A NEW ENERGY CONCEPT

FOR WEIGHT REGULATION

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ABSTRACT

In this thesis a new energy concept (—ets, or *equivalent teaspoon sugar*) is further exploited to develop a complete energy equation called —ets CalFibre to control obesity. This new, easy to understand and visualise energy concept (—ets) can more accurately predict the insulin response (R^2=0.929) to ingested carbohydrates than the Glycaemic Index (GI).

Insulin promotes fat storage and prevents fat burning that causes obesity. —ets intake can regulate obesity by controlling the insulin response of certain foods. A complete energy equation can now be constructed by adding—ets, protein and fat energy values together.

$$\text{—ets CalFibre} = 13[\text{kCal/—ets}] \times \text{—ets}_{CHO} + 9[\text{kCal/g}] \times \text{Mass}_{Fat}[\text{g}] + 4[\text{kCal/g}] \times \text{Mass}_{Protein}[\text{g}]$$

More than one billion adults are overweight and at least 300 million are clinically obese. The economical impact is far reaching with losses of $117 billion in the USA alone. The relative risk factor for developing diabetes increases 40 fold with a Body Mass Index (BMI) of 35. Analysis indicates that there exists a direct correlation between carbohydrate (CHO) intake and obesity.

A Spraque-Dawley rat protocol was designed with an energy restricted diet, to test weight variations against a predetermined —ets intake. A linear relationship was found between the —ets CalFibre values of a food containing CHO and the % mass loss, with a resulting Pearson's R^2 value of 0.69. This shows that the —ets CalFibre equation is more representative of the energy conversion of CHO in a body than the constant 4kCal/g historically used.

By establishing the —ets CalFibre energy equation, the total amount of metabolizable energy from carbohydrates can now be calculated. Now that doctors and dieticians know the exact amount of energy that their patients consume, they can better regulate their weight by prescribing the right amount of —ets. This capability brings scientists and physiologists one step closure to a total obesity solution.

SAMEVATTING

In hierdie verhandeling word 'n nuwe energie konsep (_ets, of ekwivalente teelepels suiker) vêrder afgelei, sodat 'n volledige energievergelyking, genaamd _ets CalFibre, verkry kan word. Hierdie vergelyking kan gebruik word, om obesitiet te beheer. Hierdie nuwe, eenvoudig, verstaanbare energie konsep gee 'n beter aanduiding van insulien se reaksie teenoor die inname van koölhidrate (R^2=0.929), as die Glisemiese Indeks (GI).

Insulien bevorder die stoor van vet en is direk aanspreeklik vir obesiteit. _ets inname kan vetsug reguleer omrede daar beter beheer oor die insulien reaksie van sekere kosse toegepas kan word. Deur _ets, proteïne en vet energiewaardes bymekaar te voeg, kan 'n nuwe energievergelyking saamgestel word.

$$\text{-\_ets CalFibre} = 13[\text{kCal/\_ets}] \times \text{\_ets}_{CHO} + 9[\text{kCal/g}] \times \text{Mass}_{Fat}[\text{g}] + 4[\text{kCal/g}] \times \text{Mass}_{Protein}[\text{g}]$$

Meer as een biljoen van vandag se volwassenes is oorgewig en ten minste 300 miljoen is klinies oorgewig. Die ekonomiese impak strek wyd, met verliese van soveel as $117 biljoen in die VSA alleen. Die relatiewe risikofaktor vir die ontwikkeling van diabetes verhoog 40 voudig met 'n Liggaams Gewig Indeks (LGI) van 35. Navorsing toon dat daar 'n direkte korrelasie bestaan tussen die inname van koölhidrate(CHO) en obesiteit.

'n Spraque-Dawley rot protokol was ontwerp met 'n energiebeperktedieët, sodat gewig variasies getoets kan word, teenoor 'n voorafbepaalde _ets inname. 'n Lineêre verwantskap word verkry tussen die _ets CalFibre waardes van die voedselsoorte wat CHO bevat en die persentasie massaverlies met 'n resultante Pearson's R^2 waarde van 0.69. Dit bewys dat die _ets CalFibre vergelyking meer verteenwoordigend is van die energie omskakeling van CHO in 'n liggaam, as die konstante 4kCal/g wat histories gebruik was.

Deur die _ets CalFibre energievergelyking te bepaal, kan die totale hoeveelheid metaboliseerbare energie vanaf die koölhidrate bereken word. Nou, deurdat dokters en dieëtkundiges kennis dra van die presiese hoeveelheid energie wat hul pasiënte verbruik, kan

hulle die pasiënte se gewig beter beheer, deur die regte hoeveelheid ~~ets~~ voor te skryf. Hierdie moontlikheid bring wetenskaplikes een stap nader aan 'n totale obesiteits oplossing.

ACKNOWLEDGEMENTS

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I would also like to thank Dr. Arnold van Dyk for allowing me to use his graphical description of the metabolic pathways in Chapter 3.

I would also like to thank Dr. Cor Botha who assisted Prof E. H. Mathews with the development and verification process of *ets* in Chapter 4 that formed the basis of further development.

I would also like to thank Human-Sim (Pty) Ltd. for their financial support during this study.

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NOMENCLATURE

Glossary

Amino acids	an organic acid in which one of the hydrogen atoms on a carbon atom has been replaced by NH_2. Usually refers to an aminocarboxylic acid. However, taurine is also an amino acid.
Anabolism	**1.** the building up in the body of complex chemical compounds from simpler compounds (e.g., proteins from amino acids), usually with the use of energy. *Cf.* catabolism, metabolism. **2.** the sum of synthetic metabolic reactions.
Anthropology	the scientific study of human beings with respect to physical features, classification, distribution, and social and cultural relationships.
Bariatrics	that branch of medicine concerned with the management of obesity.
Carbohydrates	class name for the aldehydic or ketonic derivatives of polyhydric alcohols. Most such compounds have formulas that may be written $C_n(H_2O)_n$, although they are not true hydrates. The group includes simple sugars (monosaccharides, disaccharides, etc.), as well as macromolecular (polymeric) substances such as starch, glycogen, and cellulose polysaccharides.
Catabolism	**1.** the breaking down in the body of complex chemical compounds into simpler ones, often accompanied by the liberation of energy. **2.** the sum of all degradative processes.
Co-morbidities	a concomitant but unrelated pathologic or disease process; usually used In epidemiology to indicate the coexistence of two or more disease processes.

Epidemiology	the study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to control of health problems.
Etiology	**1.** the science and study of the causes of disease and their mode of operation. *Cf.* pathogenesis. **2.** the science of causes, causality; in common usage, cause.
Fat	**1.** Syn: adipose tissue. **2.** common term for obese. **3.** a greasy, soft-solid material, found in animal tissues and many plants, composed of a mixture of glycerol esters; together with oils they make up the homolipids. **4.** a triacylglycerol or a mixture of triacylglycerols.
Fatty acids	any acid derived from fats by hydrolysis (e.g., oleic, palmitic, or stearic acids); any long-chain monobasic organic acid; they accumulate in disorders associated with the peroxisomes.
Glucagon	a hormone produced by pancreatic alpha cells. Parenteral administration of 0.5 to 1 mg results in prompt mobilization of hepatic glycogen, thus elevating blood glucose concentration. It is used in the treatment of glycogen storage disease (von Gierke's) and hypoglycemia, particularly hypoglycemic coma due to exogenously administered insulin.
Glyceamic Index	any of various measures of the rise in blood glucose level after ingestion of carbohydrate.
Glucogenesis	formation of glucose
Gluconeogenisis	the formation of glucose from noncarbohydrates, such as protein or fat. *Cf.* glyconeogenesis

Glucose	a dextrorotatory monosaccharide found in the free form in fruits and other parts of plants, and in combination in glucosides, glycogen, disaccharides, and polysaccharides (starch cellulose); the chief source of energy in human metabolism, the final product of carbohydrate digestion, and the principal sugar of the blood; insulin is required for the use of glucose by cells; in diabetes mellitus the level of glucose in the blood is excessive, and it also appears in the urine.
Insulin	a polypeptide hormone, secreted by beta cells in the islets of Langerhans, that promotes glucose utilization, protein synthesis, and the formation and storage of neutral lipids; available in a variety of preparations including genetically engineered human insulin, which is presently favored, insulin is used parenterally in the treatment of diabetes mellitus.
Ketones	a substance with the carbonyl group linking two carbon atoms; the most important in medicine and the simplest in chemistry is dimethyl ketone (acetone).
Leptin	a helical protein secreted by adipose tissue and acting on a receptor site in the ventromedial nucleus of the hypothalamus to curb appetite and increase energy expenditure as body fat stores increase. Leptin levels are 40% higher in women, and show a further 50% rise just before menarche, later returning to baseline levels; levels are lowered by fasting and increased by inflammation.
Monosaccharide	a carbohydrate that cannot form any simpler sugar by simple hydrolysis; e.g., pentoses, hexoses.
Oligosaccharides	a compound made up of the condensation of a small number of monosaccharide units.
Pathogonomic	characteristic or indicative of a disease; denoting especially one or more typical symptoms, findings, or pattern of abnormalities specific for a given disease and not found in any other condition.

Pharmacotherapy	treatment of disease by means of drugs
Protein	macromolecules consisting of long sequences of)-amino acids [H_2N–CHR–COOH] in peptide (amide) linkage (elimination of H_2O between the)-NH_2 and)-COOH of successive residues). Protein is three-fourths of the dry weight of most cell matter and is involved in structures, hormones, enzymes, muscle contraction, immunological response, and essential life functions. The amino acids involved are generally the 20)-amino acids (glycine, L-alanine, etc.) recognized by the genetic code. Cross-links yielding globular forms of protein are often effected through the –SH
Triacylglycerol	glycerol esterified at each of its three hydroxyl groups by a fatty (aliphatic) acid.
Thermodynamics	**1.** the branch of physicochemical science concerned with heat and energy and their conversions one into the other involving mechanical work. **2.** the study of the flow of heat.
Triglycerides	glycerol esterified at each of its three hydroxyl groups by a fatty (aliphatic) acid.groups of two sulfur-containing L-cysteinyl residues, as well as by noncovalent forces (hydrogen bonds, lipophilic attractions, etc.).

Abbreviations

ANOVA	Analysis of Variance
AUC	Area Under the Curve
BMI	Body Mass Index
CDC	Centre for Disease Control
CHO	Carbohydrate(s)
CVD	Cardiovascular Disease
ETS	Equivalent Teaspoons Sugar (Branded as –*ets*)

GI	Glycaemic Index
GL	Glycaemic Load
HDL	High Density Lipoprotein
II	Insulin Index
LCL	Lower Control Level
NAFLD	Non-alcoholic fatty liver disease
NHANES	National Heath and Nutrition Examination Survey
NSP	Non-starch polycaccireds
UCL	Upper Control Level
UPBRC	University of Pretoria Biomedical Research Centre
RDA	Recommended Daily Allowance
WHO	World Health Organisation

Symbols

AUC_{BS}	Area under the curve of blood sugar response.
AUC_{Food}	Area under the curve of the food being tested.
$AUC_{Ingested}$	Area of the blood glucose response curve of ingested glucose.
$AUC_{Reference}$	Area under the curve of the reference food in the test.
ΔBS_{Rise}	Absolute rise in blood sugar concentration due to an ingested meal.
ΔBS_{Fall}	Absolute drop in blood sugar concentration due to injected (or secreted) insulin.
$BI(t)$	Blood insulin response.
$BS(t)$	Blood sugar response.
$BS_{Blood(t)}$	Blood sugar concentration at a specific time.
E_{CHO}	Converted carbohydrate energy potential.
E_{ets}	Total amount of blood glucose energy available from ingested ets.
$E_{teaspoon\ \ sugar}$	Energy available from a teaspoon of sugar.
ets	Equivalent teaspoons sugar.
f_{AUCI}	Insulin response area / ets relationship efficiency factor.

f_{CHO}	Efficiency factor for converting ingested carbohydrates into blood sugar energy.
K	Blood sugar / ets conversion factor.
k_{CHO}	Maximum amount of energy available from carbohydrates.
m_{CHO}	Mass of carbohydrates contained in the food.
$m_{teaspoon\ sugar}$	Mass of carbohydrates contained in a teaspoon of sugar.
t	Time.
W	Weight.

Units

ets	Equivalent Teaspoons Sugar
g	Grams
kCal	Kilocalories
kg	Kilograms
l	Litre
min	Minutes
mmol	Milli-mol
unit(s)	Insulin units
W	Watt

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1 INTRODUCTION

1.1 Background to Western diseases

Scientists predict that for the first time, the current generation will have a life expectancy less than the previous generation. The main adverse consequences are cardiovascular disease, type 2 diabetes, cancer, coronary artery diseases and hypertension. One common physiological factor that is present with all of these diseases is excess body weight. Obesity is the sixth most important risk factor contributing to the overall burden of disease worldwide.

The World Health Organisation (WHO) describes obesity as one of the most blatantly visible, yet most neglected public-health problems that threaten to overwhelm both more and less developed countries [1]. Obesity has achieved global recognition only during the past 10 years, in contrast to underweight, malnutrition, and infectious diseases, which have always dominated thinking.

Figure 1 shows the average regional prevalence of obesity by age and sex in the sub regions of the world. These estimates, based on measured BMI (Body Mass Index) in appropriate population samples, show that the only region in which obesity is not common is sub-Saharan Africa as a whole. However, the prevalence in South Africa is high, especially among the poorest women, and reflects the general worldwide finding that obesity is linked to poverty, [2] particularly when a country's GDP (Gross Domestic Product) exceeds about US$5000 per year. [3], [4]

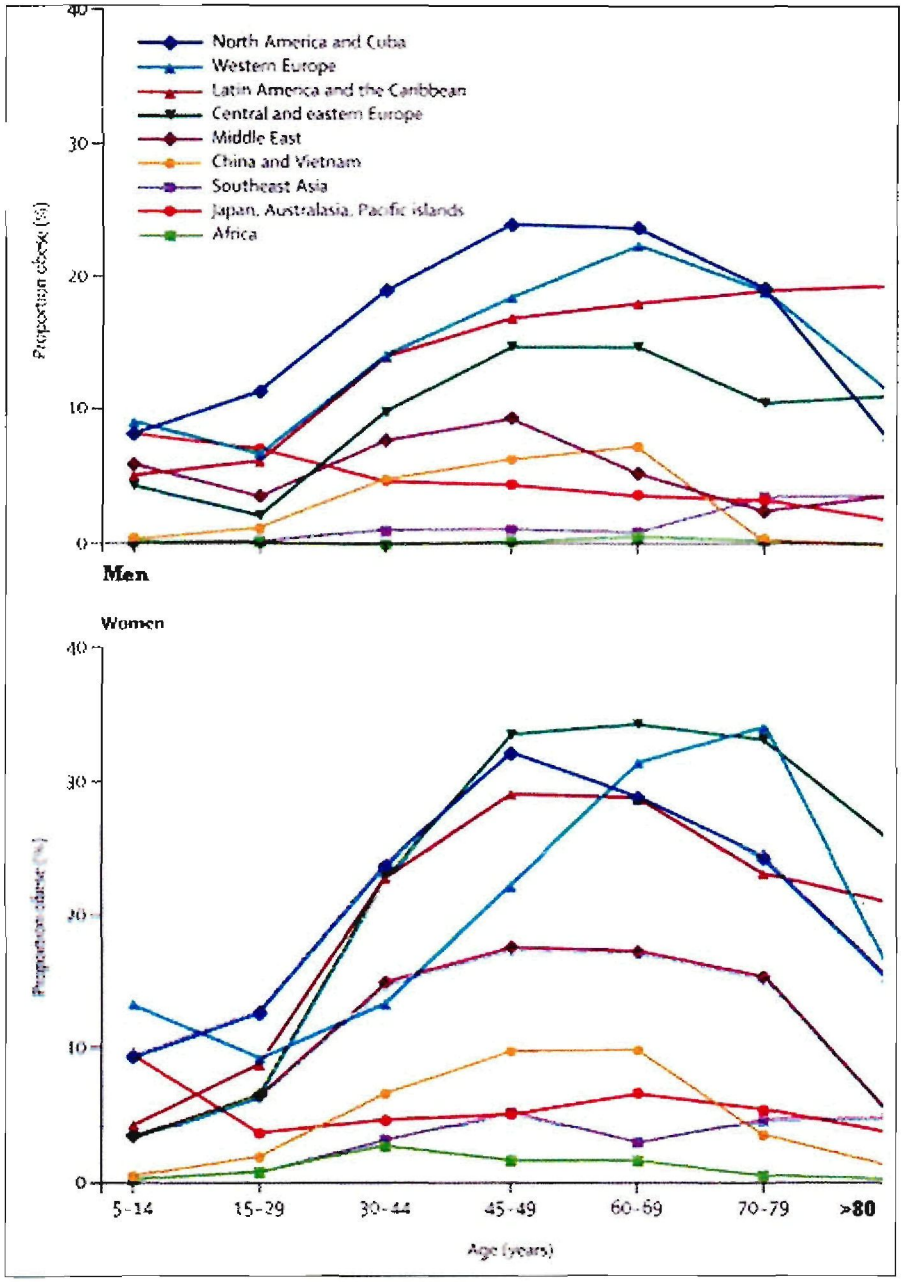

Figure 1: The average regional prevalence of obesity

Detailed estimates of the years of ill health and lives lost between the ages of 30 years and 75 years because of excess weight are shown for the sub regions of the world in ***Figure 2***. These predictions are based on detailed estimates of the prevalence of various disorders and deaths from them, the prevalence of high BMI according to age, and the proportion of the disease burden attributable to the excess weight. [3] Cardiovascular disease dominates, followed by diabetes and some cancers, especially in women. Again, the burden of disease is high in Eastern Europe and Latin America, but the Asian countries have a surprisingly high burden in view of

their lower obesity rates. This finding relates to the higher absolute risk of diabetes and probably cardiovascular disease among Asian, [3], [5] Hispanic, [6] and perhaps African populations, partly because they are more prone to abdominal obesity with its excess risks.

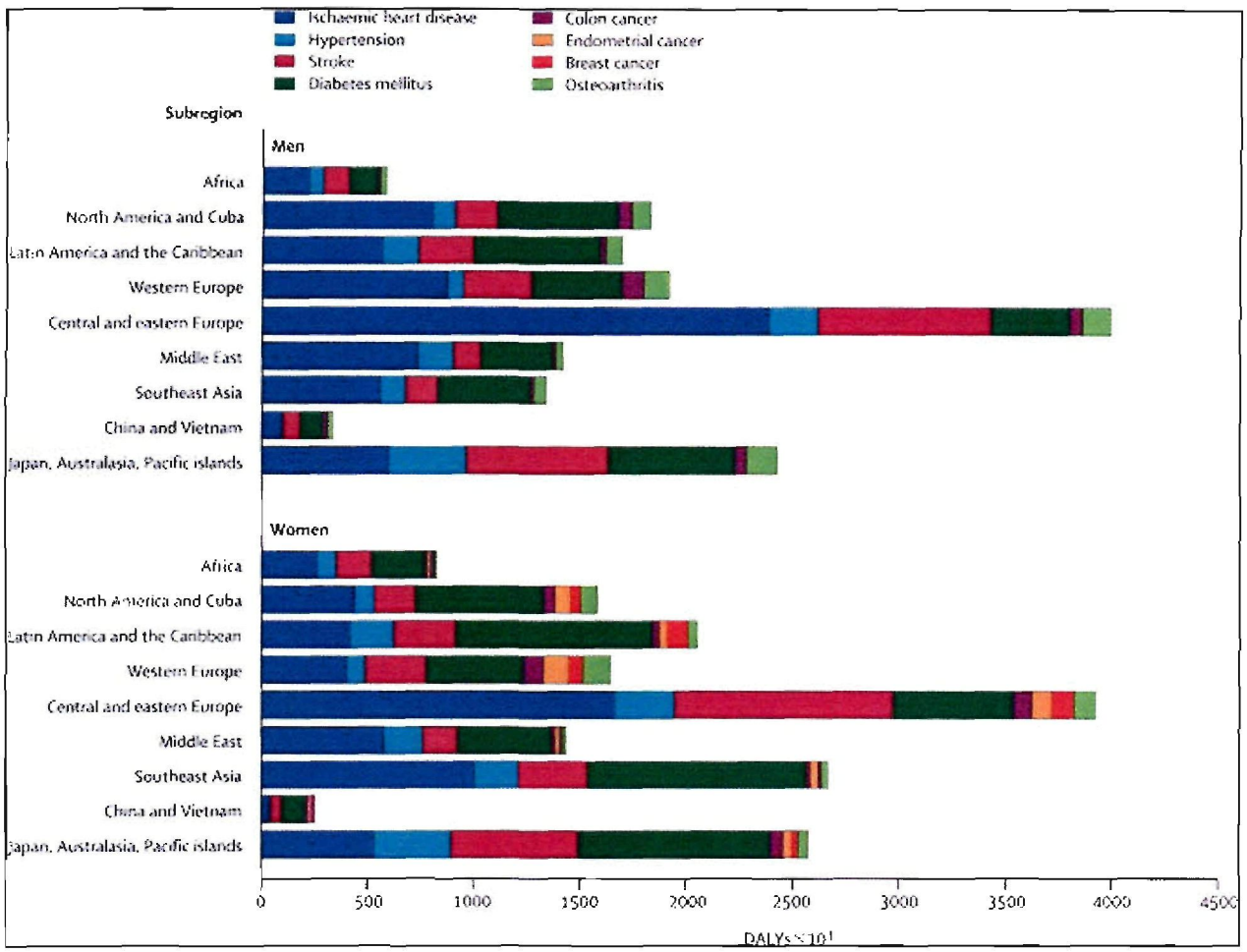

Figure 2: Disability-adjusted life-years (DALYs) lost as a result of obesity in men and women worldwide

The complex pathological processes reflect environmental and genetic interactions, and individuals from disadvantaged communities seem to have greater risks than more affluent individuals partly because of foetal and postnatal imprinting. Obesity, with its array of co-morbidities, necessitates careful clinical assessment to identify underlying factors and to allow coherent management.

The epidemic reflects progressive secular and age-related decreases in physical activity, together with substantial dietary changes with passive over-consumption of energy despite the neurobiological processes controlling food intake. Effective long-term weight loss depends on permanent changes in dietary quality, energy intake, and activity. Neither the medical management, nor the societal preventive challenges, are currently being met.

1.2 Problem statement

Currently there does not exist a metabolic conversion efficiency (η) for different carbohydrates in the human body. The objective of this study is therefore to *determine a metabolic conversion factor and incorporate it into an energy equation that includes all macronutrient energy.*

The study team hypothesises that blood sugar response (GI) from ingested carbohydrates could be used as an indication of the available *metabolically* energy from ingested carbohydrates. The secondary objective is to express this conversion efficiency as an easily understandable and quantifiable energy unit - *ets* (Equivalent Teaspoon Sugar)

Does *ets* intake correlate with weight variation and can this correlation be successfully tested with clinical trials on Spraque-Dawley rats? If this correlation is verifiable, then the study team has accomplished its goal of quantifying the human beings' energy homeostasis equation that could regulate obesity.

1.3 Methodology

- Firstly, a study of the general obesity problem was done, that specifically focused on the medical consequences and economical impact on society, justifying further research.
- Then the human energy model, as proposed by Mathews, was investigated as a possible solution in quantifying *metabolizable* energy correctly.
- The new energy concept - *ets* was derived and incorporated into a new energy equation called *ets* Cal.

- To verify the —ets Cal equation, foods with different —ets values were tested in a protocol designed for Spraque-Dawley rats.
- These clinical trial data were biostatically analyses and presented.
- A comparison analysis was done and it was concluded that —ets CalFibre (—ets Cal that includes fibre) is a better method to determine metabolizable energy.

1.4 Overview

The problem statement, and methodology that has been discussed above, is now presented in a thorough study to seek an answer to the obesity epidemic.

The literature study of Chapter 2 outlines the true nature of the obesity problem. Obesity is classified and the statistics are presented so that it is possible to grasp the relevance of this study. This chapter also includes the medical and economical impact of obesity and identifies how the human diet has changed over the last century. Chapter 2 also refers to some of the currently popular weight loss solutions, and discusses why they are not effective.

In Chapter 3 the literature study is expanded to gain a better understanding of how the human energy system and its controls work. Specific attention is paid to the understanding of the metabolic pathways of each macronutrient and the energy that arises from it. The utilization of this energy in the form of blood glucose is exploited as a better method to quantify the available energy from macronutrients. The Glycaemic Index (GI) of carbohydrates is considered as a utilization coefficient or conversion factor and forms a very important key to the research methodology.

In Chapter 4 the new energy equation derived by Mathews *et al,* that quantifies the available energy from carbohydrates, is discussed. This new energy concept is called—ets and will form the backbone of any further derivations and conceptual thinking. It is confirmed that some carbohydrates have less energy than previously believed, and this can now be measured in —ets Calories.

A confirmation of the integrity of the new —ets concept can only be made by means of clinical trial results. In Chapter 5 a protocol is designed to test the—ets concept on a rat model. Groups of Spraque-Dawley rats were fed different foods with the same calorie values, but with different —ets Cal values. The average group weight was monitored for four weeks, as a reflection of the energy that the rats can extract from different foods.

The results captured in the clinical trial go through a sequence of bio-statistical analyses in Chapter 6. This ensures that any conclusions drawn from the data are statistically correct and meet the requirements to establish a new theory. It is clearly demonstrated that not only do different carbohydrates contain different amounts of available metabolic energy, but also that a linear relationship exists between —ets Cal and weight changes in Spraque-Dawley rats.

Chapter 7 concludes the development from the—ets concept to a theory that can contribute to a better obesity solution. Although it certainly is not a total solution to the obesity epidemic,—ets brings us closer to an understanding of the fine energy homeostasis of the human body.

1.5 References

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2 INTRODUCTION TO OBESITY

Over the years, animals have adopted certain physiological changes that enabled them to survive in a changing environment. This rate of evolution has always been synchronized with the rate of the changing environment, and has ensured the survival of the species. In rare events, like a sudden change in the earths orbit due to impact from asteroids, dramatic changes in temperature have resulted in the extinction of certain species.

Although human physiology has stayed pretty much the same for the past 50,000 years or so, we humans have utterly transformed our environment. It seems that the human race is changing its environment at a rate faster than we are physiological able to evolve. Can we contribute the cause of obesity, heart disease, high blood pressure, stroke, diabetes, infertility, gall-bladder disease, osteoarthritis and many forms of chronic disease that threaten our survival to our inability to adapt quickly enough?

The single most significant identifiable factor that has changed during the last two millennia is probably the diet of Homo Sapiens. This study will take a closer look at obesity; it's impact on society and possible causes.

2.1 The obesity epidemic

Obesity is an excessive accumulation of fat in the body. It can be assessed by various ways including Body Mass Index (BMI), waist circumference, life insurance tables, CT (computed tomography) / MRI (magnetic resonance imaging) and absorptiometry. Body Mass Index (BMI) is the most widely accepted means of assessing obesity (expressed as weight/height^2 - kg/m^2). The relationship of BMI to total body and visceral fat, and consequent complications varies between ethnic groups. [1]

Asian population (particularly those from South East Asia) have more fat and co-morbidities for any given BMI, resulting in different suggested BMI cut-off points. [2] Adult BMI cut offs cannot be used in children and adolescents to assess obesity, as BMI varies throughout childhood See ***Table 1: Classification of obesity***.

Classification	Caucasian	Asian
Normal Range	18.5 - 24.9	18.5 - 22.9
Overweight	25.0 - 29.9	23.0 - 25.9
Obese	>30.0	>26
Class 1	30.0 - 34.9	26.0 - 29.9
Class 2	35.0 - 39.9	30.0 - 35.0
Class 3	>40.0	>35

Table 1: Classification of obesity

In children, the BMI is higher in the second year of life and then drops at ages 4-7 years, rising slowly to adult values. BMI for age charts can be used in clinical practice to assess obesity in children. [3] According to these charts, a child is overweight if it has a BMI between 85th - 95th percentiles, and obese above 95th percentiles.

Central obesity, particularly visceral fat, is a risk factor for metabolic syndrome. Waist circumference cut-offs have been internationally accepted for adults, but there are no internationally accepted criteria for waist circumference in children.

Gender	Caucasian	Asian
Men	>102	>90
Woman	>88	>80

Table 2: Waist Circumference cut-off points for obesity

2.1.1 Epidemiology of obesity

The prevalence of obesity is steadily increasing across the world; particularly in the developed countries. In 1980, 39% of men and 32% of women in UK were overweight or obese and during 1991, this figure rose to 53% and 44% respectively. [4]

The World Health Organisation (WHO) estimates the prevalence of obesity to be 4.8% in the developing countries, 17.1% in countries in economic transition and 20% in the developed world. More than one billion adults worldwide are overweight and at least 300 million of them are clinically obese. [5]

The increase in prevalence of overweight and obesity is not limited to adults, but is even more widespread in children. In Australian children, over the decade 1985 – 1995, the combined prevalence of the two conditions almost doubled, while that of obesity on its own more than tripled. [6]

2.1.2 Etiology of obesity

The etiology of obesity is complex and multifactorial. Both environmental and genetic influences play a role. In particular, over the past century, technology has almost completely removed physical exercise from the day-to-day lives of most people. At the same time it has filled supermarket shelves with cheap, mass-produced, good-tasting food that is packed with calories. And finally, technology has allowed advertisers to deliver constant, virtually irresistible messages that say "Eat this now" to every one old enough to watch TV.

This artificial environment is most pervasive in the U.S. and other industrialized countries, and that is exactly where the fat crisis is most acute. When people move to the U.S. from poorer nations, their collective mass begins to rise. As developing areas like Southeast Asia and Latin America catch up economical and the inhabitants adopt Western lifestyles, their problems with obesity catch up as well. By contrast, among people who still live in conditions like those of our distance Stone Age ancestors – such as the Maku or the Yanomami of Brazil – there is virtually no obesity at all. [7]

It was some 2.5 million years ago that hominid ancestors developed a taste for meat. The fossil records show that the human brain became markedly bigger and more complex about the same time. According to Katherine Milton, an anthropologist at the University of California, Berkeley, "the incorporation of animal matter into the diet played an absolutely essential role in human evolution."

However, the new appetite for meat didn't mean we lost our passion for sweets. As Berkeley Milton points out, the brain's growth may have been facilitated by abundant animal protein, but the brain operates on glucose that serves as the major fuel for cellular function. The sugar in fruits and the carbohydrates in edible grains and tubers are particular good source of glucose.

The appetite for meat and sweets was essential to human survival, but that did not lead to obesity for several reasons. For one thing, the wild game our ancestors ate was high in protein and very low in fat – only about 4%, compared with up to 36% in grain-fed supermarket beef. For another reason, our ancestors could not count on a steady supply of any particular food. Fruit might be in season, or it might not.

Beyond that, hunting and gathering took enormous physical work. In essence, early humans ate what amounted to the best of the high-protein Atkins diet and the low-fat Ornish diet, and worked out almost non-stop. To get a sense of their endurance, cardiovascular fitness, musculature and body fat, (say evolutionary anthropologists), just look at a modern marathon runner.

Then came what anthropologists call humanity's worst mistake: the invention of agriculture. Nutritionally, the shift away from wild meat, fruits and vegetables to a diet mostly of cultivated grain, robbed humans of many of the essential amino acids, vitamins and minerals they have thrived on. The average lifespan increased, thanks to the greater abundance of food, but the average height diminished. Skeletons also began to show a jump in calcium deficiency, anaemia, bad teeth and bacterial infections.

It's really only in the last 100 years that cars and other machinery have reduced the need for physical labour. As exercise has vanished from everyday life, the technology of food production has become much more sophisticated. Farmers with powerful fertilizers and high-tech equipment, are growing enormous quantities of corn and wheat, most of which is processed and refined to be tastier and more convenient - but less nutritious.

There is no doubt that the obesity epidemic is real and our collective health has been getting progressively worse. Indeed, says Dr. David Katz, "today's kids may well be the first generation in history whose life expectancy is projected to be less than that of their parents."

2.1.3 Carbohydrate Intake and Rate of Obesity

Evaluating trends in dietary intake is an important step in understanding the factors that contribute to the increase in obesity. To assess trends in intake of energy (i.e., kilocalories

[kcals]), protein, carbohydrate, total fat, and saturated fat during 1971--2000, CDC (Centre for Disease Control) analyzed data from four National Health and Nutrition Examination Surveys (NHANES): NHANES I (conducted during 1971--1974), NHANES II (1976--1980), NHANES III (1988--1994), and NHANES 1999--2000.

This report summarizes the results of that analysis, which indicate that, during 1971--2000, the mean energy intake in kcals increased the mean percentage of kcals from carbohydrate increased, but the mean percentage of kcals from total fat and saturated fat decreased.

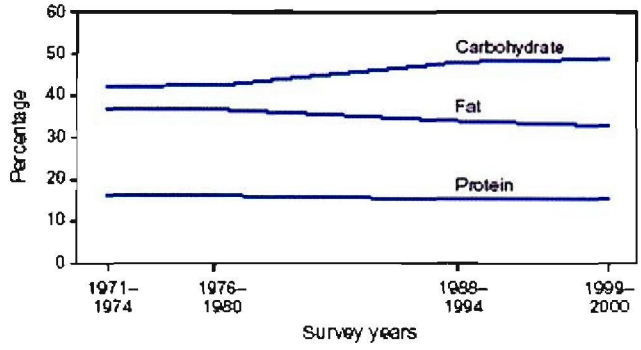

Figure 3: Percentage of kcal from macronutrient intake among men

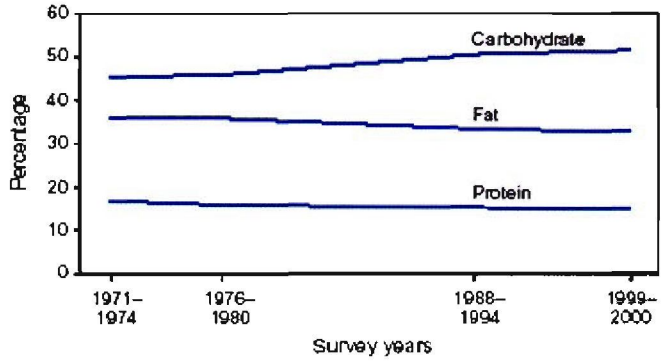

Figure 4: Percentage of kcal from macronutrient t intake among woman

Results from the 1999-2002 National Health and Nutrition Examination Survey (NHANES), using measured heights and weights, indicate that an estimated 65 percent of U.S. adults are either overweight or obese. As shown in ***Figure 3***, this represents a prevalence that is 16 percent higher than the age-adjusted overweight estimates obtained from NHANES III (1988-94).

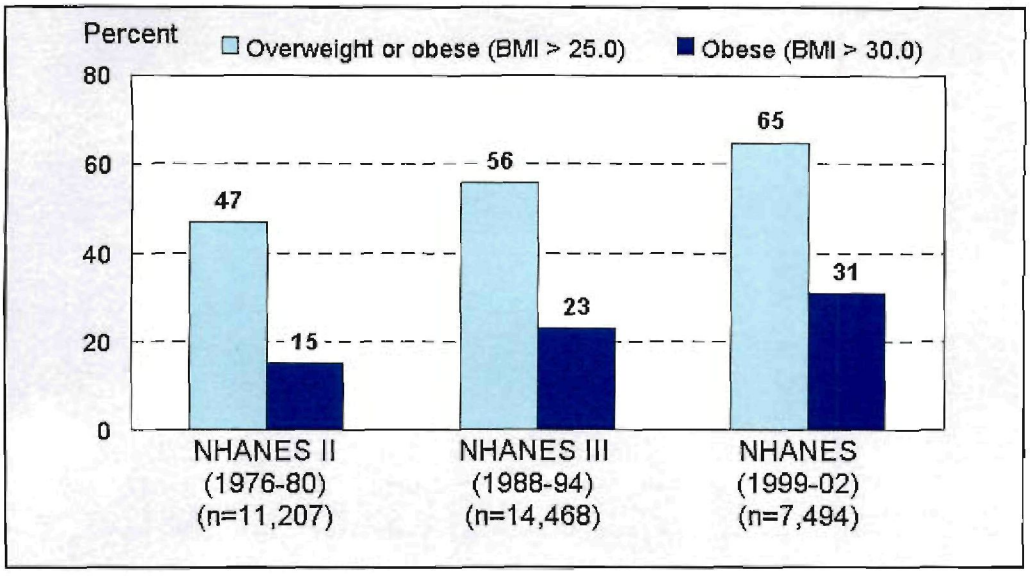

Figure 5: Age adjusted prevalence of overweight and obesity among U.S. adults

When age-adjusted prevalence estimates from the NHANES III for adult's age 20-74 years were compared with prevalence estimates from NHANES II (1976-80), there were notable increases in the prevalence of persons who were either overweight or obese. Most of this increase was attributable to an increase in the obese category (BMI greater than or equal to 30.0), whereas only minor increases occurred in the prevalence of persons who are overweight but not obese (BMI 25.0-29.9). [8]

2.2 Impact of obesity

2.2.1 Medical consequences

Obesity is associated with increased morbidity and mortality. This has been known for more than 2000 years and Hippocrates said, "Sudden death is more common in those who are naturally fat than in the lean." In obesity the excess energy is stored in fat cells that enlarge and / or increase in number. Enlarged fat cells produce clinical problems associated with obesity either because of mass of extra fat or because of increased secretion of free fatty acids and numerous peptides from enlarged cells. Medical condition resulting from obesity includes:

Diabetes Mellitus

Type 2 Diabetes is strongly associated with excessive weight in both sexes in all-ethnic population. [9],[10] The risk of diabetes increases with duration and degree of obesity and with a more central distribution of body fat. In the Nurses Health Study the risk of diabetes was lowest with BMI less than 22 kg/m2. As BMI increased the relative risk increased such that with a BMI of 35, the relative risk increased 40 fold. From 1980 through 2003, the number of Americans with diabetes more than doubled.

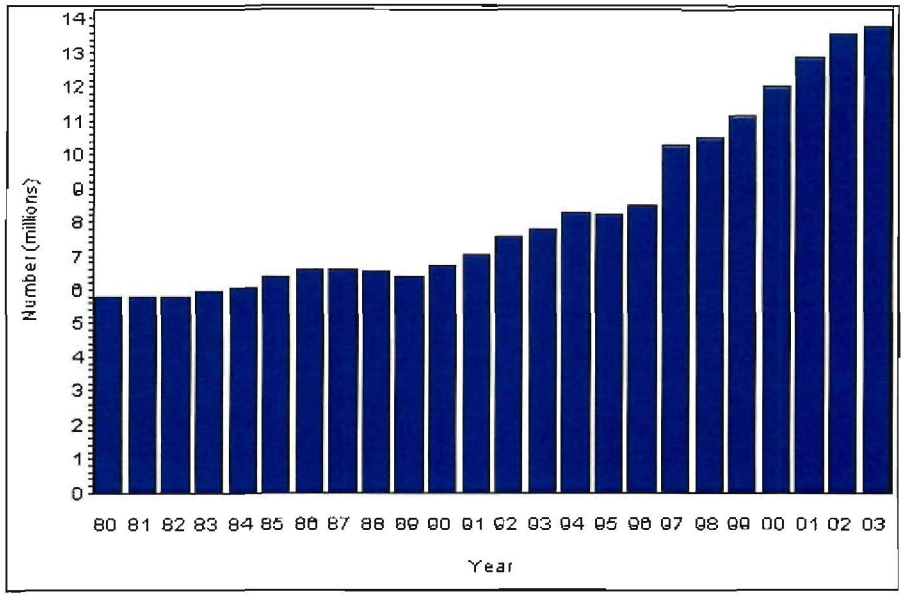

Figure 6: Number of Americans with Diabetes

Newly released statistics from the Centre for Disease Control and Prevention (CDC) illustrate that diabetes has risen by over 14 percent in the last two years. The CDC estimates that 20.8 million Americans, 7 percent of the U.S. population have diabetes. Nearly a third of these Americans are undiagnosed. According to the American Diabetes Association, the new numbers highlight the growing diabetes epidemic in the United States and reinforce the need for increased research and prevention. [11]

Hypertension

Blood Pressure is often increased in obese and overweight subjects. [12] Hypertension in obese subjects appears to be related to altered sympathetic activities. The combination of overweight

and hypertension leads to thickening of ventricular wall and larger heart volume with a greater likelihood of cardiac failure.

Dyslipidaemia

A positive correlation between BMI and triglycerides has been repeatedly demonstrated. An inverse relationship of BMI with HDL (High Density Lipoprotein), the good cholesterol, is more important as a risk factor for coronary artery disease. [13]

Heart disease

Data from the "Nurses Health Study" [14] indicate that the risk of women developing coronary artery disease is increased greater than 3 folds with a BMI greater than 29. Dyslipidaemia, Hypertension and Diabetes all contribute to this increased risk. Aerobic Centre Longitudinal Study [15] involving 25714 men who were followed for 1-10 years, has shown that the cardiovascular mortality was higher in men with BMI greater than 30 kg/m2.

Cancer

Certain cancers are significantly increased in overweight and obese individuals. These include malignant neoplasm of colon, rectum and prostrate in men, and cancers of breast, uterus and gallbladder in women.

Non-alcoholic fatty liver disease (NAFLD)

It is a disease having liver abnormalities associated with obesity comprising of hepatomegaly, elevated liver enzymes and abnormal liver histology. [16] In a cross sectional study involving liver biopsies in obese subjects have shown steatosis in 75%, steatohepatitis in 20% and cirrhosis in 2%. [17]

Gallbladder disease

The clinical saying "fat, female, fertile and forty" describes the epidemiology of gallbladder disease associated with cholelithiasis. Nurses Health Study has demonstrated this very clearly. It has been shown that the incidence of gallstones gradually increase with increased BMI up to 30

and very steeply with higher BMI. One of the explanations for increased risk of gallstones is the increased cholesterol turnover related to increased body fat.

Diseases of the bones and joints

Osteoarthritis is significantly increased in obese patients. The joints affected are usually the knees and ankles and is directly related to trauma associated with the degree of excess body weight. [18] Increased osteoarthritis of other non-weight hearing joint is also seen in obese patients.

Sleep apnea

Pulmonary functions are altered in obese patients showing a decrease in residual long volume associated with increased abdominal pressure on the diaphragm. [19] In addition to this benign effect on pulmonary function, obstructive sleep apnoea is also seen more in obese tall men.

Reproductive/Endocrine abnormalities

Varieties of endocrine changes are seen in obese patients but the changes in reproductive system in women are most profound. Irregular, infrequent and an-ovulatory menstrual cycles are common in obese women and the rate of fertility is also reduced. [20],[21] Hirsuitism is also more commonly seen in obese women who may be suffering from P.C.O.S.

Increased mortality/shortened life expectancy

Framingham Study has shown loss of 3.3 years in overweight women and 3.1 years in overweight men compared with normal weight men and women. [22] In obese women and men these shortened life years are more pronounced reaching 7.1 years and 5.8 years respectively. Despite the fact that obesity is more common in African- Americans than Caucasian-Americans, it is more lethal for whites than for blacks. [23] Nurses Health Study [12], American Cancer Society Cancer Prevention Study I and II have both shown increased mortality in both men and women with BMI in the obese range. [24],[25]

The benefits gained from subjects losing 10kg is summarised in ***Table 3***: [26],[27],[28]

Benefits of 10kg Weight Loss	
Mortality	**Lipids**
Reduction:	**Reduction:**
>20% total mortality	10% total cholesterol
>30% diabetes-related deaths	15% LDL
>40% obesity-related cancer deaths	30% triglycerides
	Increase:
	8% HDL
Blood pressure	
Reduction:	
10mmHg systolic	Respitatory
20mmHg diastolic	Reduced sleep apnoea
Decreased breathlessness	
Diabetes	
Reduction:	Gynaecological
50% fasting glucose	Improve ovarian function and fertility in PCOS

Table 3: Medical Benifits of 10kg Weight Loss

2.2.2 Economic impact

It is almost incomprehensible that Americans spend $117 billion a year on obesity-linked illness. Diet and poor exercise trail only tobacco as cause of preventable death. In the following section we lay out the facts that contribute to the loss of $117 billion in one nation. [29]

Table 4 lists America's five fastest growing health concerns and gives the percentage at which they have grown between 1999 and 2001:

Health Concerns	2001	1999
Obesity	61.0%	56.4%
Diabetes	18.3%	12.0%
Depression	19.1%	14.8%
Impotence	9.2%	7.4%
Aging Related Problems	22.5%	19.2%

Table 4: America's fastest growing health concerns

USA obesity rates reach epidemic proportions

- 58 Million Overweight; 40 Million Obese; 3 Million morbidly Obese
- Eight out of 10 over 25's Overweight
- 78% of America's not meeting basic activity level recommendations
- 25% completely Sedentary
- 76% increase in Type II diabetes in adults 30-40 yrs old since 1990

Obesity related diseases

- 80% to Type II diabetes related to obesity
- 70% of Cardiovascular diseases related to obesity
- 42% of breast and colon cancer diagnosed among obese individuals
- 30% of gall bladder surgery related to obesity
- 26% of obese people having high blood pressure

Obesity related diseases costs overwhelm healthcare systems

- Type II Diabetes - $63.14 Billion
- Osteoporosis - $17.2 Billion
- Hypertension - $3.23 Billion
- Coronary Heart Disease - $6.99 Billion
- Post-menopausal breast cancer - $2.32 Billion
- Colon Cancer - $2.78 Billion
- Endometrial Cancer - $790 Million

Cost of productivity

- Workdays lost: $39.3 Million
- Physical office visits: $62.7 Million
- Restricted Activity days: $29.9 Million
- Bed-related days: $89.5 Million

Childhood obesity running out of control

- 4% overweight 1982 / 16% overweight 1994
- 25% of all white children overweight 2001
- 33% African American and Hispanic children overweight 2001
- Hospital costs associated with childhood obesity rising from $35 Million to $127 Million (1999)

2.3 Existing weight loss solutions

2.3.1 Drug therapy

Pharmacotherapy should be considered in obese subjects with BMI greater than 27kg/m2 in the presence of co-morbidities such as Type 2 Diabetes and Hypertension, when life style modification has not resulted in desired weight loss. In the absence of co-morbidities, a BMI of 30 and above is the cut-off to consider drug therapy. [30] Only two drugs, Sibutramine (Meridia, Reductil, Abbott laboratories) and Orlistat (Xenical, Hoffman- Laroche) are licensed for use in obesity by the Food and Drug Administration for long-term use.

2.3.2 Surgery

Life style modification has limited success resulting in no more than 10% of total body weight. Bariatric surgery is the only effective modality for long-term weight loss for severely obese patients. [31] They produce weight loss and maintenance of 30 - 40%.

The indications for bariatric surgery are morbidly obese patients with BMI >40 or obese patients with BMI > 35 with associated co-morbids.[32] A comparison of different bariatic surgeries is shown in ***Table 5***:

	Gastric Bypass	Biliopancreatic diversion with duodenal switch	Gastric Band
Duration of procedure	1 - 4 hours	2 - 5 hours	0.5 - 2 hours
Length of day	2 - 3 hours	2 -4 days	1 - 2 days
Postoperative supplements	MVI, iron, calcium	MVI, iron, calcium, ADEK	MVI, calcium
Estimate weight loss	50 - 75% EBW	60 - 80% EBW	40 - 60% EBW
Side effects	dumping sundrome	diarrhea, excessive flatus, body odor changes	Vomiting
Short-term complications	DVT/PE, anastomotic leakage, pouch leakage, gastrointestinal bleeding	DVT/PE, anastomotic leakage, pouch leakage, gastrointestinal bleeding	DVT/PE. Port-site infection, esophageal perforation
Long-term complications	gastrojejunostomy stenosis, iron deficiency anemia, calsium deficiency, B12 deficiency, marginal ulcer, internal hernia	Iron deficiency, calcium deficiency, protein malnutritiun, need for common channel revision, internal hermia	Band slippage, device leakage, erosion inot stomach/seophagus, pouch enlargement, device infection

Table 5: Weight Loss Operations

These surgeries are associated with significant morbidity and mortality in inexperienced hands. Peri-operative mortality of 1% and a complication rate of 10% are reported from experienced centres across the world. Adjustable laproscopic gastric banding is becoming the favoured approach because of its reversibility and low morbidity. Excellent results have been reported for Europe and Australia. For unclear reasons the results of this surgery in USA is not very good.[33]

2.3.3 Fad diets

People are often willing to try anything that promises to help them lose weight because they want to look or feel better, or because they are worried about getting weight-related diseases. Companies that promote fad diets take advantage of this fact. They appeal to people by promising weight loss that's very quick and easy. Many people prefer to try the quick fix of a fad

diet instead of making the effort to lose weight through long-term changes in their eating and exercise habits.

In many of these diets the short-term effect is more rewarding but less sustaining in the long run. This is because most of these diets promote the loss of water in the initial stages of the diet and actually very little adipose tissue. ***Table 6*** contains a list of the type of fad diets and also the products available in the market:

Diet Type	Products on the Marked
Controlled Carbohydrates	Dr. Atkins' New Diet The Carbohydrate Addict's Diet Protein Power The Formula Sugar Busters South Beach Diet The Zone
High Carbohydrate/Low Fat	Dr. Dean Ornish: Eat More, Weigh Less The Good Carbohydrate Revolution The Pritikin Principle
Controlled Portion Sizes	Dr. Shapiro's Picture Perfect Weight Loss Volumetric Weight-Control Plan
Food Combining	Fit for Life Suzanne Somers' Somersizing
Liquid Diets	Cambridge Diet Slim-Fast
Diet Pills/Herbal Remedies	Dexatrim Natural Hydroxycut Metabolite 356
Other	Eat Right For Your Type: The Blood Type Diet Macrobiotics Mayo Clinic Diet*

Table 6: Popular Fad Diets

2.4 Conclusion

Obesity is a chronic condition that predisposes patients to multiple serious health disorders and premature deaths. Body Mass Index is the most widely accepted measure of obesity in adults. BMI though established measure of obesity; waist circumference is gaining importance as it measures central obesity, which is an important risk factor for metabolic syndrome.

The prevalence of obesity is steadily increasing across the world particularly in developed countries. This epidemic will continue to plague our society for many years with all its medical consequences. Although influenced by genetics, the current obesity epidemic appears to be driven principally by environmental factors. Lifestyle factors of high-energy food intake and lack of physical activity are the greatest contributors to the energy imbalance that causes obesity.

Treatment of obesity involves dedicated and sustained lifestyle modification assisted by anti-obesity drugs, which has modest effect in loosing weight of 5-10%. Our growing understanding of the complex mechanism of energy balance in our body will allow the development of newer and safe drugs in this field. Bariatric surgery is the only effective modality for long-term weight loss for morbidly obese patients.

Major efforts are needed to curb the escalating incidence of obesity globally. Prevention strategies that involve lifestyle interventions should be promoted. Individual and collective efforts at community and population levels are needed if we are to stem this epidemic. Despite current prevention strategies and various treatment methods, eradication of obesity does not appear to be on the scene in the foreseeable future.

Obesity is the simple result of an imbalance between energy intake and energy expenditure. When macronutrients are metabolised, one would expect the available energy to be less than the actual bomb calorie values. It is necessary to account for metabolic conversion efficiencies (η) of the body for each different macronutrient.

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3 THE HUMAN ENERGY SYSTEM AND METABOLIC FUELS

3.1 Metabolic pathways

Metabolic pathways refer to the breakdown of protein, fat and sugar or glucose to fuel the processes required to sustain life in a human body. These processes, although described separately, are integrated and interlinked, with some fuels converted to others when needed. For example, glucogen is converted to glucose in the liver in a process called glugenolysis to increase blood glucose. In a similar process called glugoneogenesis triglycerides and amino acids are converted to glucose into the liver according to the body's need.

This process of fuel consumption begins when glucose, amino acids and triglycerides are absorbed in the gastro-intestinal tract and transported via the portal vein directly to the liver as schematically represented in ***Figure 8***. The processes that follow are determined by the requirements of the body, and their proportionate contribution as a percentage of the whole is dependant on the specific state of the body at any particular time.

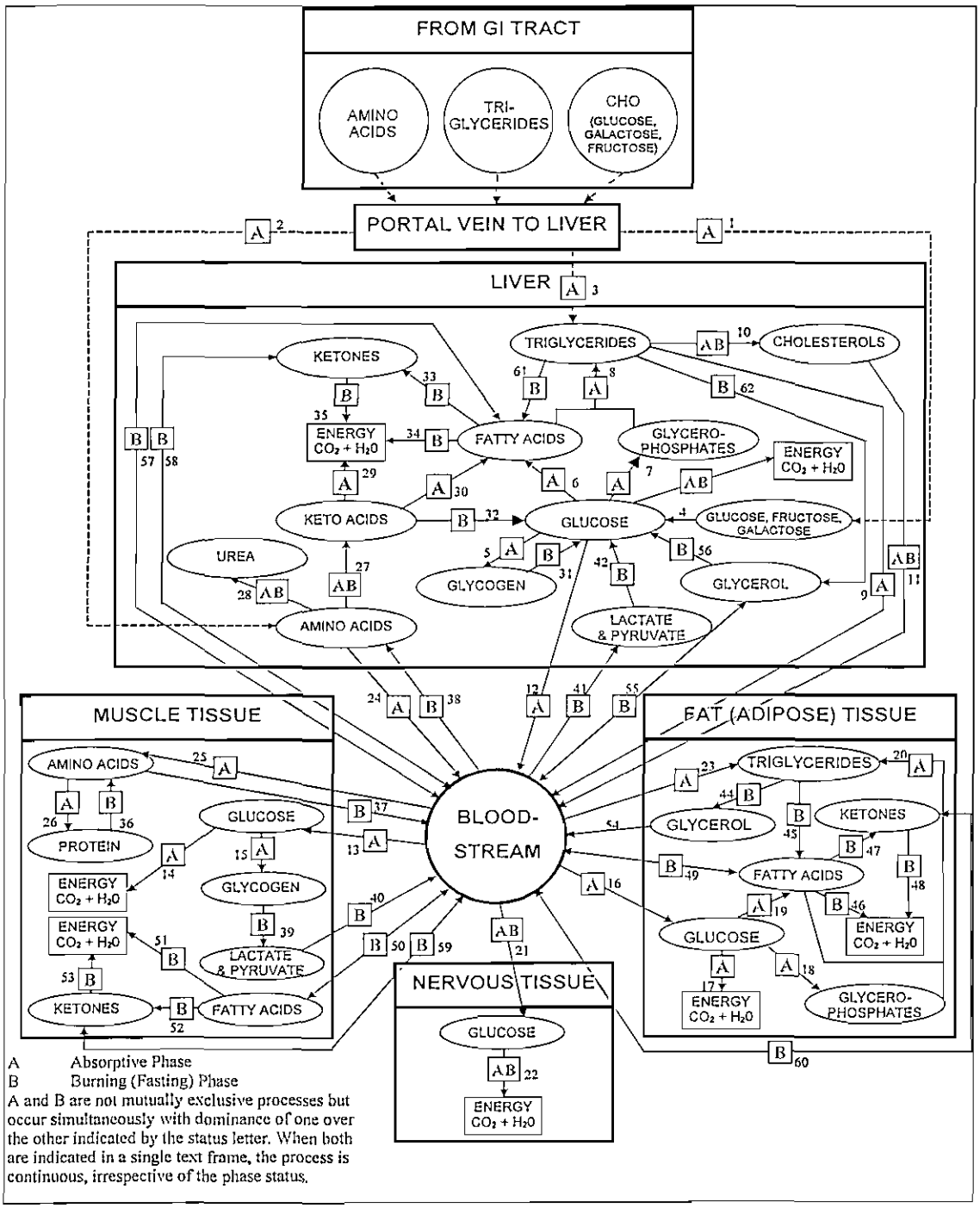


Figure 7: Simplified schematic layout of the major energy pathways in the human energy system.

3.1.1 The carbohydrate pathway

The carbohydrate pathway begins at the point where glucose, fructose and galactose are transported from the gastro-intestinal tract via the portal vein to the liver (1). In the liver galactose and fructose are converted to glucose (4). The four pathways for glucose utilisation lead into the liver, the muscle tissue, the adipose tissue and the nervous system. Here follows a short description of each of these pathways.

In the liver

A part of glucose is directly converted into glycogen and stored in the liver (5) while some is burnt for energy. Another portion of glucose is converted into fatty acids (6) and glycero-phosphates (7), which combine to form triglycerides (8). The triglycerides are then either stored in the liver, released into the bloodstream (9), or converted to cholesterols (10) and in turn released in the blood (11). Some of the glucose is released from the liver into the bloodstream (12).

The portion of glucose that was converted into fatty acids can directly be burned for energy in the liver (34). This process produces ketones as by-product (33). Ketones can in turn also be burned for energy in the liver (35) or released into the bloodstream (58). Unused ketones are excreted through the breath, urine and stool. Similarly fatty acids can also be released into the bloodstream (57) to be used as energy elsewhere. When required, glycogen stored in the liver can be reconverted back into glucose (31) and any of the above processes can again be performed.

In the muscle tissue

Glucose from the bloodstream can be absorbed in the muscle (13) and burned directly in the muscle tissue for movement and heat energy (14). It can however also be converted into glycogen for energy storage (15).

Furthermore, the glycogen can also be converted into lactate and pyruvate (39), which is released into the bloodstream (40) and converted in the liver (41) back into glucose (42). From here the pathways for glucose utilisation in the liver are the same as described above.

In the adipose tissue

Glucose absorbed from the bloodstream (16) can be burned directly for heat energy in the adipose or fat tissue (17), or, as in the case of the liver, converted into fatty acids (19) and glycero-phosphates (18). These two again combine to form triglycerides (20), which are stored as energy reserves.

Similarly to the case of the liver, the portion of glucose that was converted into fatty acids can directly be burned for energy in the adipose tissue (46), which also produces ketones as by-product (47). The ketones can in turn also be burned for energy in the adipose tissue (48).

In the nervous tissue

A constant flow of glucose is required from the bloodstream (21) to supply the nervous tissue with fuel. The glucose is then directly burned within the nervous tissue to produce energy for sustaining life (22).

3.1.2 The amino acid pathway

The amino acid pathway begins at the point where amino acids are transported from the gastro-intestinal tract via the portal vein to the liver (2). Amino acids are only utilised in the liver and muscle tissue, and the pathways for utilisation are the following:

In the liver

A portion of the absorbed amino acids is released by the liver into the bloodstream (24).

Another portion of amino acids is converted directly in the liver into keto acids (27), which can be directly utilised as energy (29). The remaining amino acids are directly excreted from the body as waste (28).

The keto acids can furthermore be converted into fatty acids (30) from where it follows the same path as was described in the carbohydrate pathway. In other words fatty acids can be directly burned for energy in the liver (34), with ketones as by-product (33). The ketones in turn can also be burned for energy in the liver (35). The fatty acids can furthermore follow the path of formation of triglycerides as was described in the carbohydrate pathway.

In the muscle tissue

The muscles can absorb the free amino acids from the bloodstream (25) and convert it into protein for building muscle tissue (26). If required in low energy availability situations, protein (muscle tissue) can be converted back into amino acids (36) and released into the bloodstream (37). From here it follows the pathway back to the liver, as described above, to replenish the energy store that is low.

3.1.3 The fat pathway

The triglyceride or fat pathway begins at the point where triglycerides are transported from the gastro-intestinal tract via the portal vein to the liver (3). Triglycerides or their derivatives are mainly utilised along the following pathways:

In the liver

A portion of the ingested triglycerides as well as the triglycerides produced in the liver (from the glucose pathway) can be stored in the liver or released directly into the bloodstream (9).
Another portion is directly converted in the liver into cholesterols (10), which are in turn also released into the bloodstream (11).

The remaining portion may be broken down into fatty acids (61) and glycerol (62) before being released into the bloodstream (55 and 57).

In the adipose tissue

The fat or adipose tissue can absorb the triglycerides from the bloodstream (23) and can directly store is as triglyceride energy reserves. When required in low energy availability situations, triglycerides can be converted into glycerol (44) as well as fatty acids (45). Glycerol is released into the bloodstream (54), conveyed back to the liver (55) and converted back into glucose (56). From here it follows the glucose pathways in the liver as already described.

A portion of the fatty acids produced from triglycerides in the adipose tissue (45) can directly be burned for energy in the adipose tissue (46), which produces ketones as by-product (47). The ketones can in turn also be burned for energy in the adipose tissue (48) or released into the blood (60). The remainder of fatty acids are then also released into the bloodstream (49) from where it is either utilised in the liver (57) along the same pathway as fatty acids produced in the liver, or used in the muscle tissue (50).

In the muscle tissue

Fatty acids produced by adipose tissue (49) or by the liver (57) and released into the bloodstream can be transported to the muscle tissue (50) or any other tissue (except nervous tissue) to be burned for energy.

The process of burning the fatty acids is very similar to that already described in the liver. The fatty acids can directly be burned for energy in the muscle tissue (51), which produces ketones as by-product (52). The ketones can in turn also be burned for energy in the muscle tissue (53). As in the case with all ketone production, some may be released into the blood (59) and utilised elsewhere.

3.2 Quantifying macro nutrient energy

Many different methods have been used to determine how much energy is available for human metabolism, termed metabolizable energy. The total amount of energy in a food can be determined by a bomb calorimeter and will be less than metabolizable energy because of incomplete absorption. This energy is lost through urine and faeces, but some is lost in the gases and heat produced during colonic fermentation. The following section discusses the total amount of energy for macro nutrients.

3.2.1 Joules and calories

The unit of energy in the International System of Units (SI) is the joule (J). A joule is the energy expended when 1 kg is moved 1 m by a force of 1 Newton. This is the accepted standard unit of energy used in human energetics and it should also be used for the expression of energy in foods. Because nutritionists and food scientists are concerned with large amounts of energy, they generally use kiloJoules (kJ = 10^3 J) or megaJoules (MJ = 10^6 J).

For many decades, food energy has been expressed in calories, which is not a coherent unit of thermochemical energy. Values for food energy in the following sections are given in both joules and calories. The conversion factors for joules and calories are: 1 kJ = 0.239 kcal; and 1 kcal = 4.184 kJ.

3.2.2 Protein

Protein is best measured as the sum of individual amino acid residues (the molecular weight of each amino acid less the molecular weight of water). When protein is expressed as the sum of amino acids, an energy conversion factor of 17 kJ/g (4 kcal/g) should be used as specified by Merrill and Watt (1973)

3.2.3 Fat

For energy purposes, fats should be analysed as fatty acids and expressed as triglycerides (FAO, 1998), as this approach excludes wax esters and the phosphate content of phospholipids, neither of which can be used for energy. For normal dietary fats, a factor of 37 kJ/g (9 kcal/g) should be used as specified by Merrill and Watt (1973).

3.2.4 Carbohydrates dietary fibre

Carbohydrate should be analysed in a way that allows determination of both available carbohydrate and dietary fibre.

Direct analysis allows separation of individual mono- and disaccharides and starch, which is useful in determination of energy values. Direct analysis is considered the only acceptable method for analysis of carbohydrate in novel foods or in foods for which a reduced energy content claim is to be made. When carbohydrate is determined by direct analysis, it is expressed as the weight of the carbohydrate with a conversion factor of 17 kJ/g (4.0 kcal/g). When expressed as monosaccharide equivalents, a conversion factor of 16 kJ/g (3.75 kcal/g) should be used.

When dealing with fibres or oligosaccharides that are specifically added to a food, an analytical method and an energy conversion factor specific for the fibre or oligosaccharide in questions should be used. For example, energy conversion factors range from 1.3 kJ/g (0.3 kcal/g) for maize bran fibre to 11 kJ/g (2.6 kcal/g) for fructo-oligosaccharides.

The energy factor to be applied to these results should be appropriate for the faction analysed. In the absence of a specific factor associated with the method, a value of 8 kJ/g (2 kcal/g) should be used.

3.3 The blood sugar hypothesis

It is clear by looking at the complex metabolic pathways that macronutrients follow, that it is not a feasible solution to use the laws of thermodynamics to determine the human energy homeostasis. The countless metabolic coefficients that differ for each individual make this approach even more difficult, and the solution non-generic.

All macronutrients are, however, converted to blood glucose that fuels the body. Blood glucose can thus be seen as a single metabolic coefficient that captures all variables into a single unit of energy potential.

3.3.1 The blood glucose control system

The effects on blood sugar levels by a number of controlling factors are much more measurable when compared with the energy pathways for all fuels, as described in the previous section. Knowledge of the energy pathways is, however, essential for the understanding of phenomena occurring in the process of blood sugar control. The blood sugar control system of the human body is schematically represented in ***Figure 9***.

Arrowed solid hairlines indicate changes in blood glucose concentrations and direction of energy flow, while broken arrowed lines indicate control or measuring mechanisms and direction of targets. The controlling hormone for each process is indicated in abbreviated form in square text frames. Processes that increase and decrease blood sugar levels are indicated with "Bs+" and "Bs-" respectively, enclosed in oval text frames. More details are given on the sketch.

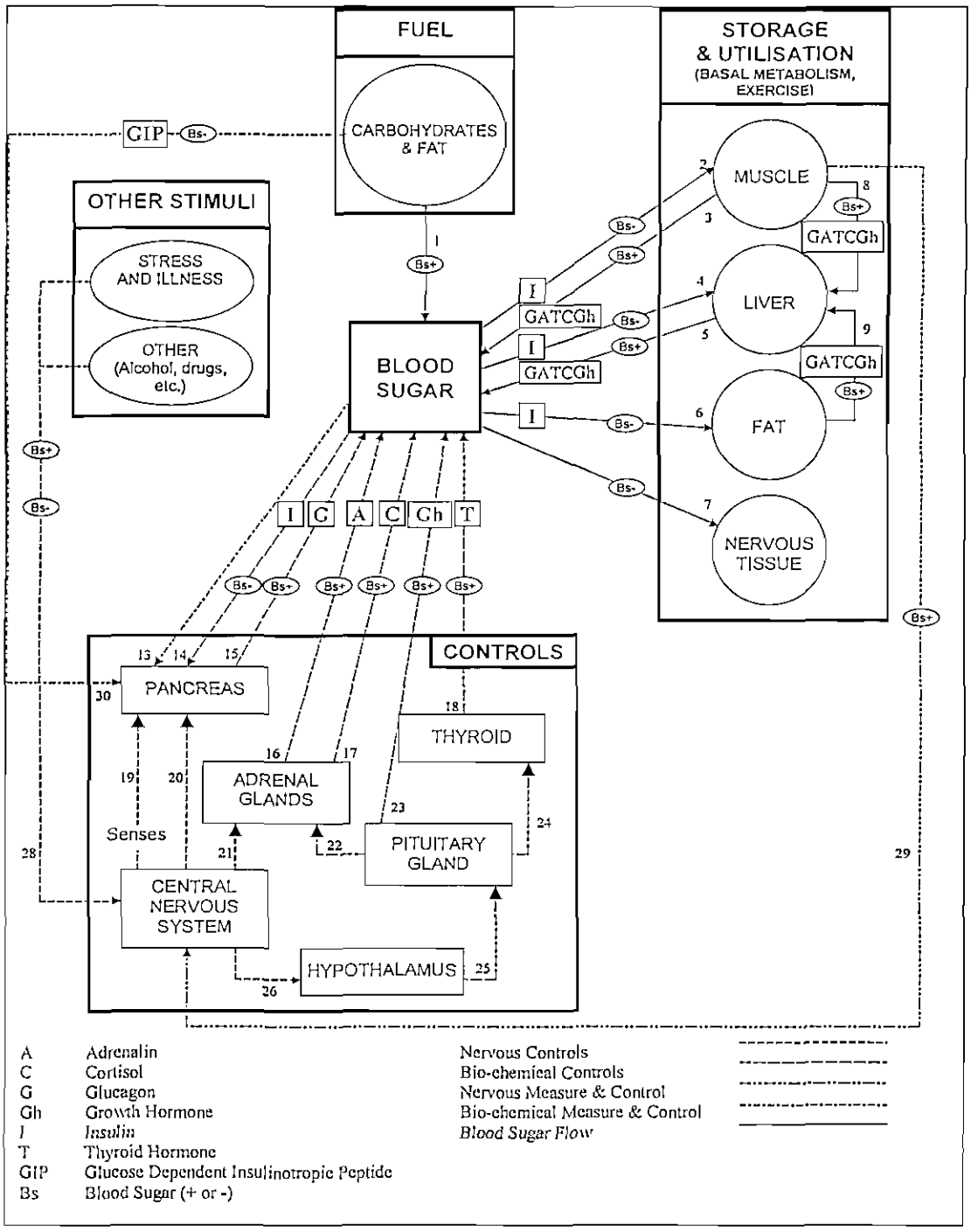


Figure 8: Schematic layout of the blood sugar control system

For the purpose of this study it is only necessary to discuss the main control hormones, namely insulin and glucagon.

Insulin

Insulin is an anabolic hormone secreted by the beta cells of the islets of Langerhans in the pancreas. Not only does it enable glucose to be used as energy, but it also allows glucose to be stored in the liver and muscles, preventing the body from using fat or body protein for energy sources. [Diabetes, The Natural Way, Catherine Steven] Amongst others its functions include:

- The regulation of blood glucose by suppressing blood glucose level and repressing gluconeogenesis and glycogenolysis. It also decreases substrates used in gluconeogenesis and inhibits lipolysis;

- Controlling the flow of carbohydrates, fatty acids and amino acids to the different body tissues. It henceforth causes enhancing of amino acid uptake and protein synthesis;

- Regulation of the production of cholesterol by the liver;

- Controlling the absorption mechanisms for storage of fat and glycogen for later use as an energy source.

Insulin is the body's top-end blood sugar level controller since an increase in insulin concentration has the direct effect of lowering high blood sugar. Type 1 diabetics do not have the substance produced in their bodies and therefore have to inject it at the appropriate times, usually before meals. Type 2 diabetics on the other hand do produce insulin, but it is either in inadequate quantities or they are unreceptive to the insulin.

Insulin acts by enhancing glucose uptake into the peripheral tissues and inhibiting glycogen breakdown and glucose release from the liver. Insulin is the primary "key" to "unlocking" tissues for glucose uptake. There are, however, some tissues that do not require insulin for efficient uptake of glucose such as the liver itself and some of the nervous tissue

Glucagon

Glucagon is a catabolic hormone produced by the alpha cells in the islets of Langerhans within the pancreas. Cellular uptake of glucose (and resulting decrease in blood glucose) stimulates secretion of glucagon. Glucagon acts in diametrically opposite fashion to insulin and some of its functions include:

- It decreases the cellular uptake rate of glucose;

- Glucagon increases synthesis (from stored glycogen and from amino acids) and release of glucose from the liver, thereby raising blood glucose levels. It is sometimes used as injections for the treatment of severe insulin reactions;

- It increases the breakdown of fats and the formation of ketones and ketoacids. These are also potential energy sources;

In other words, glucagon opposes the actions of insulin; it stimulates lipolysis, ketogenesis, proteolysis, and gluconeogenesis. Interestingly, glucagon secretion is also stimulated by stress. The delicate balance between the antagonistic effects of insulin aud glucagon results in fine-tuning of the steady-state levels of blood glucose.

Glucagon results in the breakdown and use of stored glucose, fats and protein, while insulin couversely causes storage of the fuels. Insulin causes an anabolic metabolic action and glucagon a catabolic one. Insulin to glucagon ratio of 10 mg/l to 1 mg/l respectively is considered to be the switching point between the anabolic and catabolic metabolisms.

3.3.2 Blood sugar utilisation

Utilisation of blood sugar means the usage of glucose in the blood as energy source and therefore will have a net effect of lowering blood glucose concentrations. Insulin is required to enable the body to burn glucose for energy, in the absence of which fats are burned as energy source. Glucose is burned as fuel for basal metabolism in all tissues including the muscles, liver, adipose

tissue and nervous tissue, reducing blood glucose levels. The same processes occur at an accelerated pace when the body performs any action such as exercising.

An exception to the blood glucose-lowering rule of utilisation may occur in Type 1 diabetics where the absence of insulin prevents the utilisation of blood glucose, rendering it effectively unavailable. The body interprets this unavailability of insulin as a shortage of blood glucose (since little energy can be absorbed from the bloodstream) and therefore, control mechanisms are activated to raise blood sugar levels even further. This results in the reversed phenomenon of blood sugar levels increasing under these conditions, especially during exercise.

Stress is another stimulus that effects blood sugar concentrations. Stress, both short and long term, result in increasing blood glucose levels. This is due to the secretion of the retrieval hormone adrenalin under stressful conditions that raise blood glucose.

Storage of glucose, made available in the blood from fuel intake, causes the removal of the glucose from the blood and moves it into the storage facilities. This process has a blood glucose lowering effect. As in the case of glucose utilisation for energy, insulin is also required for "unlocking" the tissues for glucose (or other fuel types) storage. It has been established that without insulin, it is difficult for a human to gain weight due to ineffective storage. Storage of blood glucose in the main storage facilities happens as described previously.

3.4 The Glycaemic Index

The glycaemic index is a measure of the ability of 50g carbohydrate food to increase blood sugar levels after ingestion. The index compares the blood sugar response to a particular food with the body's reaction to pure glucose, which is given a value of 100. Neither pure protein nor pure fat has any substantial impact on blood glucose levels. [2]

3.4.1 Measurement procedure for GI

The measurement procedure for GI is as follows: A healthy person is required to fast for at least 6 to 10 hours prior to performing the test. This fasting ensures that any traces of glucose and effects of previous meals are negligible.

The next step is to ingest the reference food (in this case glucose). For the glucose reference 50 g of pure glucose (usually diluted in water for easier consumption) is used. Over the next two hours, blood samples are taken at 15-minute intervals during the first hour followed by two 30-minute intervals for the remaining hour. Blood sugar levels of the samples are measured in the laboratory and recorded. The result is a graph of blood sugar level plotted against elapsed time.

After a similar fasting period the procedure as described above is repeated. But, instead of ingesting pure glucose, the food for which the GI has to be calculated is eaten. The amount of food that has to be taken has to be the amount that contains exactly 50 g of carbohydrates. (In the case of potatoes, for example, 250 g of potato are required because that portion will yield 50 g of carbohydrates.) Again the blood sugar measurements are taken as described for the reference food.

GI is then defined as the fractional relationship (percentage) between the glycaemic responses of the measured food and the reference food. To relate the responses the area under the curves (AUC) are calculated for each test and compared by dividing the AUC of the test food by the AUC of the reference food. The calculation of the AUC for one of the tests is graphically presented in *Figure 9*.

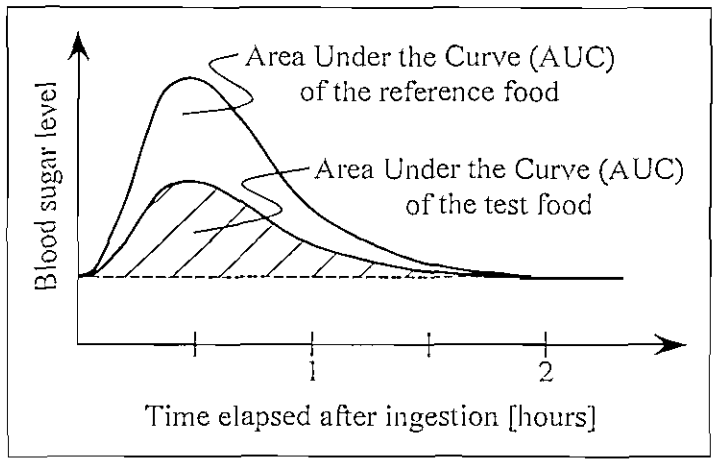


Figure 9: Measurement of AUC of the glucose response due to ingested CHO in order to determine the GI of the test food.

The test is repeated several times and with multiple test subjects to obtain an average value. To find the GI, the AUC of the test food is expressed as a percentage of the AUC of the reference food. ***Equation 6*** shows the final calculation of GI where AUC_{Food} is the area under the glucose response curve of the food in question (the test food) and $AUC_{Reference}$ is the area under the glucose response curve of the reference food (in this case pure glucose).

$$GI = \frac{AUC_{Food}}{AUC_{Reference}} \tag{6}$$

3.4.2 Limitations concerning GI

The application of GI unfortunately presents a few problems. These include the following:

- GI values are not related to food portion size. It is a property of the food but not the *amount* of the food.

  For example, a massive 1.3 kg of watermelon (containing only 8 g of carbohydrate per 150 g serving) has to be ingested to produce the same glycaemic response as 50 g of glucose powder. Because of its relatively high GI value of 72, it can be perceived by some people that watermelon is "bad" to eat. But, since the CHO content of watermelon is relatively small, a normal sized portion would produce totally acceptable blood sugar levels.

- GI values are based on average glycaemic responses measured in a number of different individuals. The problem is that there is often a significant variation in the measurements. Average GI values calculated are regarded by many as unscientific and therefore have little to contribute to general dietary planning and management. The reasons for the variances are not yet described to scientific satisfaction, and may be attributable to a host of metabolic and biochemical factors. However, as of yet the glycaemic response to GI-measured food yields acceptably repeatable results for individual test subjects.

Although generalised, GI values do provide some indication of relative variances to be expected when determining glycaemic response or energy utilisation in the human body. GI values therefore have a valid role to play in nutritional management.

3.5 Conclusion

Every human being is completely unique, with an endless amount of metabolic coefficients. These coefficients determine the energy conversion efficiency and include losses like energy needed for digestion, incomplete digestion, gas production etc. By applying the laws of thermodynamics and incorporating every predetermined metabolic coefficient the energy utilization of macronutrients can be determined. Every unique solution will be very time consuming, due to multiple tests being required. This approach is not a feasible obesity solution.

If the energy conversion efficiency is captured as one single metabolic measurement, then a more generic solution will be possible. The *way our blood glucose elevates with the consumption of food*, might just be the perfect energy conversion factor that we have been looking for.

The area under the blood sugar response curve (AUC) seems to be the trivial energy conversion factor for macronutrients in the human body. If the hypothesis holds true that the area under the blood sugar response curve is in linear relation to the *energy potential of that food*, then it is possible to determine the human energy homeostasis.

This literally means that carbohydrates with different GI values will have different available, or *metabolizable*, energy contents. If this is the case, then all previous attempts to address the

obesity epidemic with controlled energy intake are obsolete. Only by quantifying the *metabolizable* energy of food correctly, can a better obesity solution be found.

In Chapter 4 the shortcomings of the GI concept is incorporated into a more practical energy equation to better address the obesity crisis.

3.6 References

[1] Botha, C., "**The Human Energy System**", *Final project presented for in partial fulfilment of the requirements for the degree Doctoral of Engineering*, Mechanical Engineering, North West University, Potchefstroom, 2002

[2] Leeds, A. & Miller, JB., **The GI Factor**: *The Glycaemic Index Solutions*, Hodder & Stroughton, Australia. (1996)

4 DERIVATION OF ENERGY EQUATIONS

4.1 Energy conversion potential

Measurements with a bomb calorimeter suggest that energy of 4 kCal/g can be released from CHO when it is oxidised in pure oxygen as discussed in Chapter 3. Due to metabolic inefficiencies the human body converts less energy from ingested carbohydrates. Energy extracted from ingested carbohydrates is however converted into blood sugar energy.

This conversion process is difficult to measure in healthy persons because insulin enables storing and utilization of blood sugar during the conversion process. Type 1 diabetics cannot store the blood sugar energy due to the absence of insulin. The level to which diabetics' blood sugar levels rise should therefore give a good measure of the amount of blood sugar energy converted from the ingested CHO.

When equal amounts of glucose (GI=100) and fructose (GI=23) are ingested by a Type 1 diabetic the blood sugar response curve look like follows:

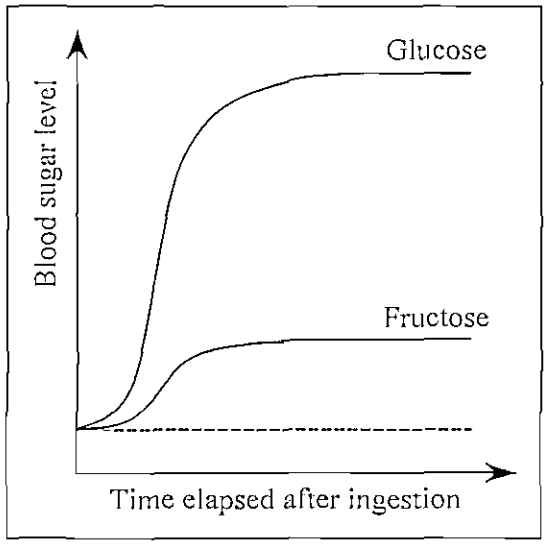


Figure 10: Blood sugar response curve for glucose and fructose in a Type 1 diabetic

Blood sugar response to glucose and thus the conversion of glucose into blood sugar energy is approximately four times more efficient than fructose. This proves that different carbohydrates release different amounts of metabolizable energy. Therefore, a new definition of GI is proposed, namely that GI provides the "energy conversion potential" of carbohydrates.

GI expressed as a percentage (%) can now be used to find the converted CHO energy potential (E_{CHO}, measured in kCal) for a mass (m_{CHO}, measured in g) that is available to the body. Since there are approximately 4 kCal of energy in 1 g of pure glucose, E_{CHO} can be approximated with *Equation 7*:

$$E_{CHO} = 4\frac{GI}{100}m_{CHO} = \frac{GI.m_{CHO}}{25} \tag{7}$$

If *Equation 7* is divided by m_{CHO} throughout, *Equation 8* is found.

$$\frac{E_{CHO}}{m_{CHO}} = \frac{GI}{25} \tag{8}$$

Equation 8 can now be used to calculate approximate values for typical energy contents available to the body from ingested carbohydrates. In *Error! Reference source not found.7* a few examples of typical GI values and their corresponding energy contents (E_{CHO}) per mass (m_{CHO}) values are shown.

Food	GI (%)	$\frac{E_{CHO}}{m_{CHO}}$ (kCal/g)
Glucose	100	4
Fructose	23	1
Apple	38	1.5
Table sugar	65	2.6
White bread	75	3
Whole-wheat bread	65	2.6

Table 7: Typical energy values in accordance to corresponding GI values.

From the list it is clear that effectively less energy is absorbed from the same amount ingested carbohydrates. The assumption that a calorie is a calorie is thus untrue.

4.2 Derivation of the ETS formula

Since only the carbohydrates in a meal have a significant effect on blood sugar levels, the assumption is made that the other two macronutrients, fat and protein, are not directly converted into blood sugar during digestion. This assumption hold true to a certain extent, since fat and protein digestion occurs significantly slower than that of CHO.

To derive the *ets* concept the amount of available blood sugar energy contained in a meal is considered. According to the above assumption, only CHO in a meal can provide blood sugar energy. The energy is then equal to the total amount of energy of the CHO contained in the meal and therefore also a function of the amount of CHO contained in the meal (m_{CHO}).

As mentioned earlier, when measured in a laboratory with processes such as bomb calorimeters, carbohydrates are found to release a certain maximum amount of energy per mass. This absolute amount of available energy is denoted as k_{CHO}. The total amount of available blood sugar energy from any meal (E_{CHO}) is then the total energy ($m_{CHO}\ k_{CHO}$) multiplied with the conversion potential (GI_{CHO}). This product is shown in ***Equation 9***:

$$E_{CHO} = GI_{CHO} m_{CHO} k_{CHO} \tag{9}$$

The next step is to relate the amount of energy from CHO in a meal to equivalent teaspoons sugar (*ets*). One *ets* (one teaspoonful of cane sugar) contains 5 g of carbohydrates. In other words the total amount of available energy from one *ets* is $5k_{CHO}$ kCal. Since the GI of sugar is 65, it follows from ***Equation 10*** that the energy that can be extracted from one teaspoon of cane sugar is:

$$E_{teaspoon\ \ sugar} = GI_{sugar} m_{teaspoon\ \ sugar} k_{CHO} = (65)(5)k_{CHO} = 325k_{CHO} \tag{10}$$

Equivalent teaspoons sugar, or *ets*, is now defined as *the fractional amount of blood sugar energy that can be extracted from any foodstuff, in relation to one teaspoonful of cane sugar*, expressed in *ets*. The equation for calculating the *ets* of any meal is

$$ets = \frac{E_{CHO}}{E_{teaspoon\ \ sugar}} = \frac{GI_{CHO} m_{CHO} k_{CHO}}{325 k_{CHO}} = \frac{GI_{CHO} m_{CHO}}{325} \qquad (11)$$

Equation 11 can now be used to calculate the *ets* value for any food with a known GI value according to the portion size.

The reasoning behind the formulation of *ets* as a measure of carbohydrate intake, is simple. People interested in glycaemic response prediction require a measure with which to relate any food, regardless of digestibility and portion size. By comparing foods with respect to the blood sugar energy they have available per portion provides a practical easy-to-use measure across any scope of foods.

If a certain food with a GI value of 50 is consumed, twice the mass of carbohydrate contained in that food will be required in order to result in the same blood glucose response as the reference food (pure glucose with a GI of 100). This same reasoning can be applied to any other food with known GI in order to calculate the mass of carbohydrate required for the equivalent glycaemic response compared to the reference. (Equivalent glycaemic response means an equivalent area under the glycaemic response curve, and not necessarily exactly the identical curve shape.)

The *ets* concept now provides a new method for determining insulin dosages for diabetics. Because foods with higher *ets* values will provide higher glycaemic responses, more insulin has to be injected for acceptable control. The specific amount of insulin will be discussed later, together with the discussion on differences in individuals. [1]

4.3 Verification of the equations

When food containing carbohydrates are ingested the body produces insulin to lower or utilise the blood sugar energy. The most well known methods for predicting insulin response due to food ingestion are CHO counting and the glycaemic index (GI) but with limited success. To

verify the ets equation it is necessary to prove that ingested ets can accurately predict the body's insulin response.

Let us first examine in more detail the quality of insulin predictions for the CHO and the GI methods using measurements by Lee and Wolever. These measurements give insulin response curves for different healthy test subjects ingesting different amounts of CHO (0 to 100 grams) with varying GI values (23 to 100).

The time integrals ($\int BI(t)dt$) of the Lee and Wolever blood insulin (BI) response curves for one subject are normalised and plotted against the amount of CHO consumed (***Figure 11***) and against the GI (***Figure 12***) of the ingested foods. Pearson's R^2-values were calculated for linearised trend fits through the plotted data. The R^2-values for the CHO and the GI methods were 0.603 and 0.558 respectively. For the CHO method the worst spread is at 50g CHO, namely a factor 12, while for GI at 65 the factor is close to three.

The need for a better insulin prediction method for CHO is obvious. Wolever and Bolognesi made a successful attempt. They developed an empirical model based on measurements in seven healthy subjects. Unfortunately, the resulting non-linear empirical equations have not found popular use, as they are difficult to use by the target market.

The easy-to-use ets method is therefore proposed for general usage. It is theoretically derived using energy balance techniques, namely the ingested CHO / blood sugar energy balance. A theoretical approach is preferred to an empirical one, since theory *inter alia* leads to better insight. The simple linear link between insulin response and ets is given by ***Equation 11.***

Let us now investigate the quality of insulin predictions by the ets method. The Lee and Wolever measurements are again used for the same test subject as in ***Figures 11*** and ***12***. The results are given in ***Figure 13***. The linear trend line for the ets method yields a R^2-value of 0.929, which is significantly better than those of the other methods.

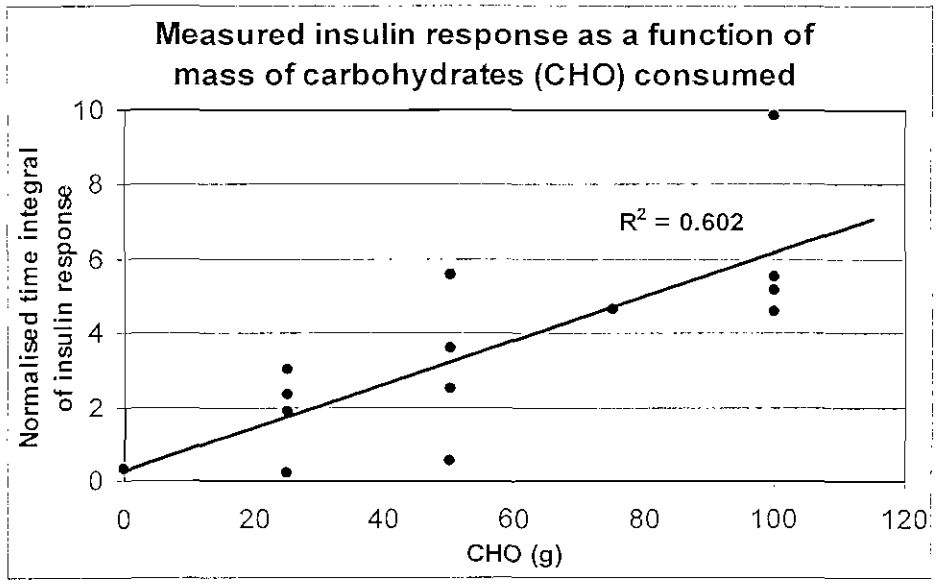


Figure 11: Linear best fit trend line and corresponding R^2-value for normalised values against CHO ingestion (one test subject).

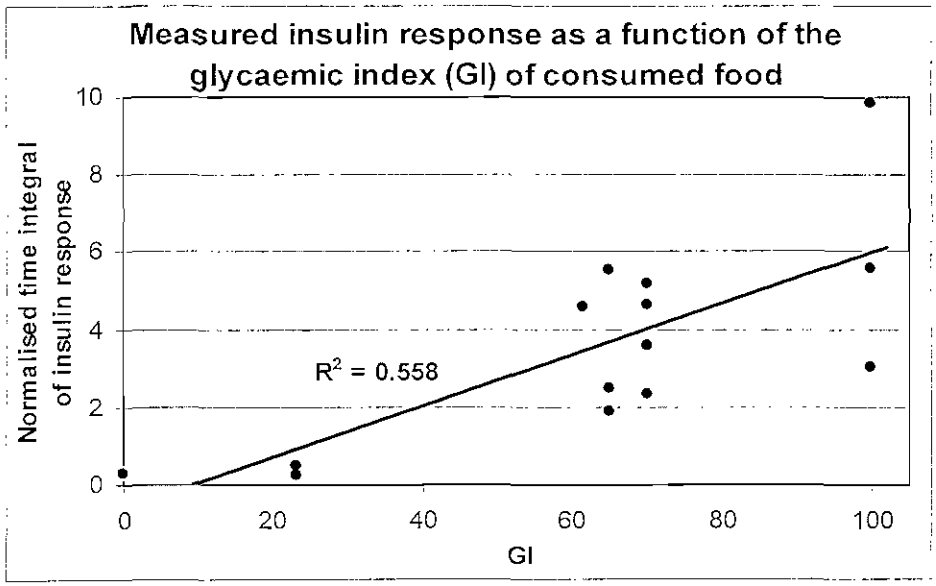


Figure 12: Linear best fit trend line and corresponding R^2-value for normalised values against GI values of ingested food (one test subject).

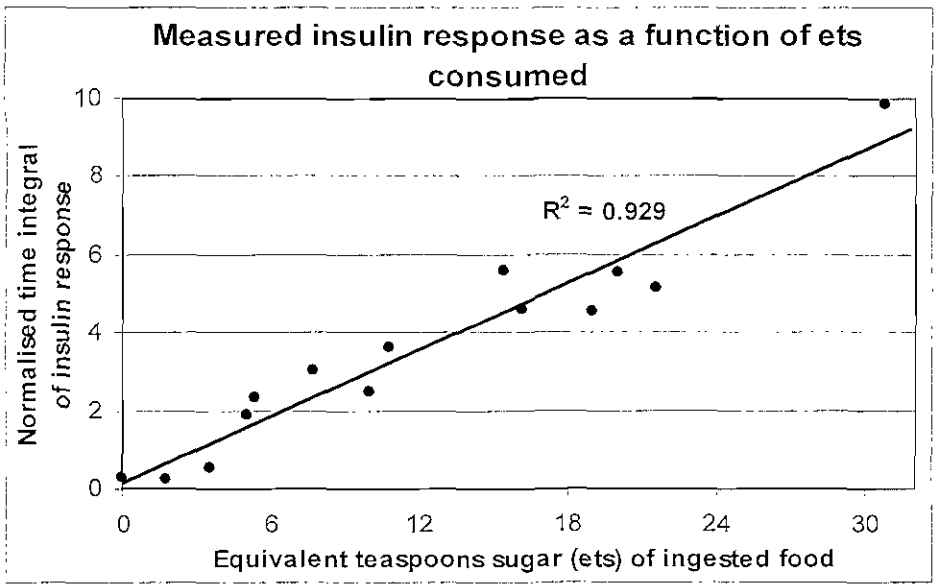


Figure 13: Linear best fit trend line and corresponding R^2-value for normalised values against ets values of ingested food (one test subject).

4.4 ETSCal as an obesity solution

Insulin promotes fat storage and prevents fat burning that causes obesity. *ets* intake can regulate obesity by controlling the insulin response of certain foods. A complete energy equation can now be constructed by adding ets, protein and fat energy values together.

Keeping in mind that $GI_{sugar} = 65$, thus having a metabolic conversion efficiency (η) of 0,65, the equivalent energy in one teaspoon sugar (*ets*) is 13kCal as calculated using ***Equation 4***:

$$E_{Teaspoon\,Sugar}\,[\text{kCal}] = \text{one } ets\,[\text{kCal}] = 0{,}65 \times 5 \times 4 = 13\,[\text{kCal}]. \quad (4)$$

Now that we have established the energy available from any CHO expressed in terms of *ets*, we can propose a new way of calculating total energy available to the body. We call the new energy value *ets* Cal, to avoid confusion with standard kCal. It is calculated by the following equation:

$$\text{ets Cal} = 13[\text{kCal/ets}] \times \text{ets}_{CHO} + 9[\text{kCal/g}] \times \text{Mass}_{Fat}[\text{g}] + 4[\text{kCal/g}] \times \text{Mass}_{Protein}[\text{g}] \quad (5)$$

If test results indicate that ets Cal is more representative of the metabolizable energy available from food, a better understanding will be gained of the energy homeostasis in humans. Only by quantifying ingested energy correctly, can obesity be controlled.

4.5 References

[1] Botha, C., "**The Human Energy System**", *Final project presented for in partial fulfilment of the requirements for the degree Doctoral of Engineering*, Mechanical Engineering, North West University, Potchefstroom, 2002

5 TESTING ETSCAL WITH CLINICAL TRIALS

5.1 Introduction

Measurements in a bomb calorie meter suggest that the energy that can be released from carbohydrates is approximately 4[kCal/g]. Obviously our bodies do not use the same system for energy conversion. Mathews *et al* have shown that the effective energy available from food might be different than currently believed. This implies that two different foods with the same calorie value might actually contain different amounts of energy.

The implication of using an energy quantification system that is incorrect, is far reaching. People on energy-restricted diets might consume more calories than they should; whilst others might be consuming less than they think they are. Diabetes management might also suffer as a result of this. If more or less energy is absorbed from carbohydrates than expected (in other words glucose absorbed into the blood), then insulin boluses might also be calculated incorrectly, causing several diabetes complications.

The purpose of this test is to determine if a relationship exists between the effective energy in food and the food's GI (while comparing foods that have the same calorie values). A secondary aim of this study is to show that calorie values of food do not necessarily represent the effective energy available from food. It is hoped that results of this clinical study will form the basis on which further research in this field can be done.

The new energy unit (namely ~~ets~~ Cal) for quantifying energy available from food was mathematically derived in Chapter 4. It takes into account various factors of the specific food being quantified. In many cases ~~ets~~ Cal values for food will be the same as their Calorie values while in other cases it might be less. ~~ets~~ Cal takes into account amongst other factors, the Calorie value as well as the GI of the food.

It was decided to conduct a clinical trial using Spraque-Dawley rats. The main reason for using animals is that their daily energy received from food can be carefully controlled in the laboratory. It is therefore possible to provide them with a homogenous diet. This allows the

researcher to provide the rat with a diet with a specific Glycaemic Index (GI), a certain mass CHO and consequently a different $_{ets}$Cal value.

5.2 Design of protocol for a rat model

In this part of the study, 11 groups of rats were used. Each group consisted of 10 and 12 Spraque-Dawley rats (n=10 and n=12). Groups where split into two trials due to capacity constraints at the UPBRC (University of Pretoria Biomedical Research Centre). Group 1 – 6 had 10 specimens and group 7 –11 had 12 specimens. Full-grown rats, older than twelve weeks and free of any chronical diseases were used for this study. Rats were assigned to the 11 groups on a random basis. Each rat was fed in a separate metabolic rat cage.

Performing power analysis and sample size estimation is an important aspect of experimental design. If the sample size (n) is to low, the experiment will lack the precision to provide reliable answers to the questions it is investigating. If the sample size is too large, time and resources will be wasted, often for minimal gain.

After consulting with Dr. P.J. Becker, an expert in biostatistics at the MRC (Medical Research Council), the statistical power for the study was found to be 95 using n=12 for 11 groups and the study utilizing n=10 and 11 groups will result in a power of 90 [1]. Both of these scenarios are for one-sided trend tests. It was assumed that the trend will be decreasing weight loss as $_{ets}$Cal is increased.

Rats were divided into groups so that the average weights were the same. Each group thus consisted of rats that might be more than 200g or less than 200g. In the case where both male and female rats were used, they were divided then equally throughout the groups.

Each group of rats was given a different diet and received an equivalent amount of Calories. The $_{ets}$Cal of the food that each group received did, however, differ. This means that all the rats in all the groups received an equivalent amount of Calories daily, but the $_{ets}$Cal of the food differed for rats that are not in the same group. The GI of the food given to the rats in the same group was, however, the same.

5.2.1 Energy requirements for Spraque-Dawley Rats

As energy requirements are related to metabolic weight, i.e. body weight $kg^{0.75}$, individual animals with different body weights will have different energy requirements. The estimated energy requirements for Spraque-Dawley Rats are related to metabolic body weight and metabolizable energy in the following way [2]:

-Maintenance requirement = 0.45 x body $weight^{0.75}$
-Growth requirements = 1.20 x body $weight^{0.75}$
-Pregnancy requirements = 0.60 x body $weight^{0.75}$
-Lactation requirements = 1.30 x body $weight^{0.75}$

The RDA Calorie value for each rat was normalized according to the initial weight of the full-grown rat, using the maintenance specification.

$$E_{RDA\_Maintenance} = 0.45 * (W_{Rat\,mal}[kg])^{0.75}[MJ/kg] \qquad [2]$$

With

E_{RDA}: Recommended daily allowance energy for rat
$W_{rat\ init}$: Initial weight of rat (at onset of trial)

This requirement is expressed as a daily intake of metabolizable energy in MJ; body weight is expressed in kg. It should be stressed that the estimate obtained assumes a minimum expenditure for energy for physical activity. Note: 1MJ = 1000kJ = 240kcal.

The daily diet of each rat (according to its group and initial weight) remained the same throughout the duration of the study except where this proved to be considerably detrimental to the health and/or well being of the rat. The ethical cut-off point for weight loss is 10% for Spraque-Drawley Rats as specified by the UPBRC [3],[4].

Table 8 gives a short description of the characteristics of the different foods:

Information of different foods used in clinical trial							
		Blood Glucose Response	Nutritional Information			Energy	
Group	Food	GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g	etsCal/100g
1	Barley	25	73	12	2	355	144
2	Chickpeas	33	46	20	10	348	230
3	Provita	49	70	11	10	420	271
4	Pronutro Flakes	64	88	5	1	348	254
5	Toasted Muesli	74	67	10	5	359	283
6	Corn Flakes	84	83	7	1	374	314
7	High Fibre Bran	43	45	13	3	261	156
8	Strawberry Pops	60	90	4	1	389	241
9	All Bran Flakes	69	59	14	3	322	246
10	Nutrific	80	66	13	3	346	290
11	Special-K	89	79	11	1	373	334

Table 8: Information of different foods used in clinical trial

5.3 Hypothesis

When Spraque-Dawley Rats are fed the same calories proportional to their weight (RDA) of different foods, *the change in weight will not be the same but in a linear relationship with the ets Cal value of that specific food.*

Table 9 shows for example that a 200g rat in any given group received the same RDA, but different weights of food, due to the different energy content of each food:

Different weight of food with the same RDA but different etsCal values							
		Energy		Weight of food a 200g Spraque Dawley Rat			
Group	Food	kCal/100g	etsCal/100g	Rat weight(g)	RDA (kCal)	Food/Rat (g)	etsCal/Rat
1	Barley	355	144	200	32.30	9.09	13.09
2	Chickpeas	348	230	200	32.30	9.28	21.39
3	Provita	420	271	200	32.30	7.69	20.85
4	Pronutro Flakes	348	254	200	32.30	9.28	23.58
5	Toasted Muesli	359	283	200	32.30	9.01	25.52
6	Corn Flakes	374	314	200	32.30	8.63	27.10
7	High Fibre Bran	261	156	200	32.30	12.38	19.35
8	Strawberry Pops	389	241	200	32.30	8.30	20.01
9	All Bran Flakes	322	246	200	32.30	10.03	24.66
10	Nutrific	346	290	200	32.30	9.34	27.09
11	Special-K	373	334	200	32.30	8.66	28.94

Table 9: RDA and etsCal values for a typical 200g Spraque-Dawley Rat

Table 9 shows that all Groups received the same amount of calories. Conventional theory implies that any change in weight, if any, of these groups should be the same for all the groups. This seems to be a very logical conclusion. The food energy values for the rats were calculated at slightly less than the prescribed RDA Calorie value. This was to ensure that the rats ate all the food they were given. Because of this, one would expect all the rats to lose weight over the duration of the study.

Taking into account that each group received the same amount of calories, conventional theory implies that the average weight loss per group should be the same for all the groups. This is shown in Figure 14a.

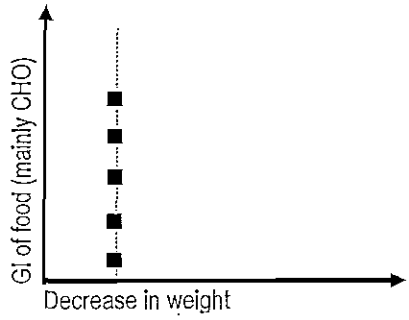


Figure 14a:

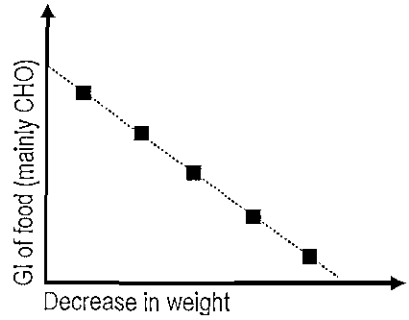


Figure 14b:

Figure 14: Conventional belief compared to the stated hypothesis

The Hypothesis states the opposite. ***Figure 14b*** shows that according to the hypothesis, food with high GI values will result in a smaller reduction in weight, compared to food with lower GI values (although the amount of calories for each group is constant.) Although ***Figure 14b*** shows an indirect linear relationship between GI of the food and decrease in weight, it might not be exactly the case; but it is proposed that, in general, food with *high* GI values will result in a *smaller* reduction in weight compared to food with lower GI values.

This part of the study tested for a trend between the GI of the diet and weight loss. In other words, between the GI of the diet and the effective energy available from this diet (which might be different from the conventional measure of energy). The GI of the diets for groups 1 to 11 varied from a low of 33 to a high of 89.

5.4 Experimental procedures

This clinical study was conducted over a period of four weeks at the UPBRC (University of Pretoria Biomedical Research Centre.

The following guidelines were prescribed at the onset of the trial:

- sufficient time (1 week) should be allowed for the Spraque-Dawley Rats to acclimatize;
- rats should be randomly assigned to the 11 groups with 10 rats per group,
- each rat should be assigned a number and its cage should be carefully marked with its number (Rat ID) and group number;
- each rat should be weighed and this information should be recorded in the spreadsheets provided; and
- the weight of food that each rat receives is calculated according to the initial weight of the rat (using the spreadsheet provided) and will remain constant throughout the duration of the study.

The following **daily guidelines** were adhered to during the clinical trial:

- the food for each rat should be carefully weighed according to the calculated tables provided
- food should be supplied daily on the same time (early in the morning) to each rat;
- information should be recorded to state whether each rat eats all the food supplied or not;
- water should be supplied on an as-required basis.

The following **weekly guidelines** were adhered to:

- weekly weight measurements should be done in the afternoon
- water should be removed the night before weight measurements are taken, to prevent any additional weight due to excessive water consumption
- rats should be weighed on the last day of each week and this information recorded;
- measurements should be accurately captured to the second decimal number;

The following **guidelines** were prescribed at the **termination** of the trial:

- rats should be weighed and this information recorded; and

- rats should either be euthanized (an overdose of CO2 and cremated) or rehabilitated for another study if the need existed.

All statistical analyses, including regression, were performed on the data recorded following each part of the clinical study. Only the raw data in a spreadsheet format was supplied to the researcher.

5.5 Conclusion

Although Spraque-Dawley rats are not fully representative of human beings, they are also omnivores with very similar metabolic functions and nutritional requirements as humans. Not only where foods chosen with different ~~ets~~ Cal values, but also foods with easily available nutritional information and foods that were at least palatable. Most cereals and legumes fulfil these requirements.

If the test results proved the hypothesis to be correct, if would certainly justify a full study on humans. Therefore, all weight measurements were done very accurately and the test environment was kept as homogeneous as possible, to ensure any changes in weight are due to the single control variable (~~ets~~ Cal) and not any other.

5.6 References

[1] Dr. Becker, P.J.: MSc (UP) PhD (UNISA), **The Medical Research Council**, Biostatistics Unit, *Personal Communication*, 22 February 2005. 012 339 8519

[2] Clarke H E, Coats M E, Eva J K, Ford D J, Milner C K. O'Donoghue P N, Scott P P, Ward R J. **Dietary standards for laboratory animals: report of the Laboratory Animals Centre Diets Advisory Committee.** Lab Anim 1977;11: 1-28.

[3] Mr. Smuts, M.P.:**Projects and Facility Manager, University of Pretoria Biomedical** Research Centre (UPBRC); *Personal Communication*, 1 February 2005. 012 529 8388

[4] Dr. Auer, R.: Veterinary Care, **University of Pretoria Biomedical Research Centre (UPBRC);** *Personal Communication*, 1 February 2005. 012 529 8388

6 VERIFICATION OF CLINICAL TRIAL DATA

6.1 Data analysis

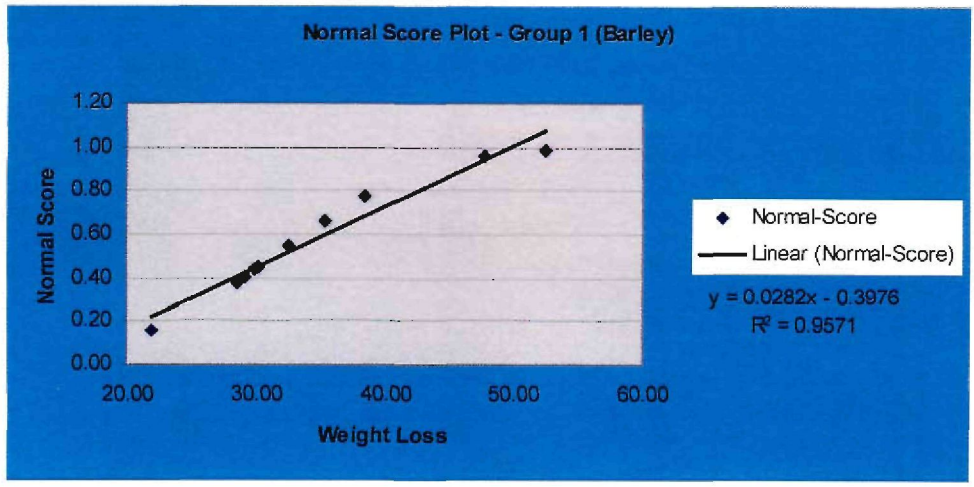
A pilot study at the University of Pretoria employed the following approach to the statistical analysis:

After the data capturing process, the average weight loss was plotted against three other variables for each group for comparison. Firstly, a relationship between the Glycaemic Index (GI) and weight loss was determined. (The GI concept is widely used in dietary science, but still lacks the scientific merit and accuracy necessary for satisfactory bolus predictions). Thirdly, the author compared the newly developed –ẽts Cal concept against the weight loss of the rats. This comparative analysis proved that –ẽts Cal equation had a far better regression when compared to weight loss, than GI.

The weight loss of each individual rat in the same group with n=10 was plotted on a "Normal Score Plot" to determine the normality of the data. The more linear the graph between the Normal Score and the weight loss, the better normal distribution function can be drawn up from the data.

A good "Normal Score Plot" will give a regression of 0.995-0.998 of normality, whereas a regression of 0.7-0.8 indicates little normality and could result in difficulty to repeat the study [1].

It was important to make provision for a sick rat that might cause extremities in the weight loss data. Such an extremity would jeopardise the integrity of the data and the hypothesis and should therefore be rejected. Here a quality assurance analysis principle was used, namely six sigma (6σ). If a data point did not fall within the UCL (Upper Control Limit) and LCL (Lower Control Limit), then that data point was rejected and the ANOVA (Analysis of Variance) started all over again. Where the UCL = $\mu + 3\sigma$ and LCL = $\mu - 3\sigma$. From the ***Figure 15*** presented below one can see that data points 1, 3 and 9 were rejected.



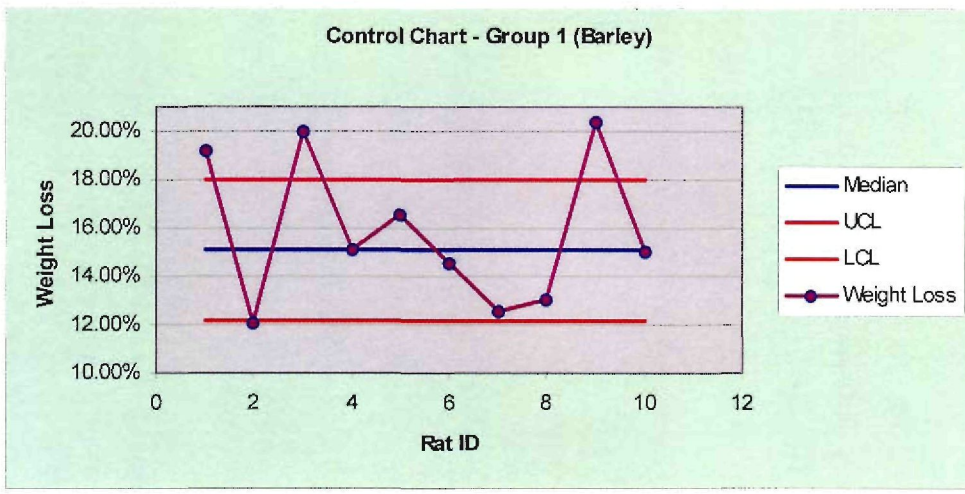


Figure 15: Example of a Normal Score Plot and a Control Chart

Only when this was done for every group, was the data copied into the final graph, which compares weight loss against the new –*ets* Cal equation.

6.2 Verification of the new ETSCal energy equation

After the data analysis of the individual groups, the data was plotted on a Weight Loss – –*ets* Cal relationship chart. The –*ets* Cal per rat was normalized to –*ets* Cal/100g rat. Due to

the energy restricted design of the experiment, one would expect a negative gradient for this graph.

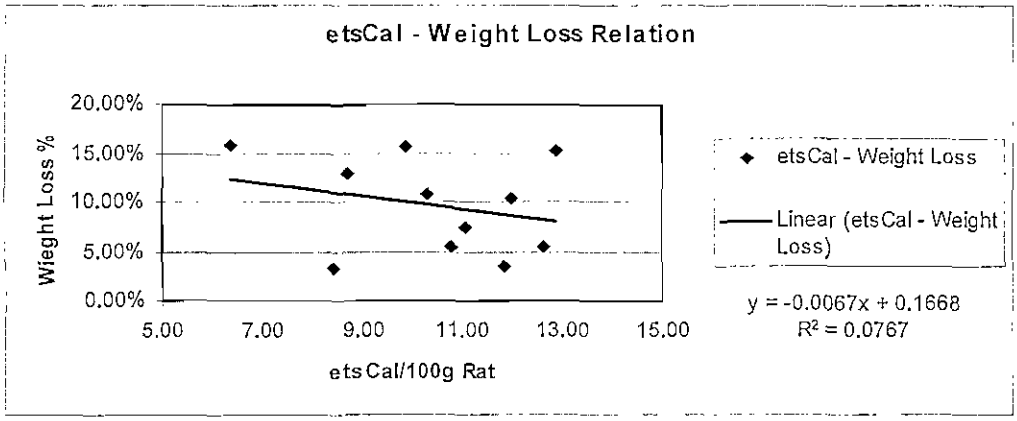


Figure 16: The correlation between ets Cal and weigh loss

With a regression of 0.0767 it is hard to come to any conclusion, other than that there exists no relationship between ingested ets Cal and the change in the weight of Spraque-Dawley rats. Group 6 (Kellogg's High Fibre Bran) and Group 7 (Kellogg's Corn Flakes) seem to represent two extremities that, if removed from the same graph, creates a better regression of $R^2 = 0.5929$.

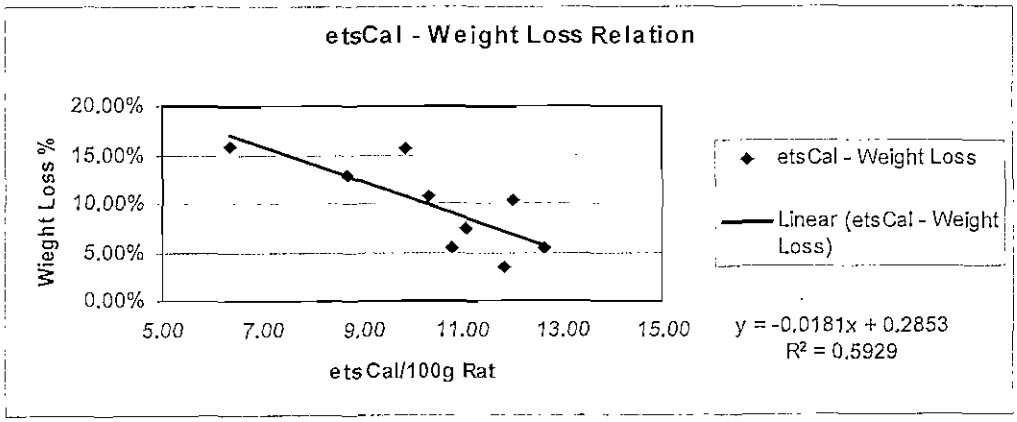


Figure 17: The correlation between ets Cal and weight loss when High Fibre Bran and Corn lakes are removed

Kellogg's High Fibre Bran can be differentiated through the fact that it contains the highest percentage of dietary fibre at 27g per 100g. (This should be further investigated, regarding the energy content of dietary fibre). Kellogg's Corn Flakes have a very high GI of 84 and consequently one of the highest ets Cal values per 100g. The cause of the Kellogg's Corn

Flakes high percentage of weight loss (15.22%) is uncertain and will be discussed later in the study. This also exceeded the cut-off point for weight loss according to the UPBRC, and the study was terminated due to animal ethics.

6.3 Fibre analysis

The equivalents of dietary protein, fat, and available carbohydrates as fuels for maintenance (kJ metabolizable energy) are known from classical experiments and are similar across species; that for dietary fibre or NSP (non-starch polysaccharide) is as yet undetermined

Normally, dietary fibre is not considered as an energy source for humans [2],[3]. This is based on the inability of the human gut to digest the material and absorb the metabolites. Nowadays, carbohydrate energy profiles are based on digestible carbohydrate, resistant starch and oligofructans, which are only attached in the lower cut by colonic bacteria that all form part of indigestible dietary fibre. There are also non-starch polysaccharides associated with the cellulosic fibre, but they are inaccessible to the enzymes. [4]

The data labels of total energy content on food packages are so determined by adding the energy content on individual macronutrients. For example, the measurements for Kellogg's High Fibre Bran on the side of the box will read: Energy = 1097kJ, Protein = 13g/100g, Carbohydrates = 45g/100g, Total Fat = 3g/100g and Fibre = 27g/100g. With the total energy (TE) calculated as follows:

$$\begin{aligned} TE/100g &= E(\text{Protein}) + E(\text{Carbohydrates}) + E(\text{Fat}) \\ &= (4\text{kCal})(13) + (4\text{kCal})(45) + (9\text{kCal})(3) \\ &= 259\text{kCal or } 1097\text{kJ} \end{aligned}$$

No energy value is allocated to dietary fibre. Assuming previous belief that NSP's have no energy value, then a bomb calorimeter measurement of Kellogg's All-Bran Flakes should yield a value close to 1097kJ.

Digital Data Systems (pty) Ltd performed all the bomb calorimeter measurements for all the test foods in this study using a CAL ^{2k} combustion calorimeter. The full report on sample preparation, spiking and analysis is given in Appendix C for each food type. The following Table summarises the results:

Test Foods	GI	Labelled kJ Value	Calorific kJ Value	Percentage kJ difference	Labelled Fibre Content
Barley	33	1448	1616	10%	10
Chickpeas	33	1450	1787	19%	16
Provita	49	1751	1888	7%	3
Pronutro Flakes	64	1451	1672	13%	11
Toasted Muesli	74	1494	1798	17%	11
Corn Flakes	84	1560	1664	6%	3
High Fibre Bran	43	1097	1781	38%	27
Strawberry Pops	60	1635	1656	1%	1
All Bran Flakes	69	1352	1725	22%	17
Nutrific	80	1454	1707	15%	10
Special-K	89	1567	1693	7%	4

Table 10: Calorific values of test foods

The difference in the energy content on the labels and the actual calorific value is perfectly correlated to the percentage of fibre in the test food with R^2=0.9555 as seen in Figure 18:

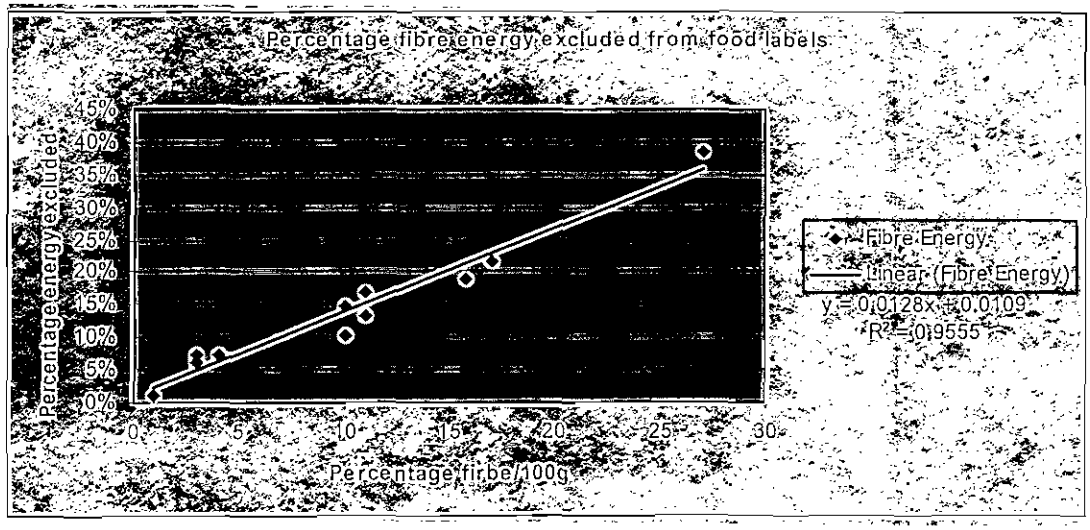


Figure 18 : Percentage of fibre energy excluded from food labels

The fact that Kellogg's High Fibre Bran with the highest percentage of fibre (27/100g) yielded the lowest tempo of weight reduction makes it hard to believe that fibre is not fermentable with no metabolizable energy. If fibre contains metabolizable energy contrary to previous belief, then

how can it be accounted for in the ets Cal energy equation? When the GI is compared to the percentage of fibre in the test foods, the correlation looks as follows:

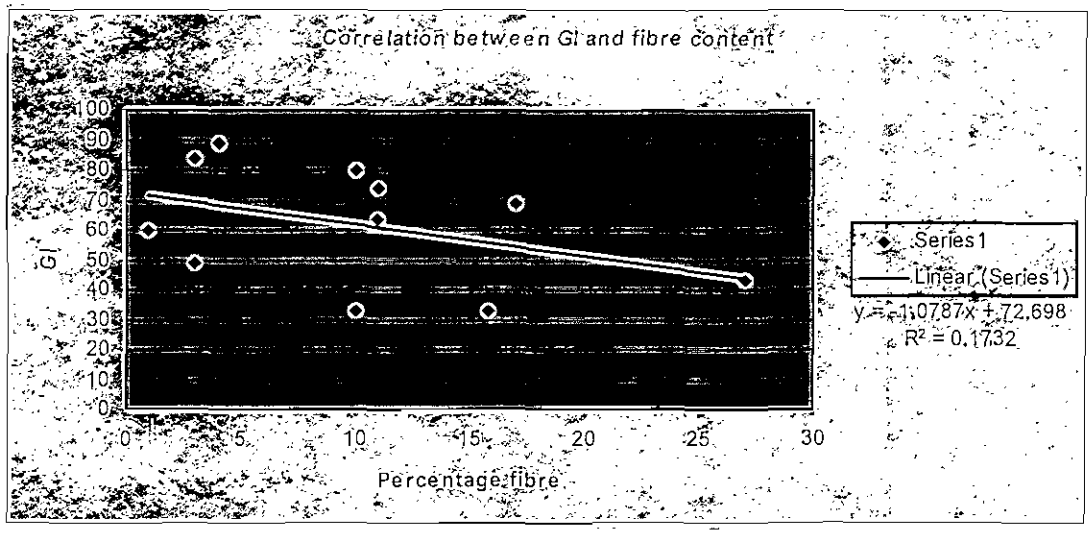


Figure 19: The Correlation between GI and the fibre content for the test foods

Although the GI correlates very badly to the percentage of fibre ($R^2 = 0.1732$) in scientific measure, there is still a slight tendency for low GI foods to contain a high percentages of fibre. It is therefore concluded that fibre should be added to the total amount of carbohydrates in the ets Cal equation. This new energy equation is now called ets CalFibre and tested with the same clinical trial data for comparison between ets Cal.

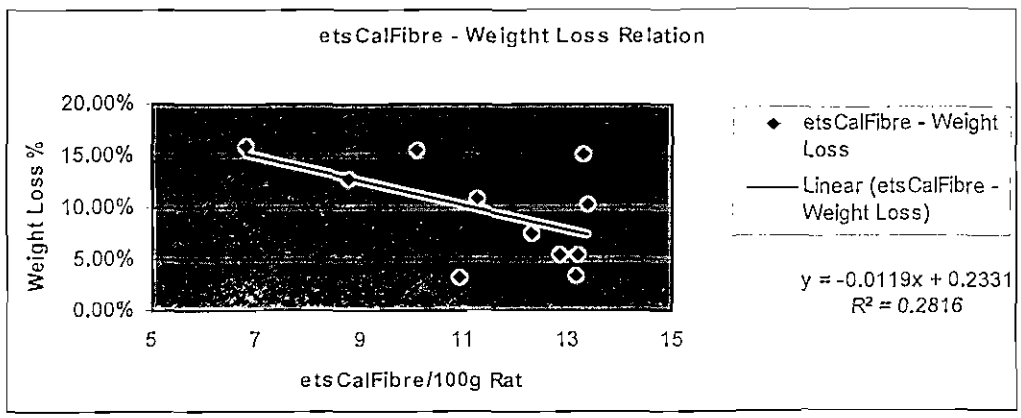


Figure 20: The correlation between ets CalFibre and weight loss

ets CalFibre has a definite improvement over ets Cal from $R^2 = 0.0767$ to $R^2 = 0.2816$ when correlated to weight loss. Once again when Kellogg's High Fibre Bran and Kellogg's Corn Flakes are removed from the group a more meaningful relationship of $R^2 = 0.6912$ exists between ets CalFibre and weight loss.

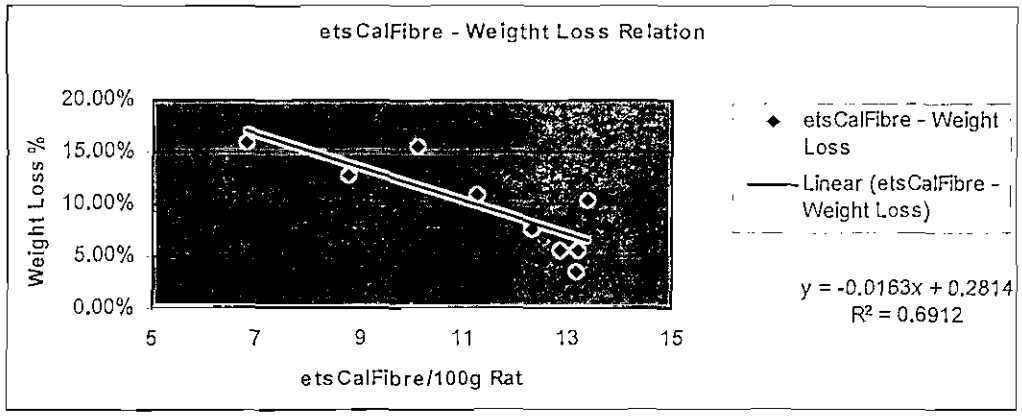


Figure 21: The correlation between ets CalFibre and weight loss when High Fibre Bran and Corn Flakes are removed

The behaviour of Kellogg's High Fibre Bran and Kellogg's Corn Flakes must be attributed to an unknown characteristic and this would justify further investigation. The Normal Score Plot graph for Kellogg's High Fibre Bran reveals little linearity of $R^2 = 0.6362$, indicating that there is very little process control. This could be caused by inaccurate measurements or possible health problems with the particular group. For the purpose of this study, the two extremities were rejected from the sample population

6.4 References

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[3] Southgate DAT, Durnin JVGA, **Calorie conversion factors: an experimental reassessment of the factors used in the calculations of the energy value of human diets.** Br J Nutr 1970;24:517-35.

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7 CONCLUSION AND RECOMMENDATIONS

7.1 Summary of contributions

7.1.1 A linear relationship was found between the ℰts CalFibre values of a food containing CHO and the % mass loss with a resulting Pearson's R^2 value of 0.69. This shows that the ℰts CalFibre equation is more representative of the energy conversion of CHO in a body than the constant 4kCal/g historically used.

7.1.2 By establishing the ℰts CalFibre energy equation,

$$\text{ℰts CalFibre} = 13[\text{kCal/ℰts}] \times \text{ℰts}_{CHO} + 9[\text{kCal/g}] \times \text{Mass}_{\text{Fat}}[\text{g}] + 4[\text{kCal/g}] \times \text{Mass}_{\text{Protein}}[\text{g}]$$

the total amount of metabolizable energy from carbohydrates can now be calculated. Now that doctors and dieticians know the exact amount of energy that their patients consume they can better regulate their weight by prescribing the right amount of ℰts. This capability brings scientists and physiologists one step closure to a total obesity solution.

7.1.3 The ℰts CalFibre energy equation does not only benefit people trying to control their weight but also Type 1 diabetics trying to control their blood sugar. This is because the ℰts concept is based on predicting insulin response from ingested carbohydrates to determine the available metabolizable energy. Type 1 diabetics can now accurately bolus the right amount of insulin based on ingested ℰts to prevent hyperglycaemia.

7.2 Recommendation for further work

This study focussed mainly on the characteristics of carbohydrates to narrow down the problem of energy homeostasis and, consequently, obesity. To design and built a complete model to simulate the human energy balance, more research should go into characterising triglycerides

and amino acids. Defining a conversion efficiency for triglycerides and amino acids will make for a more complete metabolizable energy equation.

The inclusion of fibre with carbohydrates definitely improves the linearity of ~~ets~~ CalFibre against weight variation but further investigation should be done to verify that carbohydrates and NSP (non-starch polysaccharides) have the same conversion efficiency.

APPENDIX A : WEIGHT LOSS DATA

Group 1: Barley			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			25.00	73.48	12.48	2.30	355.44			
Rat ID	Weight	RDA	Barley/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
1	184.34	30.38	8.55	149	35.34	19.17%	0.43	0.67	12.32	1.57
2	181.96	30.09	8.47	160	21.96	12.07%	-1.01	0.16	12.20	1.56
3	192.49	31.39	8.83	154	38.49	20.00%	0.77	0.78	12.72	1.62
4	193.17	31.47	8.85	164	29.17	15.10%	-0.24	0.41	12.76	1.63
5	196.52	31.88	8.97	164	32.52	16.55%	0.12	0.55	12.92	1.65
6	205.96	33.02	9.29	176	29.96	14.55%	-0.15	0.44	13.39	1.71
7	241.20	37.17	10.46	211	30.20	12.52%	-0.12	0.45	15.07	1.92
8	218.51	34.52	9.71	190	28.51	13.05%	-0.31	0.38	13.99	1.78
9	257.45	39.03	10.98	205	52.45	20.37%	2.27	0.99	15.82	2.02
10	317.77	45.71	12.86	270	47.77	15.03%	1.76	0.96	18.53	2.36
Total	**2189.37**	**344.65**	**96.97**	**1843.00**	**346.37**				**139.73**	**17.81**
Average	**218.94**	**34.47**	**9.35**	**184.30**	**34.64**	**15.84%**			**13.97**	**1.78**
10				**Median**	**31.36**	**15.07%**				
				Variance	**86.59**	**0.09%**				
				Std.Deviation	**9.31**	**3.08%**				
				UCL		**17.99%**				
				LCL		**12.15%**				

% Weight Loss	15.84%
etsCal/100g Rat	6.38

GL/100g Rat	0.8135927

Group 2: Chickpeas			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			33.00	46.10	19.90	10.00	348.00			
Rat ID	Weight	RDA	Chickpeas/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
11	180.6	29.92	8.60	163	17.60	9.75%	-1.48	0.07	19.81	1.31
12	180.12	29.86	8.58	162	18.12	10.06%	-1.42	0.08	19.77	1.31
13	186.89	30.70	8.82	162	24.89	13.32%	-0.70	0.24	20.33	1.34
14	188.29	30.87	8.87	159	29.29	15.56%	-0.22	0.41	20.44	1.35
15	197.37	31.98	9.19	175	22.37	11.33%	-0.97	0.17	21.18	1.40
16	209.46	33.44	9.61	178	31.46	15.02%	0.01	0.50	22.14	1.46
17	246.57	37.79	10.86	216	30.57	12.40%	-0.08	0.47	25.03	1.65
18	246.43	37.77	10.85	223	23.43	9.51%	-0.85	0.20	25.01	1.65
19	327.52	46.76	13.44	302	25.52	7.79%	-0.63	0.27	30.96	2.04
20	314.96	45.41	13.05	303	11.96	3.80%	-2.08	0.02	30.07	1.98
Total	**2278.21**	**354.50**	**99.73**	**2043.00**	**235.21**				**234.75**	**15.50**
Average	**227.82**	**35.45**	**9.87**	**204.30**	**23.52**	**10.85%**			**23.48**	**1.55**
10				**Median**	**24.16**	**10.70%**				
				Variance	**39.01**	**0.12%**				
				Std.Deviation	**6.25**	**3.51%**				
				UCL		**14.03%**				
				LCL		**7.37%**				

% Weight Loss	10.85%
etsCal/100g Rat	10.30

GL/100g Rat	0.6802267

Group 3: Provita			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			49.00	70.41	11.39	9.74	420.24			
Rat ID	Weight	RDA	Provita/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
21	176.42	29.40	7.00	140	36.42	20.64%	0.54	0.71	18.97	2.41
22	196.25	31.84	7.58	161	35.25	17.96%	0.42	0.66	20.55	2.61
23	196.73	31.90	7.59	170	26.73	13.59%	-0.50	0.31	20.59	2.62
24	193.17	31.47	7.49	174	19.17	9.92%	-1.31	0.10	20.31	2.58
25	197.21	31.96	7.61	163	34.21	17.35%	0.31	0.62	20.63	2.62
26	242.22	37.29	8.87	202	40.22	16.60%	0.95	0.83	24.07	3.06
27	283.86	42.00	9.99	239	44.86	15.80%	1.45	0.93	27.11	3.45
28	306.63	44.50	10.59	261	45.63	14.88%	1.53	0.94	28.72	3.65
29	324.35	46.42	11.05	276	48.35	14.91%	1.83	0.97	29.96	3.81
30	317.84	45.72	10.88	271	46.84	14.74%	1.66	0.95	29.51	3.75
Total	**2434.68**	**372.50**	**104.80**	**2057.00**	**377.68**				**240.41**	**30.58**
Average	**243.47**	**37.25**	**8.64**	**205.70**	**37.77**	**15.64%**			**24.04**	**3.06**
10				**Median**	**38.32**	**15.36%**				
				Variance	**88.99**	**0.08%**				
				Std.Deviation	**9.43**	**2.85%**				
				UCL		**18.06%**				
				LCL		**12.65%**				

% Weight Loss	15.64%
etsCal/100g Rat	9.87

GL/100g Rat	1.256089

Group 4: Pornutro Flakes			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			64.00	88.00	5.00	1.00	348.24			
Rat ID	Weight	RDA	Pronutro/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
31	172.79	28.94	8.31	154	18.79	10.87%	-1.35	0.09	21.13	4.68
32	182.49	30.15	8.66	163	19.49	10.68%	-1.28	0.10	22.02	4.88
33	183.55	30.29	8.70	171	12.55	6.84%	-2.02	0.02	22.11	4.90
34	195.39	31.74	9.11	186	9.39	4.81%	-2.36	0.01	23.18	5.13
35	202.55	32.61	9.36	186	16.55	8.17%	-1.59	0.06	23.81	5.27
36	295.27	43.26	12.42	275	20.27	6.86%	-1.19	0.12	31.59	7.00
37	298.49	43.61	12.52	272	26.49	8.87%	-0.52	0.30	31.85	7.05
38	343.16	48.42	13.90	321	22.16	6.46%	-0.99	0.16	35.36	7.83
39	331.79	47.21	13.56	314	17.79	5.36%	-1.46	0.07	34.48	7.64
40	311.07	44.98	12.92	292	19.07	6.13%	-1.32	0.09	32.85	7.28
Total	**2516.55**	**381.23**	**107.26**	**2334.00**	**182.55**				**278.37**	**61.65**
Average	**251.66**	**38.12**	**10.73**	**233.40**	**18.26**	**7.51%**			**27.84**	**6.17**
10				**Median**	**18.93**	**6.85%**				
				Variance	**22.65**	**0.04%**				
				Std.Deviation	**4.76**	**2.09%**				
				UCL		**8.84%**				
				LCL		**4.86%**				

% Weight Loss	7.51%
etsCal/100g Rat	11.06

GL/100g Rat	2.4499787

Group 5: Toasted Muesli			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			74.00	67.00	10.00	5.00	358.56			
Rat ID	Weight	RDA	Muesli/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
41	158.96	27.19	7.58	128	30.96	19.48%	-0.04	0.48	21.48	3.76
42	187.34	30.75	8.58	171	16.34	8.72%	-1.61	0.05	24.30	4.25
43	191.75	31.30	8.73	169	22.75	11.86%	-0.93	0.18	24.73	4.33
44	204.49	32.84	9.16	179	25.49	12.47%	-0.63	0.26	25.95	4.54
45	212.46	33.80	9.43	200	12.46	5.86%	-2.03	0.02	26.71	4.67
46	266.54	40.06	11.17	237	29.54	11.08%	-0.20	0.42	31.66	5.54
47	307.7	44.62	12.44	281	26.70	8.68%	-0.50	0.31	35.26	6.17
48	326.18	46.61	13.00	298	28.18	8.64%	-0.34	0.37	36.83	6.45
49	319.89	45.94	12.81	288	31.89	9.97%	0.06	0.52	36.30	6.35
50	328.41	46.85	13.07	304	24.41	7.43%	-0.75	0.23	37.02	6.48
Total	**2503.72**	**379.96**	**106.90**	**2255.00**	**248.72**				**300.23**	**52.54**
Average	**250.37**	**38.00**	**10.32**	**225.50**	**24.87**	**10.42%**			**30.02**	**5.25**
10				**Median**	**26.10**	**9.35%**				
				Variance	**39.37**	**0.14%**				
				Std.Deviation	**6.27**	**3.77%**				
				UCL		**12.92%**				
				LCL		**5.77%**				

% Weight Loss	10.42%
etsCal/100g Rat	11.99

GL/100g Rat	2.0984625

Group 6: Corn Flakes			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			84.00	83.00	7.00	0.80	374.40			
Rat ID	Weight	RDA	Corn Flakes/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
51	174.52	29.16	7.79	149	25.52	14.62%	-0.63	0.27	24.46	5.43
52	190.72	31.17	8.33	167	23.72	12.44%	-0.82	0.21	26.15	5.80
53	168.82	28.44	7.60	143	25.82	15.29%	-0.60	0.28	23.86	5.30
54	171.97	28.84	7.70	139	32.97	19.17%	0.17	0.57	24.19	5.37
55	176.01	29.35	7.84	143	33.01	18.75%	0.18	0.57	24.62	5.47
56	299.87	43.76	11.69	266	33.87	11.29%	0.27	0.61	36.71	8.15
57	222.05	34.94	9.33	185	37.05	16.69%	0.61	0.73	29.31	6.51
58	336.47	47.71	12.74	282	54.47	16.19%	2.48	0.99	40.03	8.88
59	324.93	46.48	12.41	290	34.93	10.75%	0.38	0.65	38.99	8.66
60	307.14	44.56	11.90	255	52.14	16.98%	2.23	0.99	37.38	8.30
Total	**2372.50**	**364.41**	**102.52**	**2019.00**	**353.50**				**305.70**	**67.86**
Average	**237.25**	**36.44**	**9.49**	**201.90**	**35.35**	**15.22%**			**30.57**	**6.79**
10				**Median**	**33.44**	**15.74%**				
				Variance	**109.62**	**0.09%**				
				Std.Deviation	**10.47**	**2.94%**				
				UCL		**18.53%**				
				LCL		**12.95%**				

% Weight Loss	15.22%
etsCal/100g Rat	12.89

GL/100g Rat	2.8602903

Group 7: High Fibre Bran			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			43.00	45.00	13.00	3.00	261.00			
Rat ID	Weight	RDA	High Fibre/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
1	222.69	35.01	13.41	208	14.69	6.60%	-1.79	0.04	20.98	2.60
2	235.27	36.48	13.98	229	6.27	2.67%	-2.70	0.00	21.86	2.70
3	248.92	38.06	14.58	226	22.92	9.21%	-0.91	0.18	22.81	2.82
4	241.41	37.20	14.25	239	2.41	1.00%	-3.11	0.00	22.29	2.76
5	249.21	38.09	14.60	237	12.21	4.90%	-2.06	0.02	22.83	2.82
6	255.79	38.85	14.88	242	13.79	5.39%	-1.89	0.03	23.28	2.88
7	405.97	54.93	21.05	410	-4.03	-0.99%	-3.80	0.00	32.91	4.07
8	405.19	54.85	21.01	405	0.19	0.05%	-3.35	0.00	32.87	4.07
9	427.7	57.12	21.88	430	-2.30	-0.54%	-3.62	0.00	34.23	4.23
10	436.37	57.98	22.22	421	15.37	3.52%	-1.72	0.04	34.75	4.30
11	454.35	59.77	22.90	443	11.35	2.50%	-2.15	0.02	35.81	4.43
12	511.3	65.30	25.02	484	27.30	5.34%	-0.44	0.33	39.13	4.84
Total	**4094.17**	**573.64**	**161.39**	**3974.00**	**120.17**				**343.74**	**33.26**
Average	**341.18**	**47.80**	**16.63**	**331.17**	**10.01**	**3.30%**			**28.65**	**3.33**
12				**Median**	**11.78**	**3.09%**				
				Variance	**95.82**	**0.10%**				
				Std.Deviation	**9.79**	**3.12%**				
				UCL		**5.79%**				
				LCL		**0.39%**				

% Weight Loss	3.30%
etsCal/100g Rat	8.40

GL/100g Rat	0.9747308

Group 8: Strawberry Pop's			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			60.00	90.00	4.00	1.00	389.00			
Rat ID	Weight	RDA	Strawberry/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
13	230.1	35.88	9.22	195	35.10	15.25%	0.40	0.66	22.23	4.98
14	245.93	37.72	9.70	206	39.93	16.24%	0.92	0.82	23.37	5.24
15	245.2	37.63	9.67	213	32.20	13.13%	0.09	0.54	23.31	5.22
16	249.03	38.07	9.79	207	42.03	16.88%	1.15	0.87	23.59	5.29
17	247.26	37.87	9.74	210	37.26	15.07%	0.63	0.74	23.46	5.26
18	258.71	39.18	10.07	220	38.71	14.96%	0.79	0.79	24.27	5.44
19	406.22	54.95	14.13	359	47.22	11.62%	1.70	0.96	34.05	7.63
20	416.49	55.99	14.39	376	40.49	9.72%	0.98	0.84	34.69	7.77
21	431.75	57.52	14.79	384	47.75	11.06%	1.76	0.96	35.64	7.99
22	434.67	57.82	14.86	405	29.67	6.83%	-0.18	0.43	35.82	8.03
23	443.77	58.72	15.10	395	48.77	10.99%	1.87	0.97	36.38	8.15
24	452.44	59.58	15.32	396	56.44	12.47%	2.70	1.00	36.91	8.27
Total	**4061.57**	**570.93**	**160.63**	**3566.00**	**495.57**				**353.71**	**62.83**
Average	**338.46**	**47.58**	**11.28**	**297.17**	**41.30**	**12.85%**			**29.48**	**6.28**
12				**Median**	**40.21**	**12.80%**				
				Variance	**58.57**	**0.09%**				
				Std.Deviation	**7.65**	**2.97%**				
				UCL		**15.38%**				
				LCL		**10.23%**				

% Weight Loss	12.85%
etsCal/100g Rat	8.71

GL/100g Rat	1.8564303

Group 9: All Bran Fakes			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			69.00	59.00	14.00	3.00	322.00			
Rat ID	Weight	RDA	All Bran/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
25	231.26	36.02	11.19	219	12.26	5.30%	-2.05	0.02	27.50	4.55
26	239.38	36.96	11.48	221	18.38	7.68%	-1.39	0.08	28.22	4.67
27	241.7	37.23	11.56	236	5.70	2.36%	-2.76	0.00	28.42	4.71
28	247.39	37.88	11.77	233	14.39	5.82%	-1.82	0.03	28.92	4.79
29	250.8	38.28	11.89	235	15.80	6.30%	-1.67	0.05	29.22	4.84
30	261.36	39.48	12.26	244	17.36	6.64%	-1.50	0.07	30.14	4.99
31	394.63	53.77	16.70	381	13.63	3.45%	-1.91	0.03	41.05	6.80
32	406.61	54.99	17.08	389	17.61	4.33%	-1.48	0.07	41.99	6.95
33	373.02	51.55	16.01	341	32.02	8.58%	0.07	0.53	39.36	6.52
34	438.88	58.23	18.09	411	27.88	6.35%	-0.37	0.35	44.46	7.36
35	441.76	58.52	18.17	421	20.76	4.70%	-1.14	0.13	44.68	7.40
36	455.74	59.90	18.60	436	19.74	4.33%	-1.25	0.11	45.74	7.57
Total	**3982.53**	**562.82**	**158.34**	**3767.00**	**215.53**				**429.70**	**56.18**
Average	**331.88**	**46.90**	**13.33**	**313.92**	**17.96**	**5.49%**			**35.81**	**5.62**
12				**Median**	**17.49**	**5.56%**				
				Variance	**47.88**	**0.03%**				
				Std.Deviation	**6.92**	**1.77%**				
				UCL		**7.09%**				
				LCL		**4.03%**				

% Weight Loss	5.49%
etsCal/100g Rat	10.79

GL/100g Rat	1.6929166

Group 10: Nutrific			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			80.00	66.00	13.00	3.00	346.00			
Rat ID	Weight	RDA	Nutrific/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
37	233.25	36.25	10.48	217	16.25	6.97%	-1.62	0.05	30.40	5.53
38	236.9	36.67	10.60	234	2.90	1.22%	-3.06	0.00	30.76	5.60
39	248.78	38.04	11.00	228	20.78	8.35%	-1.14	0.13	31.91	5.81
40	248.79	38.05	11.00	238	10.79	4.34%	-2.21	0.01	31.91	5.81
41	252.35	38.45	11.11	242	10.35	4.10%	-2.26	0.01	32.25	5.87
42	256.85	38.97	11.26	250	6.85	2.67%	-2.63	0.00	32.68	5.95
43	391.98	53.50	15.46	392	-0.02	-0.01%	-3.37	0.00	44.87	8.16
44	409.06	55.24	15.97	393	16.06	3.93%	-1.64	0.05	46.33	8.43
45	423.18	56.67	16.38	409	14.18	3.35%	-1.85	0.03	47.53	8.65
46	426.13	56.96	16.46	416	10.13	2.38%	-2.28	0.01	47.78	8.69
47	429.45	57.29	16.56	408	21.45	4.99%	-1.06	0.14	48.05	8.74
48	451.73	59.51	17.20	450	1.73	0.38%	-3.18	0.00	49.91	9.08
Total	**4008.45**	**565.60**	**159.13**	**3877.00**	**131.45**				**474.39**	**68.49**
Average	**334.04**	**47.13**	**12.58**	**323.08**	**10.95**	**3.56%**			**39.53**	**6.85**
12				**Median**	**10.57**	**3.64%**				
				Variance	**50.87**	**0.06%**				
				Std.Deviation	**7.13**	**2.49%**				
				UCL		**5.79%**				
				LCL		**1.48%**				

% Weight Loss	3.56%
etsCal/100g Rat	11.83

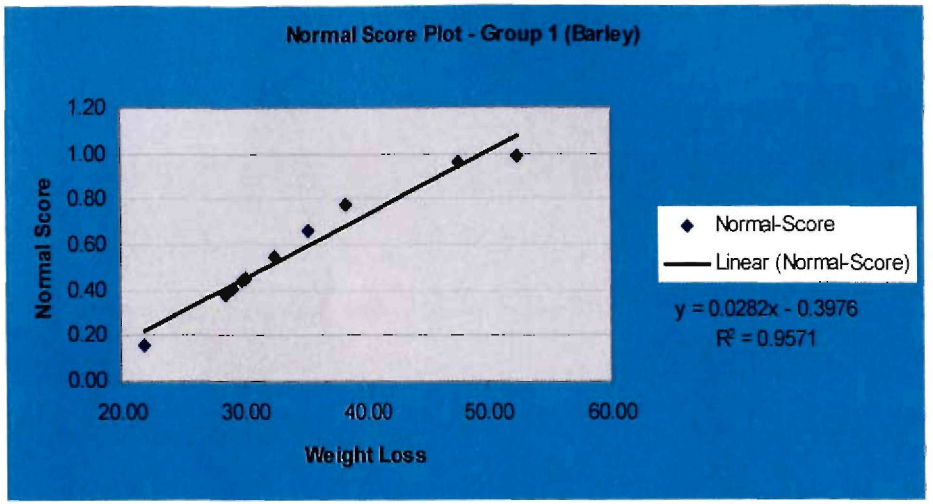
GL/100g Rat	2.0502873

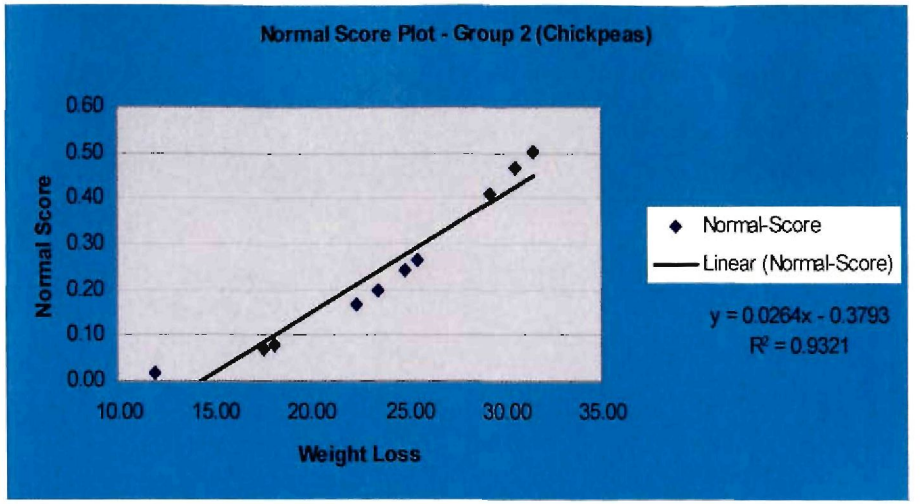
Group 11: Special K			GI	CHO/100g	PRO/100g	FAT/100g	kCal/100g			
			89.00	79.00	11.00	1.00	373.00			
Rat ID	Weight	RDA	Special K/Rat	Weight - W3	Weight Loss	Weight Loss	Z	NORMSDIST	etsCal	GL
(#)	(g)	(kCal)	(g)	(g)	(g)	(%)				
49	233.56	36.28	9.73	213	20.56	8.80%	-1.16	0.12	32.51	6.84
50	243.27	37.41	10.03	232	11.27	4.63%	-2.16	0.02	33.52	7.05
51	239.02	36.92	9.90	232	7.02	2.94%	-2.62	0.00	33.08	6.96
52	254.09	38.65	10.36	236	18.09	7.12%	-1.43	0.08	34.63	7.29
53	253.33	38.56	10.34	231	22.33	8.81%	-0.97	0.17	34.56	7.27
54	271.88	40.66	10.90	249	22.88	8.42%	-0.91	0.18	36.44	7.67
55	392.3	53.53	14.35	380	12.30	3.14%	-2.05	0.02	47.97	10.09
56	406.03	54.93	14.73	389	17.03	4.19%	-1.54	0.06	49.23	10.36
57	425.09	56.86	15.24	403	22.09	5.20%	-1.00	0.16	50.95	10.72
58	422.43	56.59	15.17	402	20.43	4.84%	-1.17	0.12	50.71	10.67
59	434.3	57.78	15.49	413	21.30	4.90%	-1.08	0.14	51.77	10.89
60	453.95	59.73	16.01	436	17.95	3.95%	-1.44	0.07	53.52	11.26
Total	**4029.25**	**567.92**	**159.78**	**3816.00**	**213.25**				**508.90**	**84.90**
Average	**335.77**	**47.33**	**11.73**	**318.00**	**17.77**	**5.58%**			**42.41**	**8.49**
12				**Median**	**19.26**	**4.87%**				
				Variance	**25.57**	**0.05%**				
				Std.Deviation	**5.06**	**2.15%**				
				UCL		**6.73%**				
				LCL		**3.01%**				

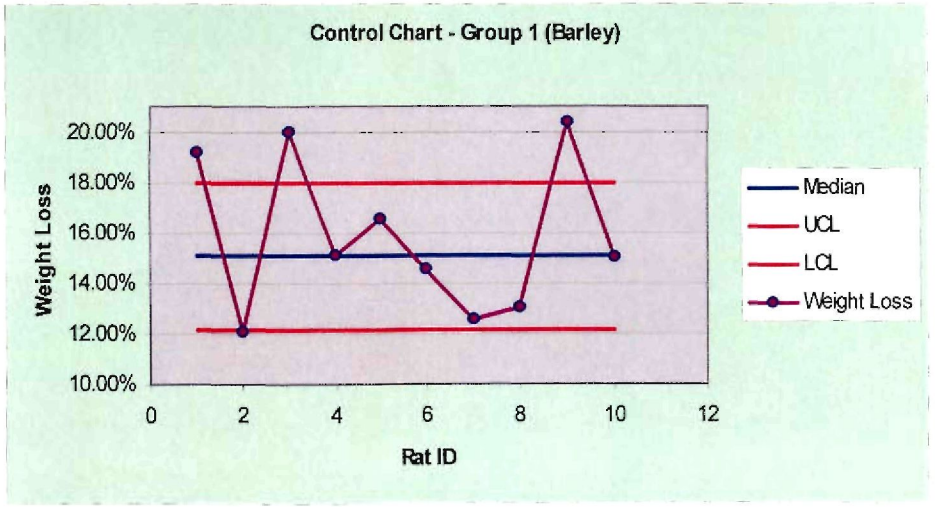
% Weight Loss	5.58%
etsCal/100g Rat	12.63

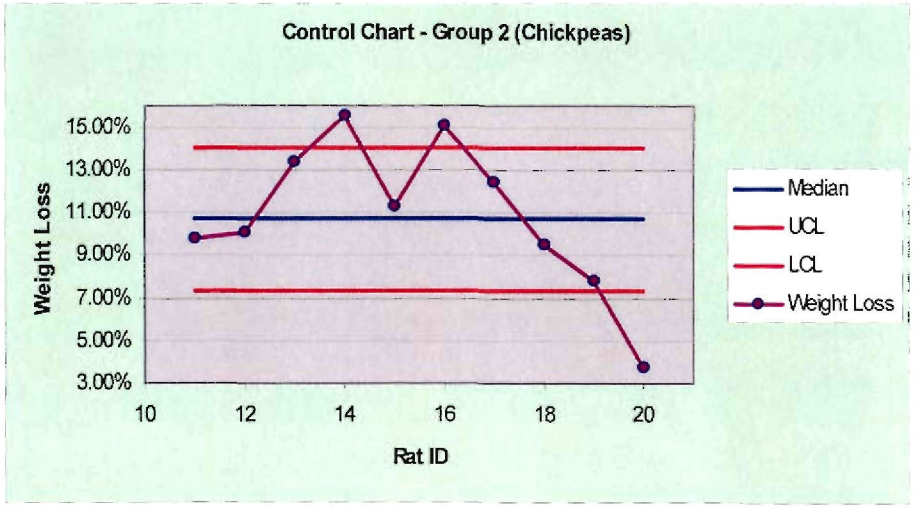
GL/100g Rat	2.528558

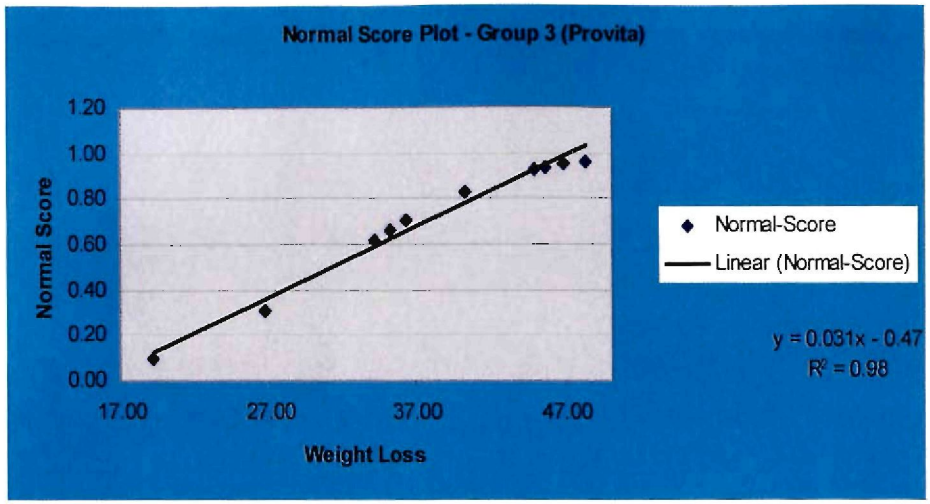
APPENDIX B : DATA ANALYSIS

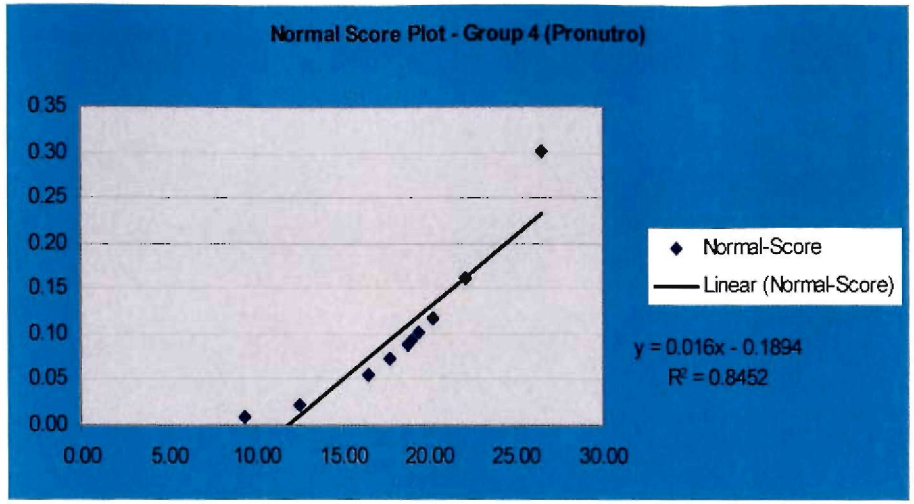

Normal Score Plot - Group 1 (Barley)
Normal Score
1.20
1.00
0.80
0.60
0.40
0.20
0.00
20.00
30.00
40.00
50.00
60.00
Weight Loss
Normal-Score
Linear (Normal-Score)
y = 0.0282x - 0.3976
R² = 0.9571

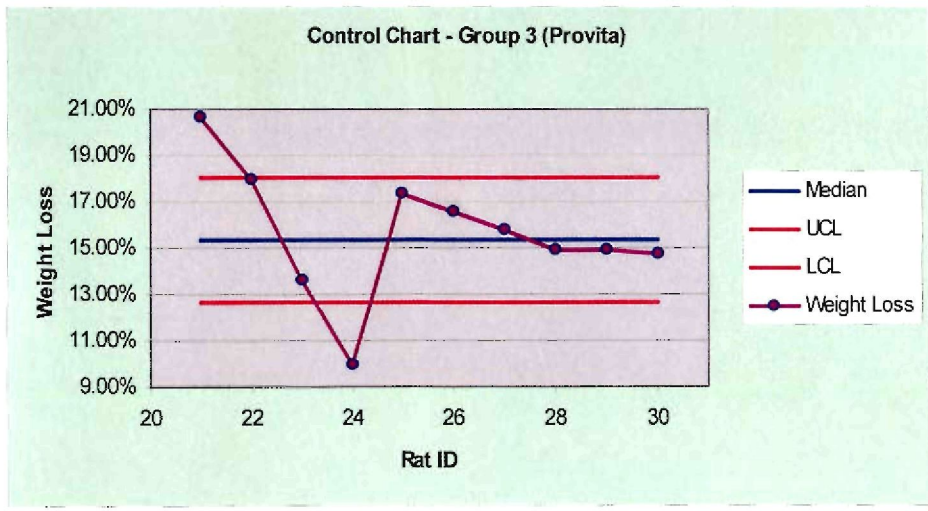

Normal Score Plot - Group 2 (Chickpeas)
Normal Score
0.60
0.50
0.40
0.30
0.20
0.10
0.00
10.00
15.00
20.00
25.00
30.00
35.00
Weight Loss
Normal-Score
Linear (Normal-Score)
y = 0.0264x - 0.3793
R² = 0.9321

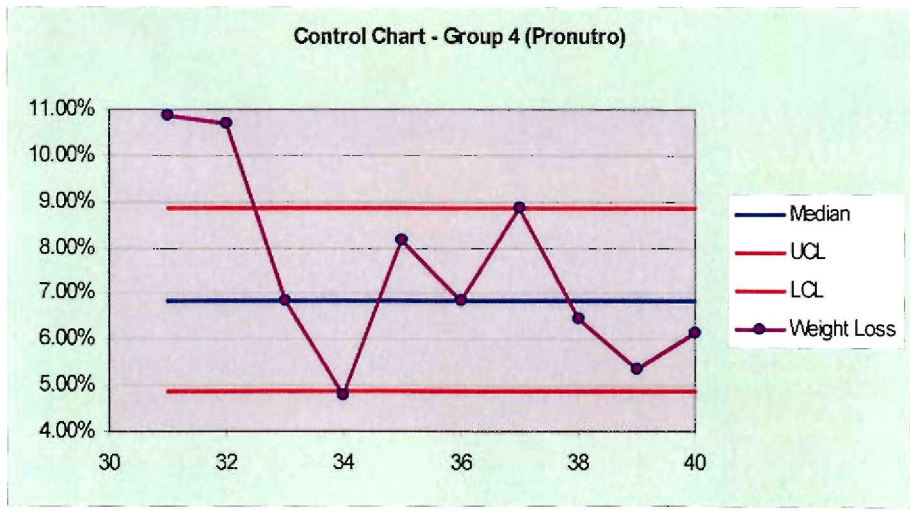

Control Chart - Group 1 (Barley)
Weight Loss
20.00%
18.00%
16.00%
14.00%
12.00%
10.00%
0
2
4
6
8
10
12
Rat ID
Median
UCL
LCL
Weight Loss

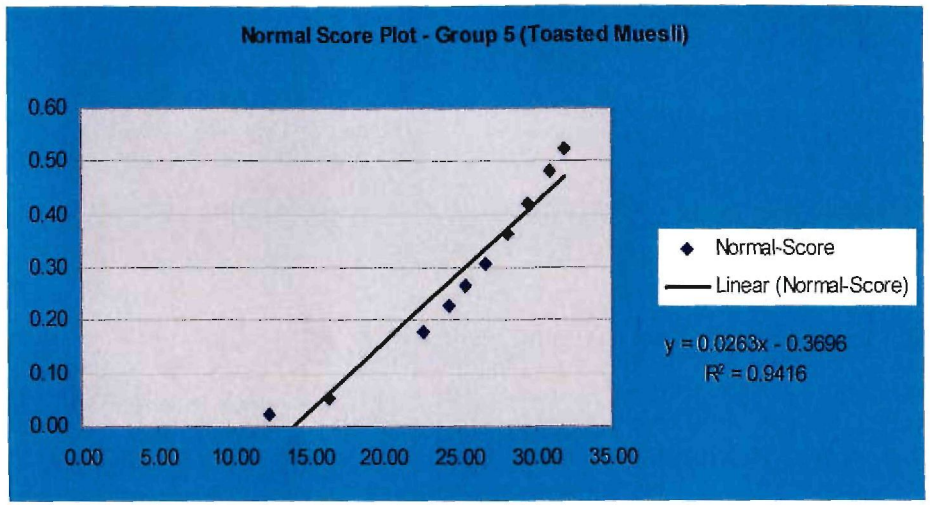

Control Chart - Group 2 (Chickpeas)
Weight Loss
15.00%
13.00%
11.00%
9.00%
7.00%
5.00%
3.00%
10
12
14
16
18
20
Rat ID
Median
UCL
LCL
Weight Loss

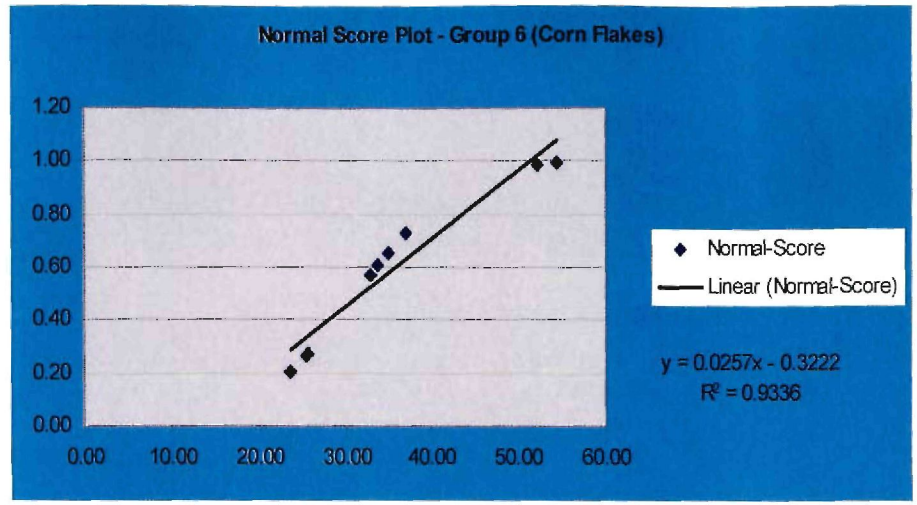

Normal Score Plot - Group 3 (Provita)
Normal Score
1.20
1.00
0.80
0.60
0.40
0.20
0.00
17.00
27.00
37.00
47.00
Weight Loss
Normal-Score
Linear (Normal-Score)
y = 0.031x - 0.47
R² = 0.98

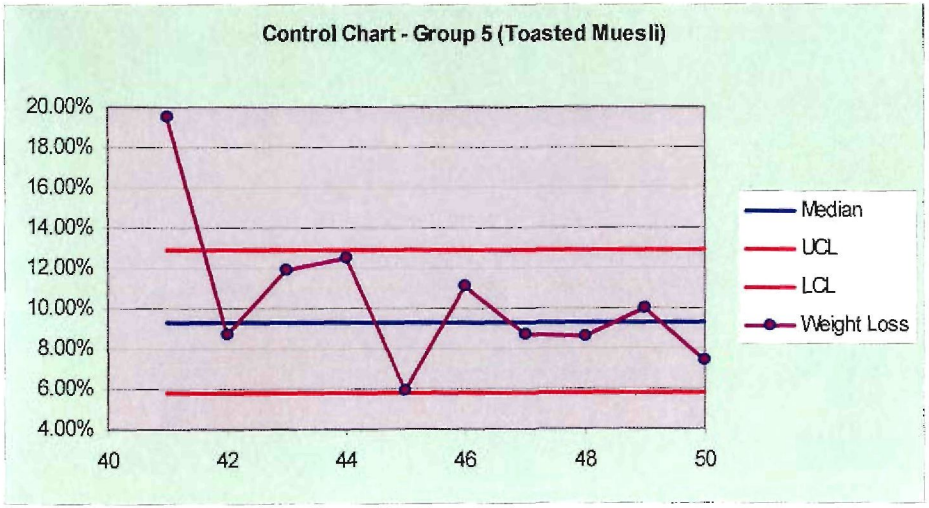

Normal Score Plot - Group 4 (Pronutro)
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.00
5.00
10.00
15.00
20.00
25.00
30.00
Normal-Score
Linear (Normal-Score)
y = 0.016x - 0.1894
R² = 0.8452

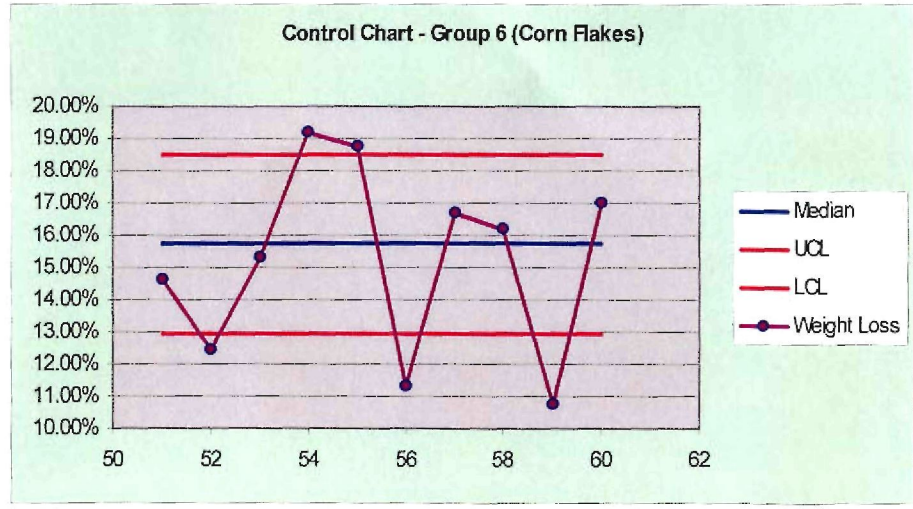

Control Chart - Group 3 (Provita)
Weight Loss
21.00%
19.00%
17.00%
15.00%
13.00%
11.00%
9.00%
20
22
24
26
28
30
Rat ID
Median
UCL
LCL
Weight Loss

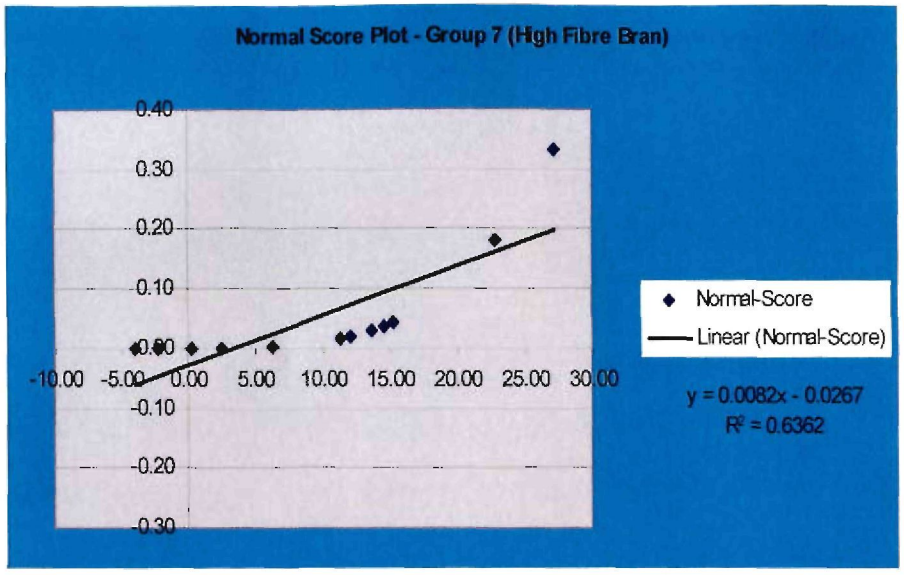

Control Chart - Group 4 (Pronutro)
11.00%
10.00%
9.00%
8.00%
7.00%
6.00%
5.00%
4.00%
30
32
34
36
38
40
Median
UCL
LCL
Weight Loss

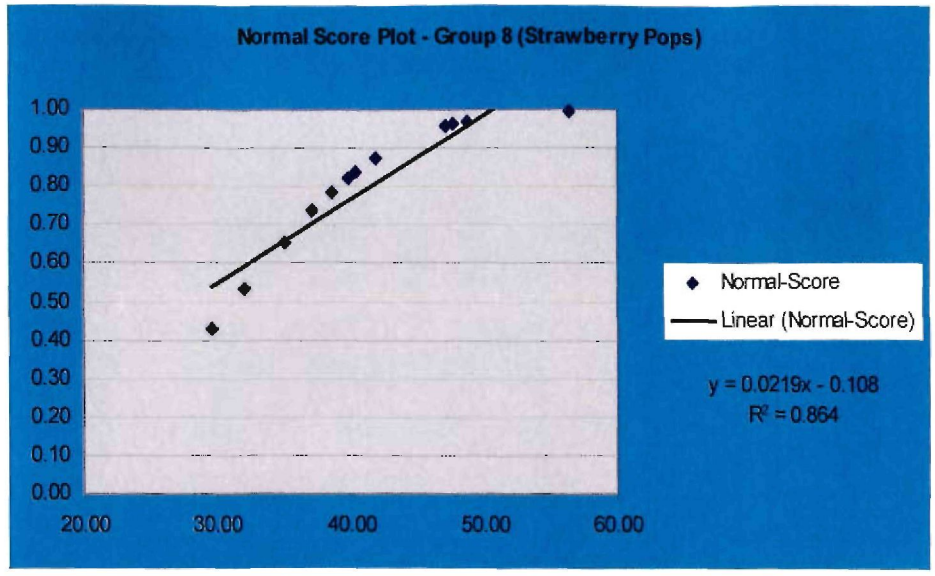

Normal Score Plot - Group 5 (Toasted Muesli)
0.60
0.50
0.40
0.30
0.20
0.10
0.00
0.00
5.00
10.00
15.00
20.00
25.00
30.00
35.00
Normal-Score
Linear (Normal-Score)
y = 0.0263x - 0.3696
R² = 0.9416

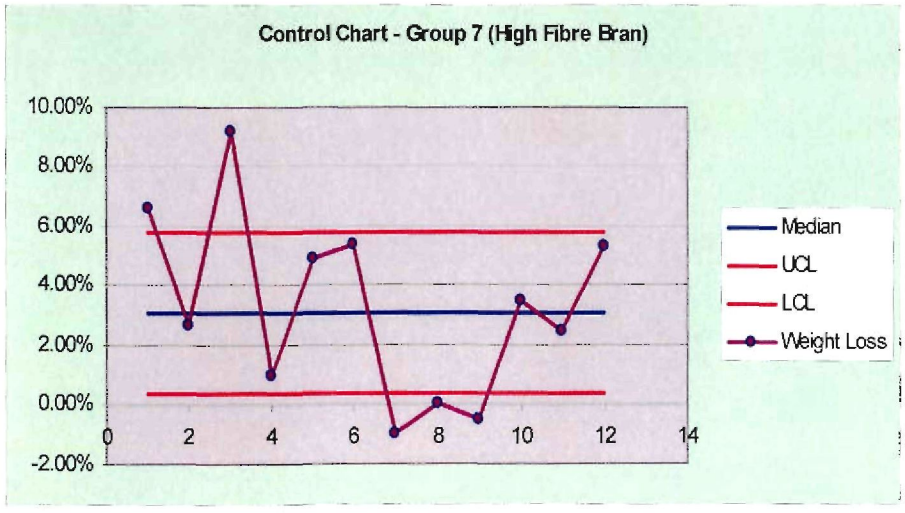

Normal Score Plot - Group 6 (Corn Flakes)
1.20
1.00
0.80
0.60
0.40
0.20
0.00
0.00
10.00
20.00
30.00
40.00
50.00
60.00
Normal-Score
Linear (Normal-Score)
y = 0.0257x - 0.3222
R² = 0.9336

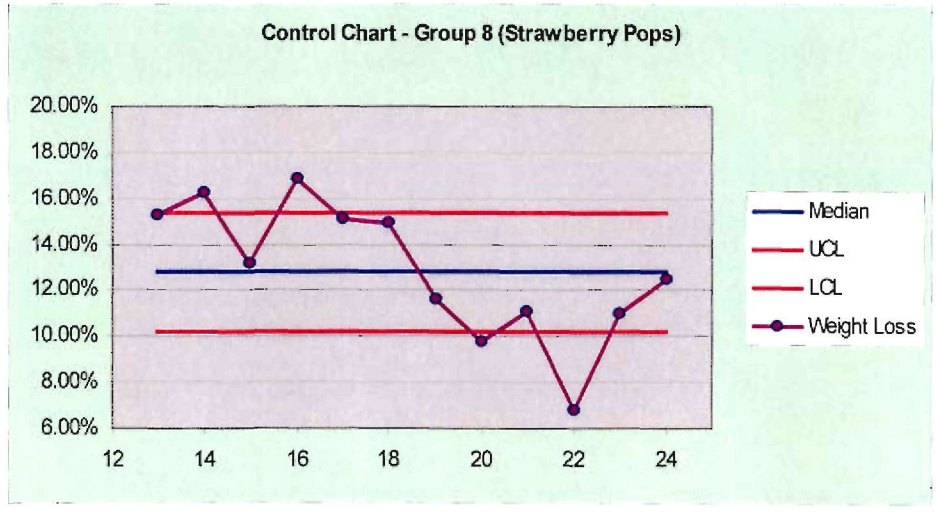

Control Chart - Group 5 (Toasted Muesli)
20.00%
18.00%
16.00%
14.00%
12.00%
10.00%
8.00%
6.00%
4.00%
40
42
44
46
48
50
Median
UCL
LCL
Weight Loss

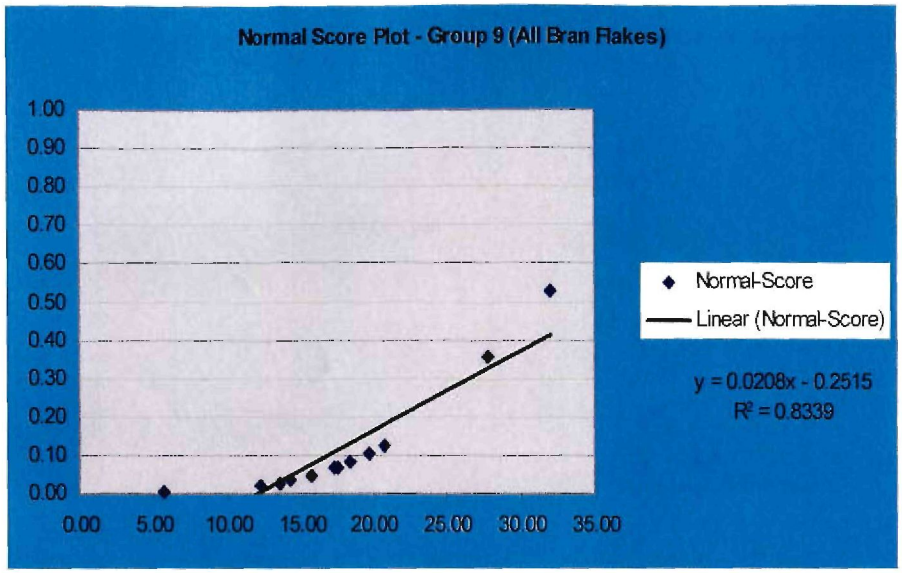

Control Chart - Group 6 (Corn Flakes)
20.00%
19.00%
18.00%
17.00%
16.00%
15.00%
14.00%
13.00%
12.00%
11.00%
10.00%
50
52
54
56
58
60
62
Median
UCL
LCL
Weight Loss

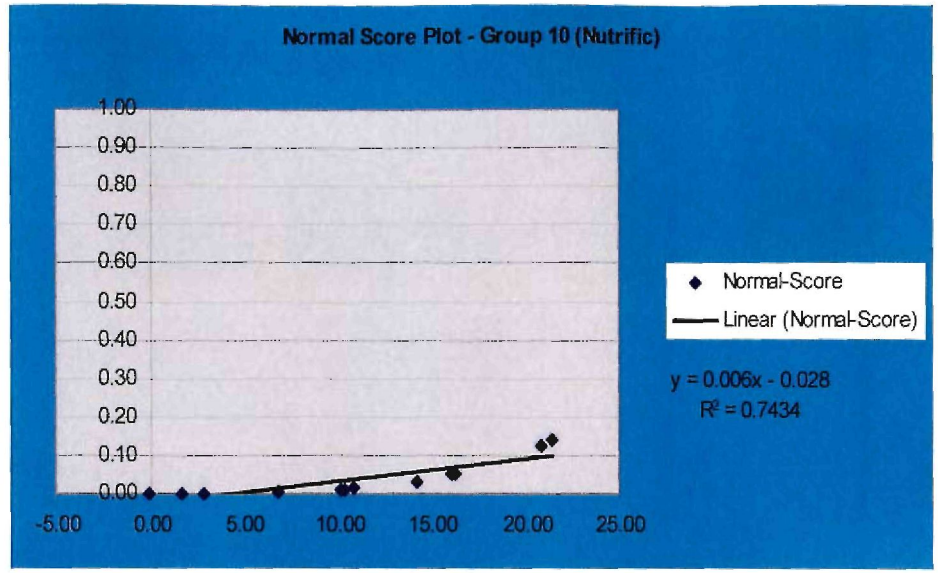

Normal Score Plot - Group 7 (High Fibre Bran)
0.40
0.30
0.20
0.10
0.00
-0.10
-0.20
-0.30
-10.00
-5.00
0.00
5.00
10.00
15.00
20.00
25.00
30.00
Normal-Score
Linear (Normal-Score)
y = 0.0082x - 0.0267
R² = 0.6362

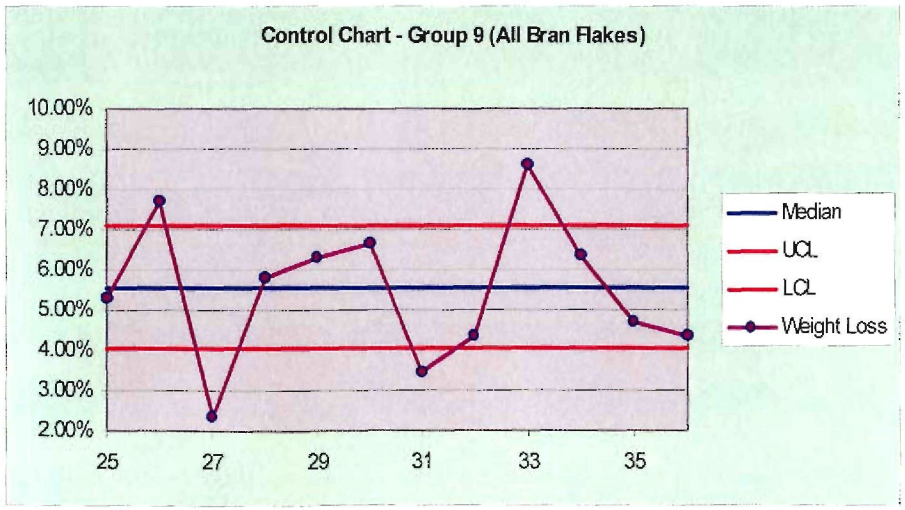

Normal Score Plot - Group 8 (Strawberry Pops)
1.00
0.90
0.80
0.70
0.60
0.50
0.40
0.30
0.20
0.10
0.00
20.00
30.00
40.00
50.00
60.00
Normal-Score
Linear (Normal-Score)
y = 0.0219x - 0.108
R² = 0.864

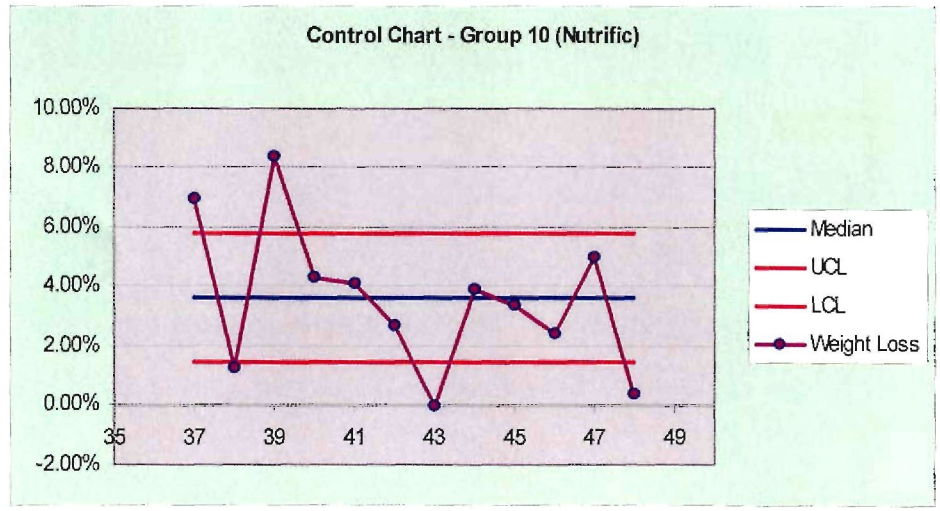

Control Chart - Group 7 (High Fibre Bran)
10.00%
8.00%
6.00%
4.00%
2.00%
0.00%
-2.00%
0
2
4
6
8
10
12
14
Median
UCL
LCL
Weight Loss

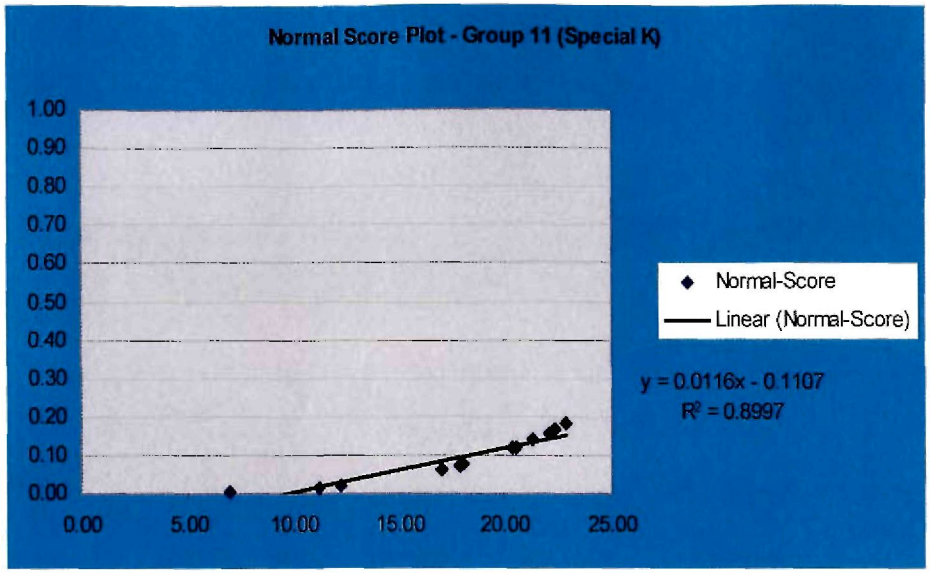

Control Chart - Group 8 (Strawberry Pops)
20.00%
18.00%
16.00%
14.00%
12.00%
10.00%
8.00%
6.00%
12
14
16
18
20
22
24
Median
UCL
LCL
Weight Loss

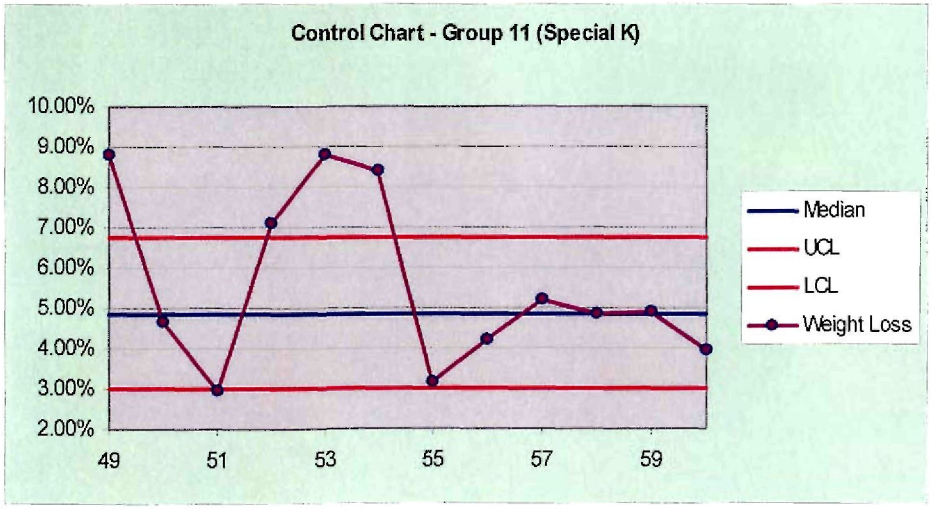

Normal Score Plot - Group 9 (All Bran Flakes)
1.00
0.90
0.80
0.70
0.60
0.50
0.40
0.30
0.20
0.10
0.00
0.00 5.00 10.00 15.00 20.00 25.00 30.00 35.00
Normal-Score
Linear (Normal-Score)
y = 0.0208x - 0.2515
R² = 0.8339


Normal Score Plot - Group 10 (Nutrific)
1.00
0.90
0.80
0.70
0.60
0.50
0.40
0.30
0.20
0.10
0.00
-5.00 0.00 5.00 10.00 15.00 20.00 25.00
Normal-Score
Linear (Normal-Score)
y = 0.006x - 0.028
R² = 0.7434


Control Chart - Group 9 (All Bran Flakes)
10.00%
9.00%
8.00%
7.00%
6.00%
5.00%
4.00%
3.00%
2.00%
25 27 29 31 33 35
Median
UCL
LCL
Weight Loss


Control Chart - Group 10 (Nutrific)
10.00%
8.00%
6.00%
4.00%
2.00%
0.00%
-2.00%
35 37 39 41 43 45 47 49
Median
UCL
LCL
Weight Loss


Normal Score Plot - Group 11 (Special K)
1.00
0.90
0.80
0.70
0.60
0.50
0.40
0.30
0.20
0.10
0.00
0.00
5.00
10.00
15.00
20.00
25.00
Normal-Score
Linear (Normal-Score)
y = 0.0116x - 0.1107
R² = 0.8997


Control Chart - Group 11 (Special K)
10.00%
9.00%
8.00%
7.00%
6.00%
5.00%
4.00%
3.00%
2.00%
49
51
53
55
57
59
Median
UCL
LCL
Weight Loss

APPENDIX C : CALORIFIC METHODS AND VALUES

CAL2K – APPLICATION NOTE
C1.4 : Calorific Measurements of Food Sample - Barley

Introduction

Many institutions are doing research and development on food. The aim is to improve the nutritional value of the food. The parameters may be to compare different foods or different manufactures or to generically improve the food. Other aspects may be to improve the digestion and energy absorption of animal feeds.

Part of the research involves determining the calorific value of the food. The calorific value of a particular food is the same as the energy content of that food.

The food can be for either human or animal consumption.

Institutions performing this type of research include:

- Animal and Dairy research
- Department of Agriculture
- Universities
- Technicons
- Government or private food industries

Sample Preparation

A calorimeter is used to determine the calorific value of any substance that can be ignited. The substance must be in liquid or solid form. In the food industry most samples are in solid form as generally more energy is obtained from solid foods as opposed to liquid substances.

The sample to be measured must be a representative sample and homogeneous. The sample should be ground into a powder, well mixed and then pressed into tablet form. Pressing the sample into a tablet prevents splattering when the sample burns. Splattering is when un-burnt sample is thrown out of the crucible during the combustion process, thus causing inaccurate results. In tablet form, food samples usually burn consistently and without splattering.

Some substances such as maize when ground into a powder will ignite easily and not splatter, but burns with a large open flame, which can easily destroy the o-rings in the vessel. Consequently maize should always be pressed into tablet form.

Certain items such as sugar can be analysed without pressing into tablets – weigh the sugar directly into the crucible.

All samples should have no moisture present before analysing. Freeze-drying the sample can remove the moisture.

Spiking

If a sample does not ignite easily or not at all, then the spiking method of ignition can be used. In this method a benzoic acid tablet is added to the crucible with the sample. The benzoic acid burns easily and ignites the sample; the energy of the benzoic acid is removed from the calculation of the calorific value.

Analysis

Once the sample has been prepared the determination can be carried out in the normal method.

Ensure that the firing cotton touches the sample – with tablets lay the cotton on the bottom of the crucible and then move the tablet on top of the cotton. During the filling process do not knock the vessel, ensuring that the tablet does not move off the cotton.

When substances are being analysed for the first time always check after the determination for any residue on the walls of the vessel and check that the entire sample has burnt.

After a determination clean the inside of the vessel and the crucible before starting the next determination.

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Results

Barley

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
16.046	0.8005	1	7/21/2005	123	0.0015	21.5	22.2	OK	3.1
16.126	0.8004	4	7/21/2005	4	0.0011	22.5	23.0	OK	3.1
16.192	0.8001	6	7/21/2005	4	0.0019	21.4	23.5	OK	3.1
16.189	0.8004	7	7/21/2005	123	0.0009	23.0	23.5	OK	3.1
16.192	0.8006	8	7/21/2005	4	0.0019	21.4	23.6	OK	3.1
16.234	0.8008	9	7/22/2005	123	-0.0003	16.3	16.5	OK	3.1

Average MJ/Kg = **16.163**

Conclusion

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.

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Results

Chick Peas

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
17.744	0.8006	1	7/21/2005	123	0.0012	20.0	18.2	OK	3.1
17.844	0.8002	2	7/21/2005	4	0.0004	20.2	19.2	OK	3.1
17.879	0.8000	3	7/21/2005	123	0.0006	20.0	19.6	OK	3.1
17.865	0.8004	5	7/21/2005	123	0.0019	18.3	20.2	OK	3.1
17.901	0.8001	6	7/21/2005	4	0.0005	22.0	20.6	OK	3.1
18.003	0.8004	7	7/21/2005	4	0.0011	21.6	21.3	OK	3.1
17.862	0.8002	8	7/21/2005	4	0.0010	22.2	21.7	OK	3.1

Average MJ/Kg = **17.871**

Conclusion

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.

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Results

Provita Whole-Wheat

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
18.873	0.8001	1	7/21/2005	123	1.0E-05	15.7	15.3	OK	3.1
19.048	0.8002	2	7/21/2005	4	0.0011	15.7	15.7	OK	3.1
18.982	0.8003	3	7/21/2005	123	0.0018	14.8	16.0	OK	3.1
18.802	0.8001	4	7/21/2005	4	-0.0016	20.7	16.3	OK	3.1
18.750	0.8001	5	7/21/2005	123	-0.0008	19.8	16.8	OK	3.1
18.953	0.8000	6	7/21/2005	4	0.0011	19.1	17.0	OK	3.1
18.772	0.8003	7	7/21/2005	4	-0.0009	21.2	17.5	OK	3.1
Average MJ/KG = **18.883**									

Conclusion

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.

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Results

Pronutro Flakes

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
16.673	0.8003	1	7/20/2005	123	-7E-05	21.6	22.3	OK	3.1
16.738	0.8001	2	7/20/2005	4	0.0017	21.1	22.4	OK	3.1
16.829	0.8000	4	7/20/2005	4	0.0014	21.5	22.5	OK	3.1
16.628	0.8001	5	7/20/2005	123	0.0008	21.7	22.5	OK	3.1
16.760	0.8003	6	7/20/2005	4	0.0004	23.2	22.5	OK	3.1
16.688	0.8001	7	7/20/2005	123	0.0001	22.3	22.6	OK	3.1
Average MJ/Kg = **16.719**									

Conclusion

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.

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Results

Corn Flakes

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
16.625	0.8001	1	7/19/2005	123	0.0011	20.9	19.9	OK	3.1
16.682	0.8003	2	7/19/2005	4	0.0013	20.5	20.2	OK	3.1
16.635	0.8001	3	7/19/2005	123	0.0014	21.1	20.4	OK	3.1
16.648	0.8001	4	7/19/2005	4	0.0008	21.3	20.8	OK	3.1
16.584	0.8003	5	7/19/2005	123	0.0008	21.4	21.0	OK	3.1
16.645	0.8003	6	7/19/2005	4	0.0017	20.9	21.7	OK	3.1

Average MJ/Kg= **16.637**

Conclusion

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.

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Results

Kellog's High Fibre Bran

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
17.789	0.8003	1	7/18/2005	123	0.0004	22.4	22.2	OK	3.1
17.894	0.8001	2	7/19/2005	123	-0.0012	13.0	12.9	OK	3.1
17.959	0.8003	4	7/19/2005	4	0.0010	13.7	13.2	OK	3.1
17.793	0.8002	5	7/19/2005	123	-0.0019	19.4	13.8	OK	3.1
17.757	0.8001	6	7/19/2005	4	-0.0014	19.4	14.2	OK	3.1
17.648	0.8003	3	7/19/2005	123	-0.0019	21.0	15.0	OK	3.1

Average MJ/Kg= **17.807**

Conclusion

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.

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Results

Kellog's Strawberry Pops

Conclusion

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
16.462	0.8000	1	7/19/2005	123	-0.0009	24.4	22.0	OK	3.1
16.628	0.8000	2	7/19/2005	4	0.0018	21.9	22.2	OK	3.1
16.514	0.8002	3	7/19/2005	123	0.0000	23.7	22.3	OK	3.1
16.721	0.8002	4	7/19/2005	4	0.0018	21.3	22.5	OK	3.1
16.431	0.8000	5	7/19/2005	123	-0.0007	25.4	22.5	OK	3.1
16.579	0.8000	6	7/19/2005	4	0.0000	24.6	22.7	OK	3.1
Average MJ/Kg = **16.556**									

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.

Results

Kellog's All Bran Flakes

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
17.234	0.8000	1	7/20/2005	123	0.0014	20.7	21.0	OK	3.1
17.314	0.8002	2	7/20/2005	4	0.0011	21.6	21.3	OK	3.1
17.159	0.8001	3	7/20/2005	123	0.0013	21.2	21.4	OK	3.1
17.294	0.8003	4	7/20/2005	4	0.0017	22.0	21.5	OK	3.1
17.161	0.8000	5	7/20/2005	123	0.0010	21.9	22.0	OK	3.1
17.330	0.8003	6	7/20/2005	4	0.0017	21.4	22.2	OK	3.1

Average MJ/Kg = **17.249**

Conclusion

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.

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Results

Kellog's Nutrific

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
17.059	0.8000	1	7/19/2005	123	-0.0012	19.2	16.1	OK	3.1
16.990	0.8003	3	7/19/2005	123	-0.0010	20.4	16.7	OK	3.1
17.148	0.8001	4	7/19/2005	4	0.0010	18.8	17.1	OK	3.1
17.063	0.8001	5	7/19/2005	123	0.0003	20.0	18.2	OK	3.1
17.069	0.8001	7	7/19/2005	4	-0.0001	21.9	18.4	OK	3.1
17.079	0.8000	8	7/19/2005	123	0.0003	21.1	19.2	OK	3.1
17.107	0.8000	9	7/19/2005	4	0.0004	22.0	19.5	OK	3.1
Average MJ/Kg = **17.074**									

Conclusion

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.

Results

Kellog's Special K

Result	Mass	S-ID	Date	BN	Init Drift	Firing Temp	Ambient Temp	RS	Final Time
16.844	0.8001	3	7/20/2005	123	-0.0002	16.4	16.5	OK	3.1
17.115	0.8000	4	7/20/2005	4	0.0017	17.0	16.8	OK	3.1
16.992	0.8001	6	7/20/2005	4	0.0002	19.6	17.8	OK	3.1
16.858	0.8000	7	7/20/2005	123	0.0004	20.0	19.3	OK	3.1
16.972	0.8003	8	7/20/2005	4	0.0010	20.2	19.7	OK	3.1
16.809	0.8003	9	7/20/2005	123	0.0005	21.0	20.0	OK	3.1
16.942	0.8003	10	7/20/2005	4	-0.0007	24.1	20.3	OK	3.1
Average MJ/Kg = **16.933**									

Conclusion

The calorific value of almost any food type can be determined. Calorific value analysis of a food type is one of many results required to determine the nutritional value of any food for either human or animal consumption.