbohydrate, and fat and their caloric value was measured. The middle to late nineteenth century brought the closed and open

circuit indirect calorimetry techniques used to study disease state on human metabolism (Kinney, 1983).

By the turn of the century, Max Rubner and his associates established that the basal heat production of different species varied with their body surface area. Rubner is also credited with the discovery of energy metabolism that was stimulated by the ingestion of food, which is now termed the thermic effect of feeding or food.

The establishment of several major centers for the study of human metabolism in the early 1900's laid the ground work for many of the metabolic questions that are currently being investigated by today's researchers. The last 100 years since 1890 have been a period of rapid development of the science of nutrition and exercise physiology. The multidiciplinary integration of related areas such as exercise physiology, nutrition, physics, chemistry, biochemistry, physiology and computer science has allowed for the fruition of physiological science and continually contribute valuable information about diet and exercise to provide solutions to important medical problems as well as practical applications for improved overall health for humans.

The traditional measure of energy in biology is the calorie, defined as the amount of heat required to raise the temperature of a gram of water by 1 degree Celsius. The superficial simplicity of this simple word "kcal", has stirred more controversy and debate in scientific investigation than any one word in the brief history of biological science, and yet much of it's physiological integration in terms of human energy expenditure remains a

fascinating and challenging unknown.

Human energy expenditure can be measured in a variety of ways, including direct and indirect calorimetry. Direct calorimetry remains expensive, cumbersome and labor intensive. The alternative indirect method of calorimetry is more practical clinically for measuring energy expenditure. While there is agreement among investigators regarding the components of energy expenditure, there is broad spectrum debate about the details surrounding energy expenditure and all of the impacting parameters and protocols for data collection. There is conflicting evidence about the effect of RMR and TEM, TEM and meal size, TEM and bouts of exercise prior to meal consumption, as well as ingestion of meals prior to exercising, TEM between males and females, TEM between fit and unfit subjects, TEM and exercise intensity, TEM in lean and obese subjects, and even controversy regarding the TEM in vegetarian and non-vegetarians. A more descriptive explanation for why people gain and lose weight remains open. This controversy requires focus on a better understanding of the body's efficiency in handling various fuels from the diet as well as the extent to which an exercise augmented dietary-induced thermogenesis contributes to weight control.

CHAPTER 3

METHODS

The purpose of the current study was to compare the thermic effect of a 2510 KJ (600 kcal) meal composed of 100% carbohydrate with an equivalent caloric meal consisting of 90% fat and 10% carbohydrate after a prior bout of exercise (70% max VO_2), to determine whether the increase in dietary - induced thermogenesis after exercise was dependent on the dietary composition of the food ingested.

Subjects

The subjects for this particular study were fifteen females, ages 22 to 31 years of age (Table 1). All were apparently healthy with above average fitness level but not highly trained. They were weight stable for at least one year prior to the study. Subjects received an explanation of the procedures and gave informed consent, and the risks and benefits of the study were explained. The study was reviewed and approved by the University of Nevada-Las Vegas Human Subjects committee. All subjects were experienced with the measurement of O2 consumption using a mouthpiece and had participated in other studies in our laboratory and were recruited from the exercise

TABLE 1

Subject Characteristics

Sub. #	Age	wt(kg)	ht(cm)	% fat	VO_2Max L/min
1	24	51.5	152.5	26.5	2.64
2	27	64.4	171.1	23.4	2.90
3	21	47.5	161.4	16.8	1.91
4	24	64.3	172.0	21.0	3.07
5	27	56.2	163.5	21.9	2.11
6	26	50.6	162.5	19.5	2.13
7	22	55.9	161.5	21.5	2.33
8	21	63.5	-	20.1	2.78
9	21	72.7	-	22.3	3.40
10	24	48.0	155	17.5	1.87
11	25	58.0	172	13.0	1.97
12	32	55.0	154	19.0	2.68
13	24	70.0	171	19.8	2.66
14	24	68.0	170	25.5	2.61
15	24	66.0	164	13.8	3.13

physiology graduate student population. They initially underwent a test of maximal aerobic power (VO_2max).

Experimental Design

Risks and benefits of the study were reviewed with each subject participant in an informed consent form (appendix A). Subjects initially underwent a test of maximal oxygen uptake capacity determined by open circuit spirometry by pedalling a cycle ergometer at progressively increasing workload (50 W min-1) until volitional exhaustion. Resipiratory exchange measurements were made using a calibrated Vacumed integrated metabolic system. Subjects breathed through a two-way non-rebreathing valve, and minute volume of inspired gas was measured and minute samples of expired gas were analyzed for oxygen and carbon dioxide concentrations. All subjects were familiar with the testing procedures in our laboratory. A recent study confirmed that a mouthpiece and non-rebreathing valve is an acceptable alternative to the ventilated hood for the determination of energy expediture at rest in subjects accustomed to using a mouthpiece (Segal, 1987).

Treatment Description

The subjects participated in the conduction of four trials on non-consecutive days. Trial #1 and Trial #2 were control days in which the subjects consumed either the carbohydrate of fat load without a prior bout of exercise.

Trial #3 and Trial #4 were trials of exercise and the consumption of either the carbohydrate or fat load after returning to baseline after exercise. Subject scheduling was based upon their convenience and availability.

Treatment Day Protocol and General Procedures

1. On the day of the experiment, subjects reported to the laboratory upon rising, 6:30-7:00am.
2. 12-15 hours following their last meal and at least 36 hours after any strenous exercise.
3. Subjects weight was confirmed and recorded. Resting oxyen uptake was measured by open circuit spirometry as descibed above for 12 - 15 minutes following a five-minute equilabration period on the system. This period of time was suffecient to establish a steady-state baseline and was consistent between control and exercise days.
4. Subjects then exercised for 50 minutes at a workload predetermied to be 70% of VO_2max and this work load was confirmed once the subjects reached steady state.

5. Following exercise, oxygen consumption was again measured at 30 minute increments until it had returned to near the resting baseline rate, at which time (approximately two hours post exercise) the 600 calorie meal of either carbohydrate or fat was administered. Oxygen uptake was measured at 30 minute intervals over the next three hours to determine the thermic effect of the caloric load.
6. Thermic effect of food (kcal) was calculated from measurement of the oxygen consumption and the nonprotein RQ.
7. On a control day the procedure was repeated, except without the prior exercise.
8. The meal ingestion times were consistently the same between trials.
9. The thermic effect of the carbohydrate or fat load was calculated as the area under the three-hour response curve for oxygen uptake following the ingestion of either the fat or carbohydrate test meal.
10. For the postexercise recovery VO_2 was used as the baseline.
11. Body fat was estimated from skinfold measurements made with a Lange caliper by the equation of Durnin and Wormersley, 1974.

Statistical Analysis

Statistical analysis were performed by one-way ANOVA with repeated measures for the 3-hour thermic effect area under the curve and for comparison between the four conditions for the mean percentage increase in VO_2 over baseline. A Tukey's test was applied when the F-ratio was significant ($P<0.05$). Area under the curve was calculated using the trapezoid method. Results are represented as mean and standard error (SE).

CHAPTER 4

RESULTS

The 4 x 5 ANOVA with repeated measures revealed a significant difference between conditions at each dependent variable (mean VO_2, mean RQ, and mean kcal). Tukey's test was performed to reveal the nature of the differences. The results indicate that the thermic effect of the carbohydrate meal after exercise was 59% greater than that of the fat meal after exercise. (28.8+/- .91 vs. 15.7+/- .86 kcal/2 hours). ($p<0.05$), (Tables 2 and 7, Figure 14).

The overall thermic effect of the carbohydrate meal was 50% greater than that of the fat meal (18.6 +/- .84 vs 14.2 +/- .59 kcal/2 hours). ($p<0.05$), (Tables 2 and 3, Figures 12-15). These results suggest that the potentiation of the thermic effect of a meal by prior exercise depends on the composition of the meal, essentially excluding meals consisting primarily of fat.

The mean respiratory quotient (RQ) was significantly lower in response to the carbohydrate meal after a bout of exercise, compared with the same meal without exercise (.96 +/- .05 vs 1.06 +/- .05), ($p<0.05$) reflecting an increase in storage of glucose as glycogen.(Table 4, Figure 16). No difference in RQ was observed for the fat meal with or without exercise. Net increase in VO_2 is shown in (Table 5, Table 8, and Figure 17). VO_2 increased significantly with the carbohydrate meal after exercise compared with all other conditions (Table 6, Figure 18).

TABLE 2

Area Under Curve
for Kcals

			AREA						
		0-10	10-30	30-60	60-90	90-120			
KCAL(CHO + rest)	**Mean**	0.670	3.039	4.700	4.948	5.264	18.6210		
	Std Error	0.159	0.679	1.146	1.091	1.286	carb	47.4140	
KCAL(CHO + exer)	**Mean**	1.041	4.693	8.249	7.845	6.965	28.7930	rest	32.8507
	Std Error	0.210	0.758	1.121	1.365	1.286			
KCAL(FAT + rest)	**Mean**	0.534	2.073	3.673	4.127	3.822	14.2297	exer	44.4907
	Std Error	0.132	0.471	0.693	0.786	0.939	fat	29.9273	
KCAL(FAT + exer)	**Mean**	0.575	2.500	4.790	4.458	3.375	15.6977		
	Std Error	0.180	0.642	1.061	1.350	1.245			
		2.819	12.306	21.412	21.378	19.426			

FIGURE 12

Total/Integrated TEF
Carbohydrate vs. Fat

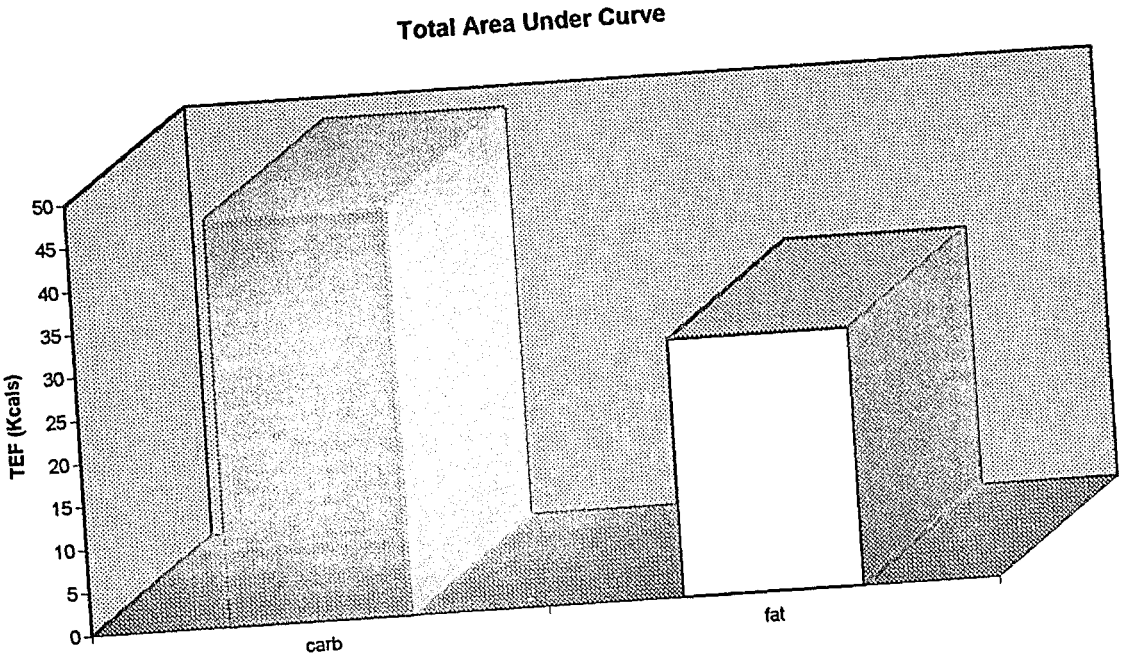


FIGURE 13

Total/Integrated TEF
Rest vs. Exercise

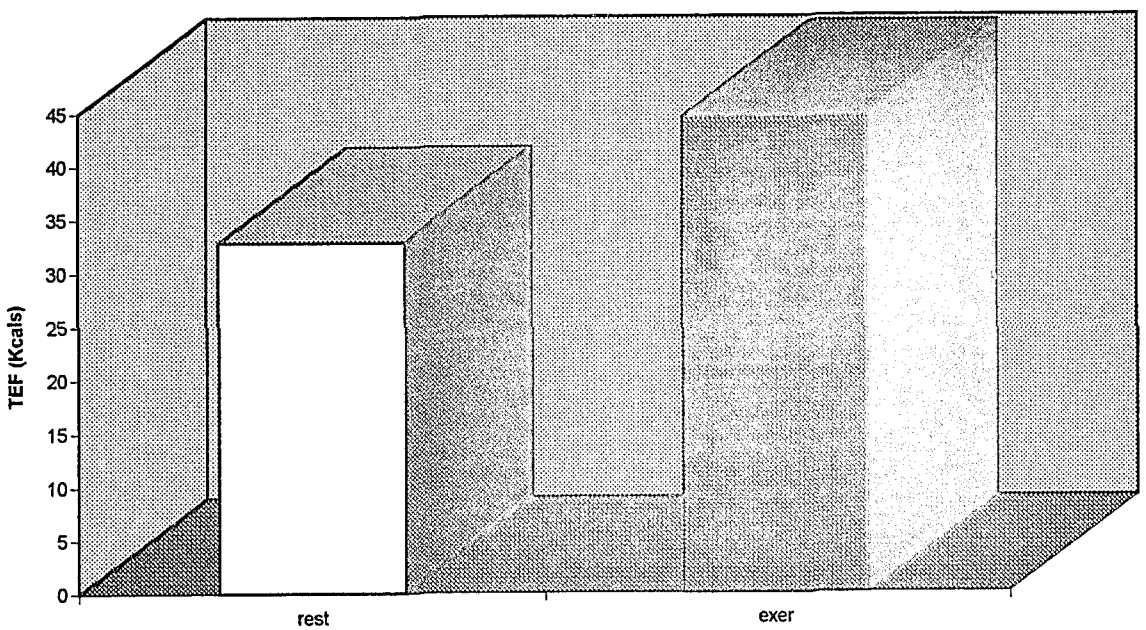


FIGURE 14

Total Integrated TEF
Fat vs. Carbohydrate
Rest vs. Exercise

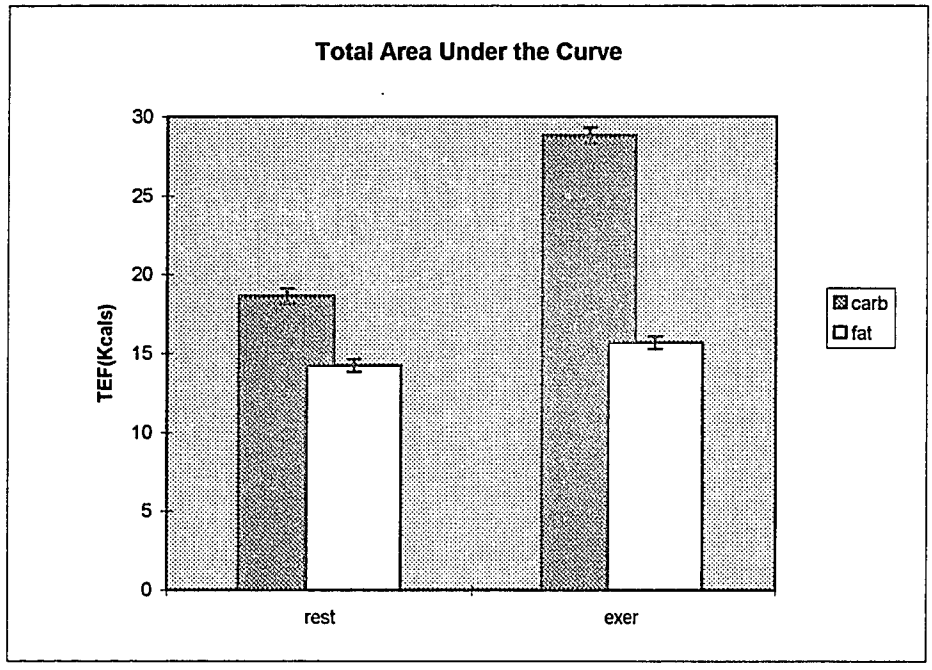


TABLE 3

TEF
Net Increase Over Baseline
(Kcals/min)

	10 min.	30 min.	60 min.	90 min.	120 min.
CHO+Rest	0.13±.03	0.17±.04	0.14±.03	0.18±.04	0.16±.04
CHO+Ex	0.21±.04	0.26±.03	0.29±.04	0.23±.05	0.23±.03
Fat + Rest	0.11±.02	0.10±.02	0.14±.02	0.13±.02	0.12±.03
Fat + Ex	0.11±..03	0.14±.03	0.18±.04	0.11±.04	0.11±.04

FIGURE 15

TEF
Net Increase Over Baseline

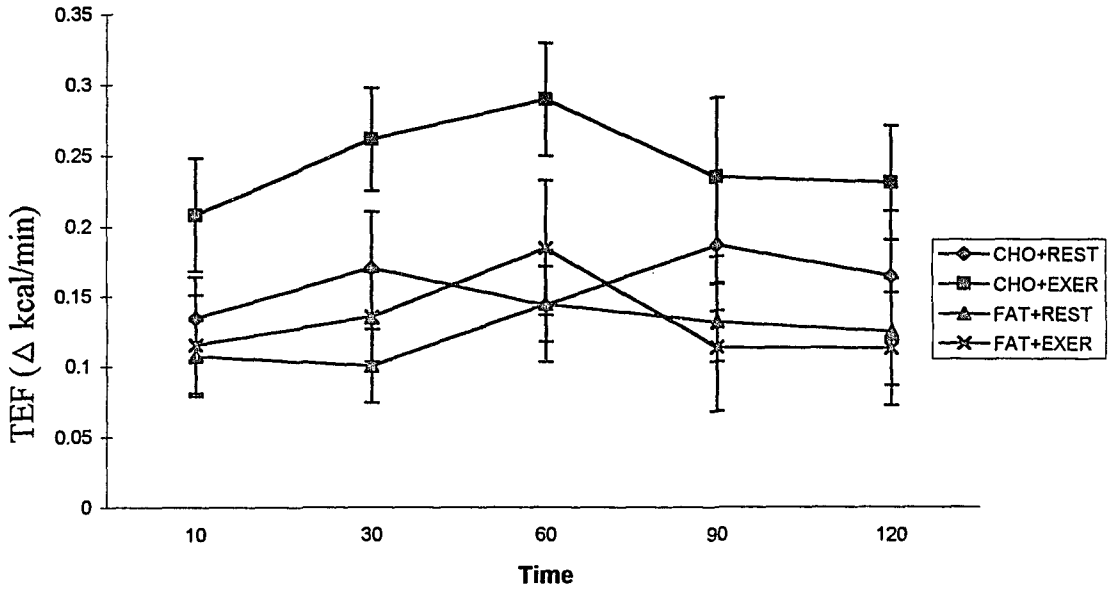


TABLE 4

Mean RQ

	Baseline	Recovery	10 min.	30 min.	60 min.	90 min.	120 min.
CHO+REST	0.82±.05		0.86±.05	0.91±.05	0.91±.05	.92±.06	.91±.04
CHO+EX	0.82±.05	0.74±.04	0.80±.06	0.85±.05	0.8±.05	0.87±.05	0.91±.05
Fat + Rest	0.81±.05		0.77±.05	0.79±.05	0.82±.05	0.83±.05	0.81±.05
Fat + Ex	0.81±.05.	0.77±.05	0.77±.05	0.79±.04	0.83±.04	0.83±.01	0.80±.06

FIGURE 16

Mean RQ

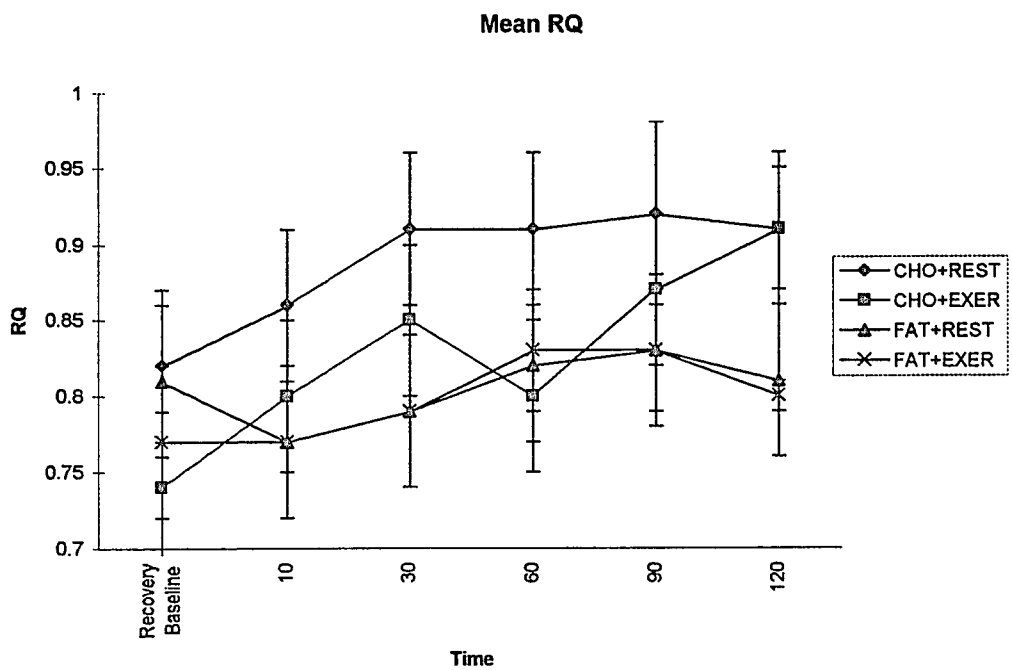


TABLE 5

Mean VO_2

	Base-line	Rec.	10 min.	30 min.	60 min.	90 min.	120 min.
CHO +Rest	0.22±.00		0.24±.002	0.24±.002	0.24±.002	0.25±.003	0.24±.002
CHO +Ex	0.20±.00	0.20± .002	0.24±.003	0.25±.003	0.26±.002	0.25±.003	0.25±.002
Fat + Rest	0.20±.00		0.23±.002	0.22±.002	0.23±.002	0.23±.002	0.23±.003
Fat +Ex	0.19±.00	.20± .002	0.23±.002	0.23±.002	0.24±.002	0.22±.002	0.22±.002

TABLE 6

Percent Change in VO2
from baseline

	10 min.	30 min.	60 min.	90 min.	120 min.
CHO+Rest	11.6	13.9	11.2	15.2	13.5
CHO+Ex	20.2	24.5	26.4	21.9	19.5
Fat+Rest	11.9	5.8	14.0	12.7	12.5
Fat+Ex	12.2	13.7	17.3	11.5	10.8

FIGURE 17

Mean VO2

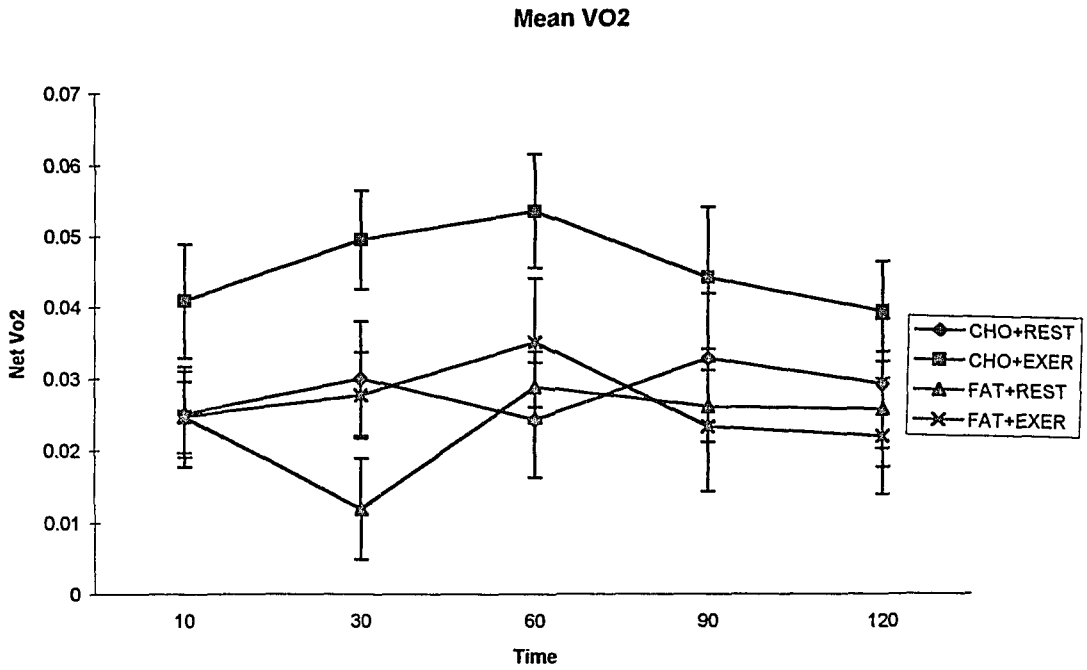


FIGURE 18

VO_2
Percent Change

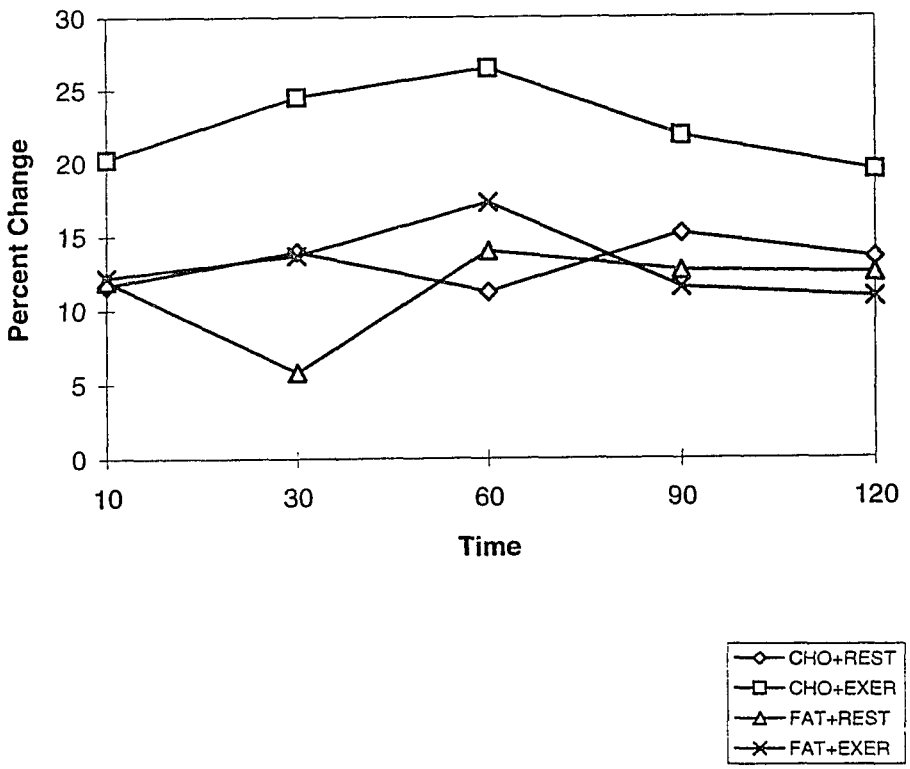


TABLE 7

ANOVA Table for Kcals

	ANOVA Table for KCAL				
	df	SS	MS	F	p
food	1	0.4272	0.4272	8.22	0.0124
exer	1	0.1705	0.1705	20.32	0.0005
time	4	0.076	0.019	2	0.1065
food*exer	1	0.1034	0.1034	2.66	0.1255
food*time	4	0.0222	0.00555	0.68	0.6076
exer*time	4	0.057	0.01425	1.68	0.1673
food*exer*time	4	0.0054	0.00135	0.16	0.9571

TABLE 8

ANOVA Table for VO2

ANOVA Table for VO2					
	df	SS	MS	F	p
food	1	0.0106	0.0106	4.56	0.0508
exer	1	0.0077	0.0077	17.41	0.0009
time	4	0.0017	0.000425	0.98	0.4262
food*exer	1	0.0037	0.0037	1.93	0.1862
food*time	4	0.0016	0.0004	1.29	0.2843
exer*time	4	0.003	0.00075	1.98	0.1094
food*exer*time	4	0.0007	0.000175	0.48	0.7511

TABLE 9

ANOVA Table for RQ

ANOVA Table for RQ					
	df	SS	MS	F	p
food	1	0.6481	0.6481	53.43	0.0001
exer	1	0.1121	0.1121	7.36	0.0169
time	4	0.1757	0.043925	18.98	0.0001
food*exer	1	0	0	0	0.982
food*time	4	0.0187	0.004675	1.22	0.3125
exer*time	4	0.0093	0.002325	1.38	0.2516
food*exer*time	4	0.0127	0.003175	1.36	0.2602

CHAPTER 5

DISCUSSION

Dietary-induced thermogenesis, or the thermic effect of food, represents approximately 10% of the daily energy expenditure in humans. TEF is comprised of two components, the energetic cost of processing and storage of glucose as glycogen in muscle and liver (obligatory thermogenesis), (Acheson, 1984, Poehlman,1989 and Horton, 1989), and an additional effect due to stimulation of the sympathetic nervous system by insulin (facultative thermogenesis) (Acheson, 1983,1984, and DeFronzo, 1984). Thus, exercise must be of sufficient intensity and duration to deplete muscle glycogen in order to potentiate the thermic effect of a meal (Maehlum et al. 1986; Nichols et al. 1986, Young, Treadway, Balon, Gavras and Ruderman, 1986). The failure to consider exercise intensity and duration in study design may account for the inconsistent findings of previous studies. Those studies (Belko, 1980; Dallosa, 1982; Jones, 1963; Pacy, 1986; Samuloff, 1984; Swindell, 1965; and Welle, 1984), which failed to demonstrate a potentiation of TEF by exercise are similar in that the exercise was of a relatively low intensity and short duration. On the other hand, TEF was potentiated when exercise was greater than 60% of maximal aerobic capacity and prolonged (Treadway and Young, 1986) . Glycogen depletion was not measured in this study, but the exercise protocol used has previously been shown to

cause approximately a 50% reduction in muscle glycogen stores (Saltin and Karlsson, 1971). Thus, meal composition should be a factor in the potentiation of TEF by prior exercise.

Insulin has been identified as a mediator of enhanced TEF in the post-exercise state (Acheson et al. 1984; and DeFronzo et al. 1984). The thermic effect of insulin has been attributed to its ability to promote glucose storage as glycogen in skeletal muscle (Schwartz et al. 1985). It has been estimated that 65% to 75% of the increase in thermogenesis during a euglycemic insulin clamp can be accounted for by the metabolic cost of processing infused glucose for storage (Ravussin and Bogardus,1982; Acheson, 1983). Approximately 2 mols of ATP are needed for each mol of glucose stored, and 1 mol of oxygen is used to produce the ATP required to incorporate 3 mols of glucose into glycogen (Ruderman et al. 1971). The remaining component of thermogenesis has also been attributed to insulin action on the sympathetic nervous system. Insulin infusion stimulates norepinephrine release (Rowe et al. 1971), independent of changes in blood glucose (Ravussin and Bogardus, 1982). Catecholamines may increase cellular respiration by stimulating the activity of the Na+/K+ pump (Gaesser and Brooks,1984), the rate of futile cycling (Newholm, 1978), or the recycling of glucose through 3-carbon compounds, primarily lactate (DeFronzo et al. 1984). Since the insulin response to a high fat meal has been shown to be decreased relative to the response to a high carbohydrate meal (Schwartz et al. 1985), a decrease in the thermic effect of the high fat meal was not unexpected in this study. The overall thermic effect of the carbohydrate meal in this study was 59% greater than the thermic effect of the fat meal. Moreover, prior exercise did not

potentiate the thermic effect of the fat meal.

In addition to nutrient absorption, insulin secretion is mediated by gastric distension (Ivy et al. 1988). Meal bulk has been shown to be a greater determinant of post-exercise TEF than was the energy content of the meal (Hill et al 1985, Young, 1994). Hence, one large meal (gorging) has a greater thermic effect than several smaller meals (nibbling) with the same total caloric content (Tia et al. 1991; Young,1994). The rate of gastric emptying is a function of meal size; a larger meal will empty at a faster rate than a smaller meal due to a greater degree of gastric distension (McHugh and Moran, 1979). This in turn will lead to a faster rate of nutrient absorption and a higher insulin response (Ivy et al. 1988). TEF also increases in response to the caloric content of a meal, although in a non-linear fashion (Hill et al. 1984). The results of this study, however, show that the composition of the meal is more important than either meal size or caloric content, since post-exercise TEF was significantly lower for the fat meal than for a calorically equivalent carbohydrate meal of the same volume.

The failure of the high fat meal to potentiate TEF after exercise can be attributed to differences in metabolic processing between carbohydrate and fat. Ingestion of carbohydrate after exercise enhances the rate of glycogen synthesis (Ivy et al. 1988). However, similar rates of glycogen synthesis were found when 225 and 450 grams of carbohydrate were ingested (Ivy et al. 1988), suggesting that carbohydrate ingestion can exceed the rate of glycogen synthesis. Overfeeding carbohydrate results in an increase in carbohydrate oxidation until carbohydrate balance is reestablished (Ravussin et al.1985). Thus RQ was higher with carbohydrate feeding in the no exercise trial than following

exercise. In contrast, there appears to be no limitation to fat storage. With fat overfeeding, lipid utilization is reduced and lipid storage is actually enhanced (Ravussin et al. 1985). Thus the potentiation of the thermic effect of a meal by prior exercise depends on the composition of the meal, essentially excluding meals consisting primarily of fat.

In summary, the results of this study indicate that the potentiation of TEF by prior exercise is a function of the composition of the meal. If bulk and caloric content are equal, a high carbohydrate meal will produce a greater thermic effect than a high fat meal. This difference can be attributed to the energetic cost of processing carbohydrate for storage as glycogen which does not occur with the fat meal.

CHAPTER 6

CONCLUSION

The purpose of this study was to examine the effect of prior exercise on the thermic effect of a meal composition of either fat or carbohydrate. Based on the results of this study it can be concluded that the thermic effect of a carbohydrate meal in conjunction with a prior bout of exercise does have a significant effect over and above the thermic effect of a fat meal combined with a prior bout of exercise. The hypothesis tested was that the thermic effect of a carbohydrate meal combined with exercise of significant intensity, does not differ from a test meal composed of fat combined with significant exercise intensity. Thus, this hypothesis was rejected based upon the findings of this study which supports a significant differentiation between the two test meals and exercise conditions.

Future Research

Important questions remain unanswered regarding the influence dietary composition and the influence of physical activity and represent interesting areas for future research. Macronutrient intake in specific populations in conjunction with the impact of physical activity and energy expenditure provides a window of opportunity to further explore and advance our understanding of energy metabolism and will help develop strategies and protocols that can be designed to specifically plan for nutritional and energy requirements in exercising and non-exercising individuals, accounting for physiological variations in lean versus the obese populations as well as refining genetic variations and gerontological adaptations.

APPENDIX I
Submission Request to Human Subjects Committee

Research Abstract

The Effect of Prior Exercise on The Thermic Effect of Carbohydrate versus Fat Feeding in Man

1. **Subjects:** Eight female Graduate Assistants in the Exercise Physiology were asked to volunteer for the study.

2. **Purpose, Methods, Procedures:** The purpose of this study is to compare the thermic effect of a carbohydrate load to an equivalent caloric load of fat after a prior bout of exercise. Subjects were asked to participate in a study to determine whether the increase in thermogenesis (heat produced by the body) after exercise is dependent on the type of food ingested. Prior to initiating this study, subject's maximal oxygen uptake capacity will be determined by open circuit spirometry by pedalling a cycle ergometer at a constant speed and progressively increasing the workload until voluntary exhaustion. The experiment itself will consist of riding on the cycle ergometer at 70% of subject's maximal oxygen uptake capacity for 45 minutes. Prior to exercise, resting oxygen uptake will be measured for a period of time sufficient to establish a baseline. Oxygen uptake will again be measured following the exercise to verify the return to the baseline condition. When the baseline has been reestablished, (approximately 1-2 hours after exercising), the subject will ingest one of the following 600 calorie meals: a) 100% carbohydrate, or b) 90% fat, 10% carbohydrate. and oxygen uptake will be measured at specific times over the next two to three hours. This procedure will be repeated on separate days for the two meals. For the control trials, the procedure will be repeated, except without exercising beforehand. Subjects will also be asked to fast overnight, beginning by 9:00 pm, prior to each trial, and to refrain from strenuous activity on the day before each trial. The meals will be given at the same time of day in each case. To estimate body fat content, measurements of skinfold thickness will be made with a Lang caliper at the following sites: triceps, suprailiac, subscapular, pectoral, umbilical, and front thigh. In addition, for each trial, three blood samples may be taken by finger prick for the purpose of glucose monitoring; one before, and at 60 and 120 minutes after ingestion of the meal.

3. **Risks and Benefits:** Certain normal changes will occur during the exercise. For example, heart rate, blood pressure, and ventilation will increase. These responses are to be expected, and are due to the exercise. In rare cases, abnormal responses are observed in apparently healthy individuals. These include abnormal very rare instances of heart attack. Every effort will be made to minimize the risks. If any of these were to happen, emergency equipment and trained personnel will be available on site to deal with the situation. The information obtained in this experiment will be used only for statistical research purposes and will be treated as privileged and confidential. Your identity will not be revealed in any description or publication of this research.

4. **Benefits:** The results of the subjects performance in this study will be given and explained. It will provide information about their cardiovascular endurance and body composition.

5. **Risk-Benefit Ratio:** The benefits of further understanding the effects of substrate utilization during exercise far outweigh the risks.

6. **Cost to Subjects:** There is no monetary cost to the subjects other than the investment of their time.

7. **Informed Consent:** Before beginning any of the measurements, the principle investigator will witness the reading and signing of the informed consent form. Once this document is signed, the forms will be permanently stored with the original data collected from this study and housed in the Exercise Physiology Department archives. At any point in time during the study, subjects will be encouraged to resolove any questions regarding the research or their participation. Their questions will be directed to Dr. Jack Young, who may be reached at 895-3766, or to Beth Palmer-Lynch, R.D. who can be reached at 456-9610.

Subject Statement of Consent:

I have read the above description of this research study, and I understand it. I have been informed of the risks and benefits involved, and all of my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered by a member of the research team. I understand that I will receive a copy of this form.

I understand that in the event injury occurs resulting from the research procedures, I will receive no monetary compensation. However, emergency equipment and trained personnel will be available to provide immediate emergency medical treatment on site, and any such treatment which may be necessary will be made available without charge. I understand that this paragraph is a statement of policy and does not waive any of my legal rights.

I understand that I am free to withdraw this consent and discontinue participation in this study at any time without prejudice.

I voluntarily consent to my participation in the described research study.

_____

Signature of Participant

ASSOCIATE VICE PRESIDENT FOR RESEARCH

UNIVERSITY OF NEVADA, LAS VEGAS
4505 MARYLAND PARKWAY • LAS VEGAS, NEVADA 89154-1002 • (702) 597-4240 • FAX (702) 597-4242

DATE: **November 20, 1992**

TO: **Cindy Beth Palmer-Lynch**

FROM: **Dr. Lawrence Golding, Chairman, Biomedical Subcommittee of the Institutional Review Board**

RE: **Approval of Human Subjects Protocol Project Entitled: "Effect of Prior Exercise on the Thermic Effect of Carbohydrate Versus Fat Feeding in Man"**

This memorandum is official notification that the protocol for the project referenced above was approved by the Bio/Medical Subcommittee of the Institutional Review Board on Friday, November 20, 1992.

If you have any questions or require any assistance, please give us a call.

APPENDIX II

Informed Consent

Title: The Effect of Prior Exercise on The Thermic Effect of Carbohydrate versus Fat Feeding in Man

Description of the Study: You are being asked to participate in a study to determine whether the increase in thermogenesis (heat produced by your body) after exercise is dependent on the type of food ingested. Prior to this study, your maximal oxygen uptake capacity will be determined by open circuit spirometry by pedalling a cycle ergometer at a constant speed and progressively increasing the workload until voluntary exhaustion. The experiment itself will consist of riding on the cycle ergometer at 70% of your maximal oxygen uptake capacity for 45 minutes. Prior to exercise, your resting oxygen uptake will be measured for a period of time sufficient to establish a baseline. Oxygen uptake will again be measured following the exercise to verify the return to the baseline condition. When the baseline has been reestablished, (approximately 1-2 hours after exercising), you will ingest one of the following 600 calorie meals: a) 100% carbohydrate, or b) 90% fat, 10% carbohydrate. and oxygen uptake will be measured at specific times over the next two to three hours. This procedure will be repeated on separate days for the two meals. For the control trials, the procedure will be repeated, except without exercising beforehand. You will also be asked to fast overnight, beginning by 9:00 pm, prior to each trial, and to refrain from strenuous activity on the day before each trial. The meals will be given at the same time of day in each case.

To estimate body fat content, measurements of skinfold thickness will be made with a Lang caliper at the following sites: triceps, suprailiac, subscapular, pectoral, umbilical, and front thigh.

In addition, for each trial, three blood samples may be taken by finger prick; one before, and at 60 and 120 minutes after ingestion of the meal.

Risks and Benefits: Certain normal changes will occur during the exercise. For example, your heart rate, blood pressure, and ventilation will increase. These responses are to be expected, and are due to the exercise. In rare cases, abnormal responses are observed in apparently healthy individuals. These include abnormal very rare instances of heart attack. Every effort will be made to minimize the risks. If any of these were to happen, emergency equipment and trained personnel will be available on site to deal with the situation.

The results of your performance in this study will be given and explained to you. It will provide you with information about your cardiovascular endurance and body composition.

Confidentiality: The information obtained in this experiment will be used only for statistical research purposes and will be treated as privileged and confidential. Your identity will not be revealed in any description or publication of this research.

Questions: If you have any questions regarding the research or your participation in it, either now or at any time in the future, please feel free to ask them. The research team, particularly Dr. Jack Young, who may be reached at 895-3766, or Beth Palmer-Lynch who can be reached at 456-9610, will be happy to answer any questions you may have.

Right to Withdraw: You are free to refuse to participate in this study, or to withdraw at any time. Your decision will not adversely affect your status at UNLV, or cause a loss of benefits to which you might otherwise be entitled.

Subject Statement of Consent:

I have read the above description of this research study, and I understand it. I have been informed of the risks and benefits involved, and all of my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered by a member of the research team. I understand that I will receive a copy of this form.

I understand that in the event injury occurs resulting from the research procedures, I will receive no monetary compensation. However, emergency equipment and trained personnel will be available to provide immediate emergency medical treatment on site, and any such treatment which may be necessary will be made available without charge. I understand that this paragraph is a statement of policy and does not waive any of my legal rights.

I understand that I am free to withdraw this consent and discontinue participation in this study at any time without prejudice.

I voluntarily consent to my participation in the described research study.

_____
Signature of Participant

_____
Date

APPENDIX III

Raw Data

Subject1							
CHO+rest	Baseline	Recovery	10 min.	30 min.	60 min.	90 min.	120 min.
VO2	0.192	0	0.199	0.194	0.198	0.197	0.203
RQ	0.82	0	0.85	0.9	0.87	0.86	0.86
Kcal	0.92	0	0.96	0.95	0.96	0.95	0.98
CHO+EX							
VO2	0.186	0.181	0.183	0.191	0.198	0.19	0.199
RQ	0.82	0.75	0.77	0.87	0.88	0.85	0.85
KCAL	0.89	0.86	0.87	0.93	0.96	0.92	0.96
FAT+rest							
VO2	0.193	0	0.199	0.208	0.195	0.2	0.205
RQ	0.73	0	0.65	0.66	0.74	0.78	0.76
KCAL	0.91	0	0.92	0.96	0.92	0.95	0.97
FAT+EX							
VO2	0.177	0.193	0.181	0.203	0.216	0.213	0.202
RQ	0.98	0.78	0.78	0.8	0.84	0.8	0.79
KCAL	0.88	0.92	0.86	0.97	1.04	1.02	0.96

Subject#2							
CHO+rest							
VO2	0.194	0	0.218	0.207	0.218	0.215	0.216
RQ	0.77	0	0.72	0.79	0.8	0.8	0.81
KCAL	0.92	0	1.02	0.99	1.04	1.03	1.03
CHO+EX							
VO2	0.241	0.222	0.253	0.253	0.269	0.26	0.245
RQ	0.75	0.74	0.75	0.78	0.8	0.81	0.82
KCAL	1.14	1.05	1.2	1.2	1.29	1.25	1.18
FAT+rest							
VO2	0.218	0	0.249	0.234	0.246	0.232	0.231
RQ	0.8	0	0.73	0.76	0.82	0.78	0.78
KCAL	1.04	0	1.17	1.11	1.18	1.1	1.1
FAT+EX							
VO2	0.223	0.216	0.243	0.243	0.252	0.226	0.222
RQ	0.78	0.76	0.78	0.81	0.86	0.82	0.78
KCAL	1.06	1.02	1.16	1.16	1.22	1.09	1.06

Subject#3							
CHO+rest	Baseline	Recovery	10 min.	30 min.	60 min.	90 min.	120 min.
VO2	0.185	0	0.223	0.209	0.191	0.176	0.198
RQ	0.66	0	0.72	0.76	0.8	0.78	0.83
KCAL	0.86	0	1.05	0.99	0.91	0.84	0.95
CHO+Ex							
VO2	0.167	0.16	0.208	0.21	0.206	0.191	0.194
RQ	0.69	0.68	0.73	0.79	0.77	0.8	0.83
KCAL	0.78	0.74	0.98	1	0.98	0.91	0.93
FAT+rest							
VO2	0.155	0	0.196	0.173	0.19	0.212	0.2
RQ	0.78	0	0.68	0.69	0.64	0.79	0.77
KCAL	0.74	0	0.91	0.81	0.87	1.01	0.95
FAT+Ex							
VO2	0.19	0.2	0.22	0.2	0.211	0.203	0.219
RQ	0.8	0.73	0.71	0.69	0.7	0.71	0.71
KCAL	0.91	0.94	1.03	0.93	0.99	0.95	1.03

Subject#4							
CHO+Rest							
VO2	0.202	0	0.218	0.221	0.232	0.241	0.223
RQ	0.81	0	0.76	0.8	0.89	0.87	0.9
KCAL	0.97	0	1.03	1.06	1.13	1.17	1.09
CHO+Ex							
VO2	0.2	0.218	0.244	0.249	0.267	0.238	0.244
RQ	0.82	0.71	0.7	0.7	0.77	0.81	0.8
KCAL	0.96	1.02	1.14	1.16	1.27	1.14	1.17
FAT+Rest							
VO2	0.194	0	0.251	0.222	0.231	0.217	0.205
RQ	0.73	0	0.71	0.74	0.74	0.75	0.73
KCAL	0.91	0	1.17	1.05	1.09	1.03	0.96
FAT+Ex							
VO2	0.203	0.202	0.24	0.249	0.251	0.247	0.238
RQ	0.83	0.72	0.72	0.78	0.83	0.76	0.74
KCAL	0.98	0.95	1.13	1.18	1.21	1.17	1.12

Subject#5							
CHO+Rest							
VO2	0.191	0	0.194	0.22	0.214	0.193	0.209
RQ	0.77	0	0.77	0.81	0.88	0.82	0.84
KCAL	0.91	0	0.92	1.05	1.04	0.93	1.01
CHO+Ex							
VO2	0.155	0.162	0.188	0.196	0.188	0.167	0.173
RQ	0.87	0.81	0.87	0.92	1	1.11	1.03
KCAL	0.75	0.78	0.91	0.96	0.94	0.86	0.87
FAT+Rest							
VO2	0.142	0	0.131	0.147	0.145	0.142	0.117
RQ	0.78	0	0.72	0.71	0.72	0.75	0.69
KCAL	0.68	0	0.61	0.69	0.68	0.67	0.55
FAT+Ex							
VO2	0.166	0.164	0.194	0.215	0.208	0.199	0.204
RQ	0.78	0.68	0.69	0.72	0.73	0.76	0.7
KCAL	0.79	0.76	0.9	1.01	0.98	0.94	0.95

Subject#6							
CHO+Rest							
VO2	0.201	0	0.211	0.214	0.208	0.205	0.219
RQ	0.84	0	0.79	0.82	0.85	0.93	0.89
KCAL	0.97	0	1.01	1.03	1.01	1.01	1.07
CHO+Ex							
VO2	0.191	0.131	0.22	0.226	0.239	0.213	0.211
RQ	0.82	0.68	0.74	0.78	0.93	0.81	0.81
KCAL	0.92	0.61	1.04	1.08	1.18	1.02	1.01
FAT+Rest							
VO2	0.181	0	0.199	0.21	0.214	0.218	0.19
RQ	0.83	0	0.76	0.78	0.81	0.79	0.83
KCAL	0.87	0	0.94	1	1.03	1.04	0.91
FAT+Ex							
VO2	0.184	0.198	0.205	0.227	0.154	0.145	0.151
RQ	0.79	0.71	0.68	0.69	0.7	0.75	0.72
KCAL	0.88	0.93	0.95	1.06	0.72	0.69	0.71

Subject#7							
CHO+Rest							
VO2	0.186	0	0.184	0.187	0.2	0.191	0.186
RQ	0.88	0	0.88	0.95	0.94	0.93	0.92
KCAL	0.91	0	0.9	0.92	0.99	0.94	0.91
CHO+Ex							
VO2	0.176	0.177	0.156	0.18	0.199	0.199	0.202
RQ	0.74	0.8	0.85	0.81	0.88	0.81	0.9
KCAL	0.83	0.85	0.75	0.86	0.97	0.95	0.99
FAT+Rest							
VO2	0.18	0	0.184	0.186	0.2	0.199	0.229
RQ	0.91	0	0.84	0.8	0.79	0.79	0.82
KCAL	0.88	0	0.89	0.89	0.95	0.95	1.1
FAT+Ex							
VO2	0.17	0.183	0.184	0.187	0.211	0.205	0.227
RQ	0.9	0.77	0.7	0.73	0.75	0.79	0.77
KCAL	0.83	0.87	0.86	0.88	1	0.98	1.08

Subject#8							
CHO+Rest							
VO2	0.283	0	0.289	0.281	0.298	0.297	0.291
RQ	0.87	0	0.82	0.86	0.87	0.88	0.87
KCAL	1.37	0	1.39	1.36	1.45	1.45	1.41
CHO+Ex							
VO2	0.237	0.242	0.263	0.302	0.275	0.258	0.258
RQ	0.78	0.59	0.67	0.69	0.72	0.74	0.69
KCAL	1.13	1.1	1.22	1.41	1.29	1.22	1.2
FAT+Rest							
VO2	0.253	0	0.279	0.291	0.278	0.281	0.267
RQ	0.75	0	0.69	0.67	0.7	0.68	0.68
KCAL	1.2	0	1.3	1.35	1.3	1.31	1.24
FAT+Ex							
VO2	0.233	0.243	0.247	0.271	0.266	0.264	0.274
RQ	0.83	0.68	0.67	0.68	0.67	0.7	0.66
KCAL	1.12	1.13	1.15	1.26	1.23	1.23	1.27

Subject#9							
CHO+Rest							
VO2	0.281	0	0.289	0.286	0.286	0.296	0.26
RQ	0.73	0	0.7	0.74	0.74	0.78	0.69
KCAL	1.32	0	1.35	1.36	1.35	1.41	1.21
CHO+Ex							
VO2	0.277	0.286	0.304	0.313	0.294	0.291	0.283
RQ	0.81	0.73	0.71	0.74	0.76	0.76	0.73
KCAL	1.33	1.35	1.42	1.48	1.39	1.38	1.33
FAT+Rest							
VO2	0.274	0	0.267	0.267	0.293	0.264	0.27
RQ	0.72	0	0.66	0.68	0.69	0.7	0.77
KCAL	1.29	0	1.24	1.24	1.37	1.23	1.28
FAT+Ex							
VO2	0.272	0.276	0.296	0.268	0.282	0.258	0.27
RQ	0.66	0.64	0.61	0.62	0.61	0.64	0.62
KCAL	1.26	1.27	1.35	1.23	1.29	1.19	1.24

Subject#10							
CHO+Rest							
VO2	0.201	0	0.283	0.28	0.283	0.261	0.215
RQ	0.73	0	0.764	0.815	0.783	0.83	0.886
KCAL	0.95	0	1.34	1.34	1.35	1.25	1.05
CHO+Ex							
VO2	0.203	0.196	0.256	0.244	0.272	0.266	0.244
RQ	0.971	0.921	1.03	1.16	1.13	1.08	1.71
KCAL	1	0.962	1.28	1.26	1.39	1.35	1.41
FAT+Rest							
VO2	0.208	0	0.248	0.252	0.268	0.257	0.274
RQ	0.977	0	0.958	0.994	1.04	0.997	0.972
KCAL	1.034	0	1.23	1.26	1.34	1.28	1.36
FAT+Ex							
VO2	0.147	0.142	0.146	0.158	0.243	0.233	0.208
RQ	1.04	0.89	1.05	1.03	1.07	0.973	0.936
KCAL	0.741	0.692	0.711	0.795	1.24	1.16	1.02

Subject#11							
CHO+Rest							
VO2	0.219	0	0.235	0.302	0.302	0.242	0.323
RQ	0.883	0	1.125	1.078	1.04	1.115	0.995
KCAL	1.067	0	1.207	1.536	1.52	1.24	1.61
CHO+Ex							
VO2	0.138	0.142	0.184	0.22	0.232	0.27	0.22
RQ	0.83	0.734	0.858	0.942	1	0.948	0.94
KCAL	0.664	0.668	0.891	1.083	1.16	1.334	1.085
FAT+Rest							
VO2	0.215	0	0.26	0.23	0.281	0.275	0.278
RQ	0.867	0	0.843	0.948	1.06	1.03	0.96
KCAL	1.04	0	1.255	1.136	1.423	1.384	1.377
FAT+Ex							
VO2	0.207	0.193	0.28	0.275	0.281	0.255	0.254
RQ	1	0.878	0.85	0.86	1.046	1.037	0.977
KCAL	1.035	0.939	1.354	1.332	1.419	1.285	1.263

Subject#12							
CHO+Rest							
VO2	0.199	0	0.241	0.255	0.255	0.278	0.258
RQ	0.747	0	0.873	0.878	0.871	0.872	0.94
KCAL	0.939	0	1.171	1.24	1.239	1.35	1.27
CHO+Ex							
VO2	0.182	0.208	0.265	0.278	0.297	0.27	0.248
RQ	0.816	0.686	0.738	0.824	0.828	0.823	0.84
KCAL	0.873	0.968	1.248	1.33	1.42	1.297	1.196
FAT+Rest							
VO2	0.183	0	0.19	0.186	0.206	0.218	0.214
RQ	0.765	0	0.775	0.798	0.83	0.854	0.788
KCAL	0.867	0	0.902	0.888	0.991	1.054	1.02
FAT+Ex							
VO2	0.195	0.186	0.212	0.2	0.214	0.17	0.167
RQ	0.758	0.705	0.732	0.766	0.786	0.886	0.905
KCAL	0.923	0.869	0.997	0.948	1.01	0.828	0.817

Subject#13							
CHO+Rest							
VO2	0.285	0	0.308	0.317	0.258	0.324	0.315
RQ	0.916	0	1.125	1.15	1.25	1.204	1.123
KCAL	1.39	0	1.582	1.63	1.37	1.69	1.617
CHO+Ex							
VO2	0.253	0.264	0.303	0.321	0.305	0.328	0.301
RQ	0.786	0.665	0.811	0.823	0.821	0.815	0.838
KCAL	1.205	1.22	1.45	1.54	1.46	1.57	1.45
FAT+Rest							
VO2	0.244	0	0.278	0.256	0.25	0.264	0.236
RQ	0.775	0	0.738	0.812	0.874	0.834	0.79
KCAL	1.159	0	1.309	1.22	1.215	1.271	1.125
FAT+Ex							
VO2	0.218	0.225	0.222	0.232	0.25	0.242	0.27
RQ	0.805	0.773	0.732	0.762	0.83	0.79	0.786
KCAL	1.04	1.06	1.04	1.09	1.2	1.15	1.28

Subject#14							
CHO+Rest							
VO2	0.23	0	0.283	0.25	0.212	0.312	0.276
RQ	0.769	0	0.923	1.02	0.966	0.898	0.933
KCAL	1.09	0	1.39	1.255	1.052	1.529	1.35
CHO+Ex							
VO2	0.208	0.208	0.306	0.281	0.273	0.206	0.295
RQ	0.776	0.654	0.812	0.843	0.813	0.85	0.76
KCAL	0.988	0.96	1.466	1.356	1.308	0.85	1.397
FAT+Rest							
VO2	0.218	0	0.261	0.253	0.261	0.26	0.295
RQ	0.883	0	0.906	0.926	0.988	0.971	0.895
KCAL	1.061	0	1.278	1.244	1.301	1.291	1.44
FAT+Ex							
VO2	0.144	0.195	0.275	0.248	0.225	0.245	0.248
RQ	1.15	0.921	0.935	0.928	0.98	1.03	0.975
KCAL	0.744	0.958	1.355	1.22	1.12	1.233	1.233

Subject#15							
CHO+Rest							
VO2	0.185	0	0.236	0.262	0.242	0.298	0.28
RQ	1.05	0	1.121	1.208	1.22	1.146	1.11
KCAL	0.935	0	1.21	1.36	1.26	1.53	1.43
CHO+Ex							
VO2	0.228	0.231	0.308	0.306	0.316	0.343	0.301
RQ	0.98	0.9	0.953	1.02	1.03	1.04	1.12
KCAL	1.135	1.129	1.524	1.536	1.59	1.73	1.54
FAT+Rest							
VO2	0.226	0	0.261	0.148	0.258	0.236	0.258
RQ	0.901	0	0.858	0.881	0.99	0.973	0.905
KCAL	1.1	0	1.26	1.44	1.287	1.172	1.26
FAT+Ex							
VO2	0.18	0.217	0.258	0.272	0.294	0.278	0.208
RQ	0.108	0.955	0.893	0.944	1.02	1	0.986
KCAL	0.917	1.074	1.259	1.343	1.477	1.16	1.03

APPENDIX IV

Difference Data

Subject1	10 min.	30 min.	60 min.	90 min.	120 min.
CHO+rest					
VO2	0.007	0.002	0.006	0.005	0.011
RQ	0.03	0.08	0.05	0.04	0.04
Kcal	0.04	0.03	0.04	0.03	0.06
CHO+EX					
VO2	0.002	0.01	0.017	0.009	0.018
RQ	0.02	0.12	0.13	0.1	0.1
KCAL	0.01	0.07	0.1	0.06	0.1
FAT+rest					
VO2	0.006	0.015	0.002	0.007	0.012
RQ	-0.08	-0.07	0.01	0.05	0.03
KCAL	0.01	0.05	0.01	0.04	0.06
FAT+EX					
VO2	-0.012	0.01	0.023	0.02	0.009
RQ	0	0.02	0.06	0.02	0.01
KCAL	-0.06	0.05	0.12	0.1	0.04

Subject#2					
CHO+rest					
VO2	0.024	0.013	0.024	0.021	0.022
RQ	-0.05	0.02	0.03	0.03	0.04
KCAL	0.1	0.07	0.12	0.11	0.11
CHO+EX					
VO2	0.031	0.031	0.047	0.038	0.023
RQ	0.01	0.04	0.06	0.07	0.08
KCAL	0.15	0.15	0.24	0.2	0.13
FAT+rest					
VO2	0.031	0.016	0.028	0.014	0.013
RQ	-0.07	-0.04	0.02	-0.02	-0.02
KCAL	0.13	0.07	0.14	0.06	0.06
FAT+EX					
VO2	0.027	0.027	0.036	0.01	0.006
RQ	0.02	0.05	0.1	0.06	0.02
KCAL	0.14	0.14	0.2	0.07	0.04

Subject#3					
CHO+rest					
VO2	0.038	0.024	0.006	-0.009	0.013
RQ	0.06	0.1	0.14	0.12	0.17
KCAL	0.19	0.13	0.05	-0.02	0.09
CHO+Ex					
VO2	0.048	0.05	0.046	0.031	0.034
RQ	0.05	0.11	0.09	0.12	0.15
KCAL	0.24	0.26	0.24	0.17	0.19
FAT+rest					
VO2	0.041	0.018	0.035	0.057	0.045
RQ	-0.1	-0.09	-0.14	0.01	-0.01
KCAL	0.17	0.07	0.13	0.27	0.21
FAT+Ex					
VO2	0.02	0	0.011	0.003	0.019
RQ	-0.02	-0.04	-0.03	-0.02	-0.02
KCAL	0.09	-0.01	0.05	0.01	0.09

Subject#4					
CHO+Rest					
VO2	0.016	0.019	0.03	0.039	0.021
RQ	-0.05	-0.01	0.08	0.06	0.09
KCAL	0.06	0.09	0.16	0.2	0.12
CHO+Ex					
VO2	0.026	0.031	0.049	0.02	0.026
RQ	-0.01	-0.01	0.06	0.1	0.09
KCAL	0.12	0.14	0.25	0.12	0.15
FAT+Rest					
VO2	0.057	0.028	0.037	0.023	0.011
RQ	-0.02	0.01	0.01	0.02	0
KCAL	0.26	0.14	0.18	0.12	0.05
FAT+Ex					
VO2	0.038	0.047	0.049	0.045	0.036
RQ	0	0.06	0.11	0.04	0.02
KCAL	0.18	0.23	0.26	0.22	0.17

Subject#5					
CHO+Rest					
VO2	0.003	0.029	0.023	0.002	0.018
RQ	0	0.04	0.11	0.05	0.07
KCAL	0.01	0.14	0.13	0.02	0.1
CHO+Ex					
VO2	0.026	0.034	0.026	0.005	0.011
RQ	0.06	0.11	0.19	0.3	0.22
KCAL	0.13	0.18	0.16	0.08	0.09
FAT+Rest					
VO2	-0.011	0.005	0.003	0	-0.025
RQ	-0.06	-0.07	-0.06	-0.03	-0.09
KCAL	-0.07	0.01	0	-0.01	-0.13
FAT+Ex					
VO2	0.03	0.051	0.044	0.035	0.04
RQ	0.01	0.04	0.05	0.08	0.02
KCAL	0.14	0.25	0.22	0.18	0.19

Subject#6					
CHO+Rest					
VO2	0.01	0.013	0.007	0.004	0.018
RQ	-0.05	-0.02	0.01	0.09	0.05
KCAL	0.04	0.06	0.04	0.04	0.1
CHO+Ex					
VO2	0.089	0.095	0.108	0.082	0.08
RQ	0.06	0.1	0.25	0.13	0.13
KCAL	0.43	0.47	0.57	0.41	0.4
FAT+Rest					
VO2	0.018	0.029	0.033	0.037	0.009
RQ	-0.07	-0.05	-0.02	-0.04	0
KCAL	0.07	0.13	0.16	0.17	0.04
FAT+Ex					
VO2	0.007	0.029	-0.044	-0.053	-0.047
RQ	-0.03	-0.02	-0.01	0.04	0.01
KCAL	0.02	0.13	-0.21	-0.24	-0.22

Subject#7					
CHO+Rest					
VO2	-0.002	0.001	0.014	0.005	0
RQ	0	0.07	0.06	0.05	0.04
KCAL	-0.01	0.01	0.08	0.03	0
CHO+Ex					
VO2	-0.021	0.003	0.022	0.022	0.025
RQ	0.05	0.01	0.08	0.01	0.1
KCAL	-0.1	0.01	0.12	0.1	0.14
FAT+Rest					
VO2	0.004	0.006	0.02	0.019	0.049
RQ	-0.07	-0.11	-0.12	-0.12	-0.09
KCAL	0.01	0.01	0.07	0.07	0.22
FAT+Ex					
VO2	0.001	0.004	0.028	0.022	0.044
RQ	-0.07	-0.04	-0.02	0.02	0
KCAL	-0.01	0.01	0.13	0.11	0.21

Subject#8					
CHO+Rest					
VO2	0.006	-0.002	0.015	0.014	0.008
RQ	-0.05	-0.01	0	0.01	0
KCAL	0.02	-0.01	0.08	0.08	0.04
CHO+Ex					
VO2	0.021	0.06	0.033	0.016	0.016
RQ	0.08	0.1	0.13	0.15	0.1
KCAL	0.12	0.31	0.19	0.12	0.1
FAT+Rest					
VO2	0.026	0.038	0.025	0.028	0.014
RQ	-0.06	-0.08	-0.05	-0.07	-0.07
KCAL	0.1	0.15	0.1	0.11	0.04
FAT+Ex					
VO2	0.004	0.028	0.023	0.021	0.031
RQ	-0.01	0	-0.01	0.02	-0.02
KCAL	0.02	0.13	0.1	0.1	0.14

Subject#9					
CHO+Rest					
VO2	0.008	0.005	0.005	0.015	-0.021
RQ	-0.03	0.01	0.01	0.05	-0.04
KCAL	0.03	0.04	0.03	0.09	-0.11
CHO+Ex					
VO2	0.018	0.027	0.008	0.005	-0.003
RQ	-0.02	0.01	0.03	0.03	0
KCAL	0.07	0.13	0.04	0.03	-0.02
FAT+Rest					
VO2	-0.007	-0.007	0.019	-0.01	-0.004
RQ	-0.06	-0.04	-0.03	-0.02	0.05
KCAL	-0.05	-0.05	0.08	-0.06	-0.01
FAT+Ex					
VO2	0.02	-0.008	0.006	-0.018	-0.006
RQ	-0.03	-0.02	-0.03	0	-0.02
KCAL	0.08	-0.04	0.02	-0.08	-0.03

Subject#10					
CHO+Rest					
VO2	0.082	0.079	0.082	0.06	0.014
RQ	0.034	0.085	0.053	0.1	0.156
KCAL	0.39	0.39	0.4	0.3	0.1
CHO+Ex					
VO2	0.06	0.048	0.076	0.07	0.048
RQ	0.109	0.239	0.209	0.159	0.789
KCAL	0.318	0.298	0.428	0.388	0.448
FAT+Rest					
VO2	0.04	0.044	0.06	0.049	0.066
RQ	-0.019	0.017	0.063	0.02	-0.005
KCAL	0.196	0.226	0.306	0.246	0.326
FAT+Ex					
VO2	0.004	0.016	0.101	0.091	0.066
RQ	0.16	0.14	0.18	0.083	0.046
KCAL	0.019	0.103	0.548	0.468	0.328

Subject#11					
CHO+Rest					
VO2	0.016	0.083	0.083	0.023	0.104
RQ	0.242	0.195	0.157	0.232	0.112
KCAL	0.14	0.469	0.453	0.173	0.543
CHO+Ex					
VO2	0.042	0.078	0.09	0.128	0.078
RQ	0.124	0.208	0.266	0.214	0.206
KCAL	0.223	0.415	0.492	0.666	0.417
FAT+Rest					
VO2	0.045	0.015	0.066	0.06	0.063
RQ	-0.024	0.081	0.193	0.163	0.093
KCAL	0.215	0.096	0.383	0.344	0.337
FAT+Ex					
VO2	0.087	0.082	0.088	0.062	0.061
RQ	-0.028	-0.018	0.168	0.159	0.099
KCAL	0.415	0.393	0.48	0.346	0.324

Subject#12					
CHO+Rest					
VO2	0.042	0.056	0.056	0.079	0.059
RQ	0.126	0.131	0.124	0.125	0.193
KCAL	0.232	0.301	0.3	0.411	0.331
CHO+Ex					
VO2	0.057	0.07	0.089	0.062	0.04
RQ	0.052	0.138	0.142	0.137	0.154
KCAL	0.28	0.362	0.452	0.329	0.228
FAT+Rest					
VO2	0.007	0.003	0.023	0.035	0.031
RQ	0.01	0.033	0.065	0.089	0.023
KCAL	0.035	0.021	0.124	0.187	0.153
FAT+Ex					
VO2	0.026	0.014	0.028	-0.016	-0.019
RQ	0.027	0.061	0.081	0.181	0.2
KCAL	0.128	0.079	0.141	-0.041	-0.052

Subject#13					
CHO+Rest					
VO2	0.023	0.032	-0.027	0.039	0.03
RQ	0.209	0.234	0.334	0.288	0.207
KCAL	0.192	0.24	-0.02	0.3	0.227
CHO+Ex					
VO2	0.039	0.057	0.041	0.064	0.037
RQ	0.146	0.158	0.156	0.15	0.173
KCAL	0.23	0.32	0.24	0.35	0.23
FAT+Rest					
VO2	0.034	0.012	0.006	0.02	-0.008
RQ	-0.037	0.037	0.099	0.059	0.015
KCAL	0.15	0.061	0.056	0.112	-0.034
FAT+Ex					
VO2	-0.003	0.007	0.025	0.017	0.045
RQ	-0.041	-0.011	0.057	0.017	0.013
KCAL	-0.02	0.03	0.14	0.09	0.22

Subject#14					
CHO+Rest					
VO2	0.053	0.02	-0.018	0.082	0.046
RQ	0.154	0.251	0.197	0.129	0.164
KCAL	0.3	0.165	-0.038	0.439	0.26
CHO+Ex					
VO2	0.098	0.073	0.065	-0.002	0.087
RQ	0.158	0.189	0.159	0.196	0.106
KCAL	0.506	0.396	0.348	-0.11	0.437
FAT+Rest					
VO2	0.043	0.035	0.043	0.042	0.077
RQ	0.023	0.043	0.105	0.088	0.012
KCAL	0.217	0.183	0.24	0.23	0.379
FAT+Ex					
VO2	0.08	0.053	0.03	0.05	0.053
RQ	0.014	0.007	0.059	0.109	0.054
KCAL	0.397	0.262	0.162	0.275	0.275

Subject#15					
CHO+Rest					
VO2	0.051	0.077	0.057	0.113	0.095
RQ	0.071	0.158	0.17	0.096	0.06
KCAL	0.275	0.425	0.325	0.595	0.495
CHO+Ex					
VO2	0.077	0.075	0.085	0.112	0.07
RQ	0.053	0.12	0.13	0.14	0.22
KCAL	0.395	0.407	0.461	0.601	0.411
FAT+Rest					
VO2	0.035	-0.078	0.032	0.01	0.032
RQ	-0.043	-0.02	0.089	0.072	0.004
KCAL	0.16	0.34	0.187	0.072	0.16
FAT+Ex					
VO2	0.041	0.055	0.077	0.061	-0.009
RQ	-0.062	-0.011	0.065	0.045	0.031
KCAL	0.185	0.269	0.403	0.086	-0.044

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