INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI

films the text directly from the original or copy submitted. Thus, some

thesis and dissertation copies are in typewriter face, while others may be

from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the

copy submitted. Broken or indistinct print, colored or poor quality

illustrations and photographs, print bleedthrough, substandard margins,

and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete

manuscript and there are missing pages, these will be noted. Also, if

unauthorized copyright material had to be removed, a note will indicate

the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by

sectioning the original, beginning at the upper left-hand corner and

continuing from left to right in equal sections with small overlaps. Each

original is also photographed in one exposure and is included in reduced

form at the back of the book.

Photographs included in the original manuscript have been reproduced

xerographically in this copy. Higher quality 6" x 9" black and white

photographic prints are available for any photographs or illustrations

appearing in this copy for an additional charge. Contact UMI directly to

order.

UMI

A Bell & Howell Information Company 300 North Zeeb Road, Ann Arbor MI 48106-1346 USA 313/761-4700 800/521-0600

NOTE TO USERS

The original manuscript received by UMI contains pages with indistinct and slanted print. Pages were microfilmed as received.

This reproduction is the best copy available

UMI

University of Alberta

Energy metabolism and body image of chronic dieters

by

Jacqueline Rochelle Gingras

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science

in

Nutrition and Metabolism

Department of Agricultural, Food, and Nutritional Science

Edmonton, Alberta

Spring 1998



National Library of Canada

Acquisitions and Bibliographic Services

395 Wellington Street Ottawa ON K1A 0N4 Canada Bibliothèque nationale du Canada

Acquisitions et services bibliographiques

395, rue Wellington Ottawa ON K1A 0N4 Canada

Your file Votre reference

Our file Natre reférence

The author has granted a nonexclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.

L'auteur a accordé une licence non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de cette thèse sous la forme de microfiche/film, de reproduction sur papier ou sur format électronique.

L'auteur conserve la propriété du droit d'auteur qui protège cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

0-612-28938-9



University of Alberta

Faculty of Graduate Studies and Research

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled *Energy metabolism and body image of chronic dieters* submitted by *Jacqueline Rochelle Gingras* in partial fulfillment of the requirements for the degree of *Master of Science* in *Nutrition and Metabolism*.

Linda J. McCargar, PhD

Supervisor

Vicki Harber, PhD

Catherine J. Field. PhD

Approved by Committee: 4pril 16, 1998

ABSTRACT

Dieting Syndrome can have negative physiological and Chronic psychological consequences. The study objective was to describe differences between two groups (15/group) of female chronic dieters (aged 21-49) with either a high resting energy expenditure (H-REE) (≥100% of predicted) or a low REE (L-REE) (≤85% of predicted). Body composition, aerobic fitness, physical activity, glucose/insulin response, leptin/thyroid hormone status, dietary intake, dietary restraint, and body image were measured. Both groups were similar with respect to age, height, weight, and BMI. The H-REE group displayed higher lean body mass, insulin response to test meal, thyroxine, reverse-triiodothyronine, and lower dietary restraint. Differences in insulin response were associated with higher ratios of abdominal:gluteal body fat in H-REE. Chronic dieters were less satisfied with their bodies than reference populations. Groups did not differ with respect to dietary intake, aerobic fitness, and physical activity. It appears that H-REE does not necessarily predict positive metabolic health among chronic dieters.

ACKNOWLEDGEMENT

I would first like to recognize the support and encouragement of my supervisor, Linda McCargar, who possessed the unique ability to guide me though the perils of research while allowing me the freedom to make this project my own. I truly appreciate the many meaningful opportunities you have provided.

A generous thank-you goes to the research participants who enthusiastically donated their time. Their commitment to women's health was gratefully appreciated.

My time at The University of Alberta was especially memorable thanks to my fellow graduate students. I acknowledge the support, encouragement, perspective, and laughter you gave so freely.

Along the way, several unique individuals make your life richer for their acquaintance, generous personality, and sheer joy for living. Judy Hancock is one of those people whose friendship will last a lifetime.

Certain people were instrumental in providing their expertise for the timely completion of this project. Thank-you to Vicki Harber, Nigel Gann, Susan Goruk, Catherine Field, Shirley Shostak, and the Dynacare-Kasper Medical Laboratory staff for generously offering your time whenever it was requested.

A very special thanks to the exceptional departmental support staff who were always quick to offer their help in times of crises and who often go unrecognized for their valuable contributions.

My family has always been a tremendous source of inspiration and support. Thank-you for your love and encouragement during my degree and always.

To the complex, intriguing Leanna who gave me a sense of myself beyond my research and continues to provide unparalleled discovery and sustenance.

Finally, this thesis would not have been possible if not for the strength, wisdom, integrity, and patience of my closest friend. Mere words can not express my gratitude, my respect, and my love for you. I am constantly amazed by your rare ability to wade through the chaos, to maintain a rational perspective, and to bring out the best in me, all while devoting the time required to establish yourself as a remarkable researcher, scholar, and teacher. You are truly extraordinary and forever beside me. Thank-you again and again and again.

TABLE OF CONTENTS

CHAPTER ONE ~ INTRODUCTION	1
A. Rationale	1
B. Purpose	3
C. Hypotheses	4
D. Objectives	5
CHAPTER TWO ~ LITERATURE REVIEW	6
A. Chronic dieting syndrome	6
B. Factors influencing energy metabolism of chronic dieters	7
1. Resting energy expenditure 2. Body composition 3. Aerobic fitness and physical activity 4. Biochemical indices 5. Dietary intake 6. Dietary restraint	11 16 22 29
C. Factors influencing body image of chronic dieters	36 37
D. Summary of Literature	40
CHAPTER THREE ~ EXPERIMENTAL DESIGN & METHODOLOGY	41
A. Experimental design	41
1. Initial research	
2. Research design	
B. Methodology	42
1. Anthropometric measurements	
2. Resting energy expenditure	
3. Body composition	
4. Aerobic fitness and physical activity	48
5. Biochemical indices	50
6. Dietary intake	
7. Dietary restraint	
8. Body image	
9. Ethical approval	
10. Statistical analysis of the data	57

1. Participant recruitment	
1. Participant recruitment	58
· · · · · · · · · · · · · · · · · · ·	
5. Biochemical indices	
6. Dietary intake	67
7. Dietary restraint	68
8. Body image	68
9. Summary of results with reference to the study hypotheses	
CHAPTER FIVE ~ DISCUSSION	72
A. Major findings	72
B. Other findings	72
1. Participant recruitment	73
2. Participant characteristics	73
3. Body composition	74
4. Aerobic fitness and physical activity	
5. Biochemical indices	78
6. Dietary intake	83
7. Dietary restraint	
8. Body image	86
CHAPTER SIX ~ CONCLUSION AND RECOMMENDATIONS	88
A. Conclusion	88
B. Recommendations	89
1. Limitations of the research	89
2. Future research	89
3. Recommendations for chronic dieters	90
LITERATURE CITED	92
ADDENDICES	112

LIST OF APPENDICES

Appendix A	Sample Size Calculation	113
Appendix B	Pre-Determined DXA Regions (R1 and R2)	114
Appendix C	Baecke Questionnaire	115
Appendix D	Normal Fasting Laboratory Values	117
Appendix E	Food Frequency Questionnaire	118
Appendix F	Dutch Eating Behaviour Questionnaire	130
Appendix G	Multidimensional Body-Self Relations Questionnaire	131
Appendix H	MBSRQ Scoring Manual	136
Appendix I	Results Package for Participants	137
Appendix J	Ethical Approval	139
Appendix K	Consent Form	140
Appendix L	Correlations	141

LIST OF TABLES

Table 1	Energy yields from oxidation of substrates	9
Table 2	MBSRQ 3 X 2 conceptual matrix	38
Table 3	Characteristics of female chronic dieters	58
Table 4	Body composition characteristics of female chronic dieters	59
Table 5	Fat distribution by pre-determined regions	60
Table 6	Aerobic fitness and activity of participants	61
Table 7	Comparisons between high fit and low fit female chronic dieters	62
Table 8	Glucose and insulin values during OGTT	63
Table 9	Thyroid hormones at fasting	67
Table 10	Dietary intake of female chronic dieters	67
Table 11	Other dietary factors of female chronic dieters	68
Table 12	MBSRQ body image subscales of female chronic dieters	69
Table 13	Body image scores of chronic dieters compared to reference norms	70
Table 14	Correlation coefficients (r) for demographic, anthropometric, body composition, aerobic fitness, and physical activity related to REE for female chronic dieters (n=30)	. 141

Table 15	Correlation coefficients (r) for metabolic indices (glucose,
	insulin, leptin, and thyroid), and dietary intake as related
	to REE indices for female chronic dieters (n=30)142
Table 16	Correlation coefficients (r) for other dietary factors, dietary
	restraint, and body image as related to REE indices for
	female chronic dieters (n=30)143
Table 17	Correlation coefficients (r) for demographic, anthropometric,
	aerobic fitness, and biochemical indices as related to body
	composition for female chronic dieters (n=30)144
Table 18	Correlation coefficients (r) for demographic, anthropometric,
	body composition, and biochemical indices as related to
	aerobice fitness for female chronic dieters (n=30)145
Table 19	Correlation coefficients (r) for body composition, fat distribution,
	insulin, as related to leptin for female chronic dieters (n=30)146
Table 20	Correlation coefficients (r) for anthropometric and dietary
	intake as related to dietary restraint and self-classified weight for
	female chronic dieters (n=30)147

LIST OF FIGURES

Figure 1	Glucose response during OGTT	64
Figure 2	Insulin response during OGTT	65
Figure 3	Leptin response during OGTT	66

ABBREVIATIONS

AE: Appearance evaluation

AO: Appearance orientation

BASS: Body areas satisfaction scale

BEE: Basal energy expenditure

BMC: Bone mineral content (g)

BMI: Body mass index (kg/m²)

DEBQ: Dutch eating behaviour questionnaire

DXA: Dual-energy x-ray absorptiometry

FE: Fitness evaluation

FFM: Fat-free mass (kg)

FFQ: Food frequency questionnaire

FO: Fitness orientation

HE: Health evaluation

HO: Health orientation

IO: Illness orientation

kcal: Kilocalories

kp: Kiloponds

LBM: Lean body mass (kg)

mRNA: Messenger ribonucleic acid

NPY: Neuropeptide Y

OP: Overweight preoccupation

PAR-Q: Physical activity readiness questionnaire

REE: Resting energy expenditure (kcal/day)

RMR: Resting metabolic rate (kcal/hr)

RQ: Respiratory quotient (VCO₂/VO₂)

r-T₃: Reverse-triiodothyronine (nmol/L)

SCW: Self-classified weight

T₃: Triiodothyronine (nmol/L)

T₄: Thyroxine (nmol/L)

VO_{2max}: Maximal oxygen consumption (mL 0₂/min)

WHR: Waist-to-hip ratio

DEFINITIONS

Dieting

Chronic dieting syndrome is the persistent overconcern with weight and shape, restriction of food choices for two years or more, and continual dieting to lose weight, either with success or with success with weight regain.

Above definition from Grodner, 1992.

Weight cycling is the repeated loss and regain of weight typically accompanied with alternating periods of weight loss and weight regain.

Above definition from National Task Force on the Prevention and Treatment of Obesity, 1994.

Body composition

Adipose tissue is the storage form of body fat plus its supporting cellular and extracellular structures.

Body fat mass is the quantity of triglyceride fat in the body.

Fat-free mass is the lean body mass plus non-fat components of adipose tissue.

Lean body mass is the non-adipose tissue body mass.

Above definitions adapted from Jensen, et al., 1993.

Aerobic fitness and physical activity

Physical activity is any body movement produced by skeletal muscles that results in energy expenditure.

Exercise is planned, structured, and repetitive bodily movement done to improve or maintain one or more components of physical fitness.

Physical fitness is the set of attributes that people have or achieve that relate to the ability to perform physical activity. The most common such attribute is aerobic power (maximal oxygen uptake).

Health is the freedom from disease and the ability to achieve the activities of daily living with ease and enjoyment.

Health-related physical fitness is the component of physical fitness that is associated with some aspect of health. The important factors in health-related

physical fitness include cardiorespiratory endurance, muscular endurance, muscular strength, body composition, and joint flexibility.

Above definitions adapted from Caspersen, Christenson, and Pollard, 1985.

Biochemical indices

Insulin sensitivity is a measure of the efficiency of glucose uptake by tissues as regulated by the presence of insulin. Decreased insulin sensitivity is also known as insulin resistance.

Above definition from Daly, et al., 1997.

Insulin resistance is a state in which a normal amount of insulin produces a subnormal biological response or the finding of higher than normal levels of insulin in the presence of normal or elevated levels of blood glucose.

Above definition from Beck-Nielson, 1992.

Body image

Body image is a multifaceted construct consisting of two main components: a 'perceptual component' and a 'attitudinal component'. Perceptions include body size estimations, while attitudes consist of self-perceptions, cognitions, affects, and behaviour influenced by one's physical attributes

Above definition from Cash and Henry, 1995.

CHAPTER ONE INTRODUCTION

A. RATIONALE

To maintain physiologic functions, the human body continuously expends energy through oxidative metabolism (Schutz, Jequier, 1994). The World Health Organization (WHO) defines an individual's energy requirement as the level of caloric intake that will balance energy expenditure when the individual has a body size, composition, and level of physical activity consistent with long-term good health (WHO, 1985). Thus, energy balance occurs when energy intake matches energy expenditure. The four main components of energy expenditure include basal energy expenditure (BEE), diet-induced thermogenesis (DIT), physical activity, and adaptive thermogenesis. BEE is the energy necessary to support life. Resting energy expenditure (REE), often used synonymously with BEE, represents the actual measurement of energy expenditure in the rested and fasted state, but tends to be approximately 10% greater than BEE (Kinney, 1983). REE accounts for the majority of an individual's total daily energy expenditure (65-75%) (Ravussin, Bogardus, 1992). Resting metabolic rate (RMR) is often used interchangeably with REE, yet RMR is expressed as kilocalories (kcal) expended per hour (or minute) and REE typically denotes the kcal expended per day. Basal and resting energy metabolism consist of energy exchanges occurring in all cells of the body (Groff, Gropper, Hunt, 1995a) especially the liver, the brain, the heart, and the kidney. These processes include ion pumps, synthesis and degradation of cell constituents, biochemical cycles, and leakage of protons across the mitochondrial membrane. Researchers have found that over two-thirds of BEE is due to the sodium pump mechanism and protein turnover (Grande, 1980). Other factors influencing BEE include age, gender, body temperature, ambient temperature, food intake, and body composition.

In dieting individuals, or those who restrict intake to facilitate weight loss, energy balance is altered such that energy intake is less than energy output with the intention of decreasing body weight. The chronic dieter is defined as an individual who consistently restricts energy intake to maintain an average or below average body weight (Beals, Manore, 1994; Grodner, 1992). The physiological and psychological consequences of chronic dieting are varied and may serve to influence metabolism long-term (Manore, 1996).

Dieting continues to be a common practice among North American women. It has been suggested that dieting among women has become the norm – it is more common for a woman to be on a diet than not (Polivy, Herman, 1983). It has also been suggested that dieting behaviours may lead to a decreased REE as an adaptive mechanism by the body to conserve energy (Blackburn, 1989; Elliot, 1989; Manore, et al., 1991; Steen, et al., 1988; Platte, 1996). Although this theory has received widespread publicity in the media, it has not been well supported in the scientific literature (Wing, 1992; National Task Force on the Treatment and Prevention of Obesity, 1994; Wadden, 1991). However, McCargar and McBurney (1996) recently assessed the REE of female chronic dieters (n=172) and found that a subset of women (n=30; 17.4%) had a reduced REE (defined as ≤85% of the predicted REE by the Mifflin equation (1991)). As such, questions remain regarding the factors responsible for low and high REE in female chronic dieters.

Chronic dieters typically possess specific psychological characteristics. Distinctive behaviours and cognitions such as preoccupation with shape and weight, perceived deprivation, and dysfunctional beliefs about food and exercise are frequently observed (Lowe, 1993). Mossavar-Rahmani, et al., (1996) found that the more inaccurate the body size estimation, the greater the likelihood of dieting. Thus, dieting and body image cognitions, affects, and behaviours are fundamentally linked.

B. PURPOSE

The current research was conducted to investigate factors that may influence energy metabolism and body image of female chronic dieters aged 25 to 49 years. Two groups of female chronic dieters were compared; one with higher REE than predicted versus one with lower REE than predicted. Indices of body composition, aerobic fitness, physical activity, metabolic hormones, dietary intake, and dietary restraint were measured in both groups.

A paucity of research has been conducted on the body images of adult women as compared to college-aged women (Altabe, Thompson, 1993; Brodie, Slade, 1988). By measuring the body images of adult, female dieters, researchers may begin to further understand the perceptions, cognitions, affects, and behaviours surrounding the impetus to diet. This information may prove useful in designing treatment modalities that promote self-awareness and self-acceptance for body image distorted women who may be otherwise metabolically healthy.

C. HYPOTHESES

The research hypotheses for this study were as follows:

- Female chronic dieters with a high REE will have more favourable values for metabolic variables including higher LBM, higher aerobic fitness, higher physical activity, normal biochemical indices (glucose, insulin, leptin, thyroid), and higher dietary intake (energy, fat, carbohydrate, protein) compared to female chronic dieters with a low REE.
- 2. Female chronic dieters with a high REE will have lower levels of both android fat distribution and dietary restraint compared to female chronic dieters with a low REE.
- 3. Female chronic dieters will have lower body image scores compared to age- and gender-matched reference norms.

D. OBJECTIVES

The objectives of the present study were:

- To determine REE at two separate time points and to classify individuals as having high (≥100% of predicted) or low (≤85% of predicted) REE.
- 2. To assess body composition and to quantify lean body mass, fat mass, and fat distribution using dual-energy x-ray absorptiometry.
- 3. To determine levels of aerobic fitness and physical activity scores using VO_{2max} and the Baecke questionnaire, respectively.
- 4. To measure biochemical indices including serum glucose, insulin, leptin, thyroxine (T_4) , triiodothyronine (T_3) , and reverse-triiodothyronine $(r-T_3)$.
- 5. To estimate dietary intake and dietary restraint scores using a food frequency questionnaire and the Dutch Eating Behaviour questionnaire, respectively.
- 6. To quantify body image using the Multi-dimensional Body-Self Relations questionnaire.

CHAPTER TWO LITERATURE REVIEW

Energy metabolism is a complex interaction of biological processes. Many factors influence energy metabolism and body image of female chronic dieters. Of the elements associated with energy metabolism, specific parameters are of relevance to the present research. The physiological components to be addressed include REE, body composition, physical activity, metabolic indices (insulin, glucose, leptin, and thyroid hormones), dietary intake, and dietary restraint. The psychological aspect of chronic dieting will be addressed through an examination of body image. Body image is associated with energy metabolism through the behaviours identified as dietary restraint.

A. CHRONIC DIETING SYNDROME

It is estimated that 40% of all North American adult women are presently trying to lose weight (Horm, Anderson, 1993; Serdula, et al., 1993). This estimate may be even higher among college-aged women (National Institute of Health, 1993). In response to this prevailing situation, the concept of "dietary restraint" has evolved to describe the cognitive behaviours associated with chronic dieting (Herman, Polivy, 1980). Dietary restraint is a descriptive phrase defining specific behaviours associated with the act of restricting food consumption, but it does not encompass the social, psychological, and physical ramifications that result from continual restrictive food consumption (Grodner, 1992). This concept is characterized by restriction of food intake to control body weight through cognitive overriding of physiological needs (Herman, Polivy, 1980). Chronic dieting is not intended to identify someone who occasionally diets, but rather those who incorporate dieting as a permanent aspect of their lifestyles (Grodner, 1992). This type of dieter is frequently referred to as a restrained eater and may be at greater risk for poor nutrient intakes and health problems than the weight cycler or yo-yo dieter (Manore, 1996). The criteria for chronic dieting syndrome include persistent over-concern with body shape and weight, restriction of food choices for two years or more, and continual dieting to lose weight without success, or success at weight loss, but weight regained (Grodner, 1992). Currently, researchers are examining the long-term health consequences associated with this type of dieting. Commonly accepted, yet equivocal, risks of chronic dieting include lowered REE, altered body composition, diminished exercise performance, marginal malnutrition, increased menstrual dysfunction, and heightened psychological stress (Manore, 1996).

B. FACTORS INFLUENCING ENERGY METABOLISM OF CHRONIC DIETERS

1. Resting energy expenditure

REE comprises 60 - 75% of total energy expenditure in most individuals and includes the cost of maintaining the integrated systems of the body and the homeothermic temperature at rest (Sims, Danforth, 1987). REE differs from RMR only by it's unit of expression; REE indicates resting energy expended per day while RMR expresses resting energy expended per minute or hour. BEE represents a more accurate measurement of energy expenditure than REE since it is determined under controlled and standardized conditions (the individual is post-absorptive, supine, relaxed, recently awakened). An 8% overestimation of BEE has been reported when metabolic rate was measured in outpatient conditions compared to inpatient conditions, underscoring the importance of standardized experimental protocol (Berke, et al., 1992).

1.1 Measurement of REE

Energy expenditure can be measured directly or indirectly. Direct calorimetry consists of the measurement of the heat dissipated by the body by radiation, convection, conduction, and evaporation (Jequier, 1985). However, this technique is impractical and rarely possible in clinical settings. Most often, REE is measured by indirect calorimetry. Other non-calorimetric methods for estimating energy expenditure include heart rate monitors, activity diaries, accelerometers, and doubly labelled water (Schutz, Jequier, 1994).

1.2 Indirect calorimetry

Indirect calorimetry is a non-invasive, non-intrusive technique that can be applied in fundamental research, in clinical studies, and when combined with other techniques (such as heart rate monitoring), in field studies (Schutz, Deurenberg, 1996). With the advent of the ventilated hood system (permitting continuous measurement of VO₂ and VCO₂ without a mask or mouthpiece), the potential of indirect calorimetry application is large (Schutz, 1995). Indirect calorimetry does not only allow one to calculate the rate of energy expenditure, but also to estimate the rate of substrate utilization. The determination of substrate oxidation depends upon both the absolute rate of O₂ consumption and CO₂ production as well as the ratio between the latter and the former, the respiratory quotient (RQ), defined as the ratio of CO₂ production to O₂ consumption (Schutz, Deurenberg, 1996).

Heat production generated by the biochemical processes within the body can be assessed from the measurement of oxygen consumption, carbon dioxide production, and urinary nitrogen excretion (Jequier, Acheson, Schutz, 1987). Since there is a proportionality between the rate of oxygen consumption and ATP synthesis, and because each mole of ATP synthesized is accompanied by the production of a given amount of heat, one can understand the rationale of using VO₂ measurements to calculate heat production within the body (Schutz, Jequier, 1994). The implicit assumptions of indirect calorimetry are: (1) all gas exchange is via the lungs (skin and urinary losses are ignored), (2) a metabolic equilibrium exists (absence of hyperventilation, acidosis), (3) urinary nitrogen excretion reflects simultaneous protein catabolism, and (4) only carbohydrate, fat, and protein are being metabolized to their end products (Ben-Porat, Sideman, Bursztein, 1983). Under conditions of thermal equilibrium in a subject at rest and in post-absorptive conditions, heat dissipation (as measured by direct calorimetry) is identical to heat production (as measured by indirect calorimetry) (Schutz, Jequier, 1994).

1.3 Variation in indirect calorimetry

Variations arise due to the complex nature of determining REE from indirect calorimetry. Although the error involved in calculating REE is minimal, the error associated with calculating substrate oxidation is considerably more substantial. Substrate level phosphorylation in the glycolytic pathway allows the synthesis of 2 mol of ATP without O₂ consumption when 1 mol of glucose is metabolized (compared to 38 mol of ATP synthesized by the complete oxidation of 1 mol of glucose). During fatty acid oxidation, ATP synthesis only occurs by oxidative phosphorylation. There is a difference in the heat produced per liter of oxygen consumed when one compares glucose (5.01 kcal/L O₂) to lipid oxidation rates (4.69 kcal/L O₂) (Schutz, Jequier, 1994) (Table 1).

Table 1: Energy yields from oxidation of substrates

				Heat	Energy Equivalent (per gm)		
			Released (per gm)	Rate of O2 consumption	Rate of CO2 consumption		
Substrate	O ₂ consumed ^a	CO ₂ produced ^a	RQb	kcal	kcal ^c	kcal ^d	
Starch	.829	.829	1.00	4.20	5.06	5.06	
sucrose	.786	.786	1.00	3.96	5.04	5.04	
glucose	.746	.746	1.00	3.74	5.01	5.01	
lipid	2.019	1.427	0.71	9.46	4.69	6.63	
protein	1.010	.844	0.83	4.70	4.66	5.58	
lactic acid	.746	.746	1.00	3.62	4.85	4.85	

a In liters per gram of substrate oxidized

Data from Livesey, Elia, 1988.

The value of 4.85 kcal/L O_2 consumption is normally used as an average energy equivalent (kcal/g) and the associated error is no greater than ± 1 to 2% (Schutz, Jequier, 1994). A statistical error analysis shows an adequately calibrated indirect calorimeter may generate uncertainty in substrate oxidation in the order of <1g/30 minutes for fat and ~2g/30 minutes carbohydrate (Schutz, Deurenberg, 1996). Other factors can influence the accuracy of the measurement of substrate oxidation such as *human* factors (adequate

BQ, respiratory quotient (RQ = CO₂ produced/O₂ consumed)

^c In kcal per liter of oxygen

^d In kcal per liter of carbon dioxide

calibration of the calorimeter), instrument-related factors (stability of the calorimeter), and physiological factors (changes in the size of the bicarbonate and urea body pools, the processes of ketogenesis and gluconeogenesis, colonic fermentation, and other biochemical transformations) (Livesey, Elia, 1988). Because indirect calorimetry does not assess the absolute rate of substrate oxidation, but provides overall disappearance rates, intermediaries produced during metabolic processes may cause an under- or over-estimation of substrate oxidation i.e. gluconeogenesis or lipogenesis (Schutz, 1995). Alternately, ketone bodies excreted in the urine or eliminated in the breath will cause the RQ to decrease below 0.70, the lowest theoretical value when complete oxidation is assumed (Schutz, Deurenberg, 1996). The use of incorrect calorimetric coefficients and equations can also add error to the measurement of REE (Schutz, 1985). The total intra-individual variation in REE measurements has been estimated to be approximately 4-6%, a value that includes both the normal day-to-day fluctuation in metabolic rate and the technical error associated with REE assessment (Ravussin, Bogardus, 1989; Bouchard, 1985; Henry, Hayter, Rees, 1989). Studies comparing indirect calorimetry and doubly labelled water have demonstrated the error at the group level to range from 1-5%, thus corroborating with theoretical equations (Schoeller, 1988; Seale, Miles, Bodwell, 1989).

1.4 REE of chronic dieters

Gender differences are important considerations since they directly relate to energy expenditure. REE is higher in males than in females independent of body composition (Arciero, Goran, Poehlman, 1994; Ferraro, et al., 1992). These gender differences are probably not due to differences in sex hormone concentrations since research has shown that REE is significantly higher in prepubescent boys than in girls even when body composition is accounted for (Goran, Kaskoun, Johnson, 1994). **Differences** in basal hepatic gluconeogenesis, sympathetic nervous activity, and decreased body core temperature are possible mechanisms that may explain lower metabolic rate in females (Ferraro, Ravussin, 1992).

The effects of chronic dieting syndrome on REE are equivocal. Research conducted to determine how chronic dieting affects REE traditionally imposes cycles of weight loss and weight regain (weight cycling) or observations of self-described chronic dieters. Some studies support the lowering of REE with restrained eating in females when controlled for LBM (Manore, 1996; Platte, et al., 1996; Blackburn, et al., 1989). More studies support the opposite finding; that chronic dieting does not affect REE (McCargar, Sale, Crawford, 1996; Lawson, et al., 1995; Wadden, et al., 1996; Jebb, et al., 1991; Refuffe-Scrive, et al., 1994; van Dale, Saris, 1989). Inconsistency in the literature regarding the metabolic effect of cyclical dieting may be due to a variety of factors. These inconsistencies include the subject characteristics (including genetic traits), the interpretation of the research data, and the length, severity, and number of times the cyclical dieting process has occurred (Manore, et al., 1991). It is important to note most research focuses on the effects of weight cycling on REE. A more relevant approach may be to study the effects of chronic dieting on REE.

2. Body composition

The quantification of body compartments is an extremely useful tool in the study of energy metabolism. Determining body composition is important to metabolic research since changes in body composition can be related to changes in energy intake or expenditure.

2.1 Measurement of body composition

Body composition can be measured in a variety of ways. The method of choice depends on one's research purposes and practical limitations. Some commonly applied techniques include hydrostatic weighing or densitometry, skinfold thickness or anthropometry, total body water, total body potassium, bioelectrical impedance, computerized tomography, ultrasound, magnetic resonance imaging, and DXA.

2.2 Dual-energy x-ray absorptiometry

Absorptiometry is a useful and highly accurate method of determining body composition. DXA has been called the "gold-standard" of body composition analysis because it allows measurement independent of electrolyte concentrations and compartmental assumptions; it also possesses a very small precision error (Hansen, et al., 1993; Mazess, Barden, Hanson, 1990). DXA is associated with a low radiation exposure and provides bone mineral content as well as fat and lean tissue mass, thus allowing construction of a three-compartment model of body composition (Aloia, et al., 1995). In addition, DXA has the unique ability to produce precise regional measurements (Pritchard, et al., 1993).

2.3 Variation in absorptiometry

Several studies have been conducted to determine the inherent variability in DXA (Jensen, et al., 1993; Aloia, et al., 1995; Pritchard, et al., 1993; Johansson, et al., 1993). In these studies, DXA was evaluated for accuracy and precision by using both anthropomorphic phantoms and a combination of human body composition techniques. Satisfactory precision for measurement of LBM, total body fat, and total body bone mineral using DXA has been demonstrated both *in vivo* and *in vitro* (Jensen, et al., 1993). Pritchard, et al. (1993) concluded that DXA determined percent body fat with greater precision than underwater weighing as reflected by the coefficient of variation. They also reported that DXA correlated well with other methods. Total body weight was accurately estimated by DXA when compared with weight measured using a high precision scale, thus indicating that DXA provides an accurate measurement of soft tissue mass (Johansson, et al., 1993).

Body thickness, fat distribution, hydration, and the fat content of bone marrow are the major sources of biologic variation when estimating fat and lean tissue mass using DXA (Aloia, et al., 1995). At extreme values of fat-free mass (higher degrees of body thickness), Aloia, et al. (1995) determined that DXA is measuring "something different" from the fat-free mass as measured by total

body water (TBW), total body nitrogen (TBN), and total body potassium (TBK). This finding supported the work of Jensen, et al. (1993) which showed three methods (DXA, TBW, and TBK) of estimating non-fat body mass produced significantly different results. These differences are most likely related to the various body compartments measured by these techniques rather than to inherent inaccuracies in the techniques themselves (Jensen, et al., 1993). Updated software programs applied in DXA attempt to compensate for varying degrees of tissue thickness by making measurement adjustments and reducing measurement error (Mazess, et al., 1992).

Researchers have found that DXA generally overestimates fat mass (Haarbo, et al., 1991; Aloia, et al., 1995). This could be related to variations in intracellular and extracellular water, which contributes 15-20% of the adipose tissue mass (Morse, Soeldner, 1963; Wang, Pierson, 1976). This suggests that changes in hydration can affect lean tissue mass as measured by DXA (Horber, et al., 1992). An assumption using DXA is that lean tissue mass accounts for 73.2% body water without considering possible gender differences in hydration status. After adjusting for this difference, minimal error (1-2%) is introduced (Horber, et al., 1992; Gann, personal communication, 1997). Theoretical calculations, together with empirical data, suggest that variations in hydration status have a relatively small effect on the assessment of soft tissue by DXA (Kohrt, 1995). Overestimations in fat mass are likely related to underestimations in hydration status.

High levels of body fat cause bone mineral density to be underestimated and the converse is also true (Mazess, et al., 1989). However, a small calibration that compensated for the average amount of fat did not correct for individual variations, including the large variations associated with aging, osteoporosis, or corticosteroid use (Mazess, et al., 1989). This calibration ensures that DXA adequately measures bone density as it may be affected by these conditions.

Original reports stated a 0.5 - 1% precision error for body composition analysis (Mazess, et al., 1989; Mazess, Braden, Hanson, 1990) which has since been supported by Pritchard, et al., (1993) using the Hologic QDR 1000W

densitometer (1.2% precision error). The coefficient of variability of body composition from DXA was reported as 1.9% for bone mineral density, 1.8% for percentage fat, 0.6 – 1.5% for lean mass, 2.1 – 5.0% for fat mass, and 1.2% for total body mass (Jensen, et al., 1993; Formica, et al., 1993; Pritchard, 1993). Based on these findings of high reliability and validity, DXA methodology is being evaluated as an alternative criterion method to replace hydro-densitometry in body composition assessment (Lohman, 1992; Hansen, et al., 1993).

2.4 Body composition of chronic dieters

The effects of weight cycling on body composition have been well documented in the literature (Wadden, et al., 1996; Rebuffe-Scrive, et al., 1994; Brownell, Rodin, 1994; McCargar, et al., 1993; Prentice, et al., 1992; Wadden, et al., 1991; Jebb, et al., 1991). More relevant to this thesis is the determination of body composition among chronic dieters, a population that has received significantly less attention (Mortenson, Hoerr, Garner, 1993; Manore, et al., 1991; Emmons, 1994).

Mortenson, Hoerr, and Garner (1993) found that higher levels of chronic dietary restraint in women were associated with a higher body mass index (BMI). In addition, most of these women *perceived* themselves to be heavier than their actual weight. The dissatisfied restrained eaters were very inaccurate in identifying their actual BMIs, even though approximately half were within an acceptable BMI range (20 - 25 kg/m²). These results may indicate that women who are dissatisfied with their bodies and who engage in restrictive dieting are genetically predisposed to be heavier (Mortenson, Hoerr, Garner, 1993).

A study comparing energy expenditure at rest and during exercise, found chronic dieters to be significantly heavier (mean BMI of 23.6 kg/m²) and fatter (mean body fat of 26%) than nondieting controls (Manore, et al., 1991). Although heavier and fatter, the dieters could not be classified as obese (BMI > 27.3 kg/m²). LBM as determined by hydrodensitometry was similar between groups. This research defined dieters as having dieted at least four times for \geq seven to ten days during the previous year. Dieting was defined as eating \leq 1000 kcal

below the energy requirements necessary for weight maintenance. This study demonstrated that chronic dieting may influence body composition.

Factors thought to be associated with overweight in adolescents, such as parental weights, birth order, and socioeconomic status, were not found to be significantly different between adolescent dieters and nondieters (Emmons, 1994). In fact, the majority of adolescent chronic dieters were not overweight (above the 85th percentile of BMI). The clearest delineation between dieters and nondieters was their *perception* of overweight as indicated by higher Body Dissatisfaction and Drive for Thinness scores on the Eating Disorder Inventory questionnaire (Emmons, 1994). Thus, adolescent dieters may be difficult to identify. As such, all adolescents could potentially benefit from educational programs to help them accept more realistic body weights.

Studies reveal several body composition adaptations during periods of energy restriction. Following a 12-month, low-calorie weight reduction program, obese females with android fat distribution showed significant decreases in fat cell number, fat cell weight, and WHR compared to obese females with gynoid fat distribution (Krotkiewski, 1988). Alterations in fat patterning under conditions of negative energy balance may be the result of altered rates of lipolysis in the abdominal region as compared to the gluteal or femoral adipose tissue (Rebuffe-Scrive, et al., 1985; Krotkiewski, 1988). Changes in body composition may affect fat patterning independent of changes in body weight, especially when the changes in adipose tissue and muscle tissue are not parallel (Krotkiewski, 1988).

Hyperphagic response has been correlated to the degree of fat and lean recovery after periods of chronic energy restriction. Such findings may be extrapolated to chronic dieters, as energy restriction is the main characteristic of this population. Post hoc analysis of the subjects from the classic Minnesota Experiment (Keys, et al., 1950) revealed that the magnitude of post-starvation hyperphagia is a function of the degree of body fat depletion; the greater the degree of fat depletion, the greater the hyperphagic response (Dulloo, Jacquet, Girardier, 1997). The correlation between hyperphagia and the recovery of fat tissue persists independent of changes in lean tissue such that the recovery of

body fat occurs while the amounts of lean tissue remain unaltered (Dulloo, Jacquet, Girardier, 1997). Findings from this post hoc analysis also indicate a body fat "overshoot" of original body fat levels due to post-starvation hyperphagia. Suggestions for control mechanisms in post-starvation hyperphagia include energy partitioning between lean and fat tissue, thermogenesis, and autoregulatory signaling (Dulloo, Jacquet, Girardier, 1996; Dulloo, Jacquet, Girardier, 1997). Chronic dieters may also experience body composition changes related to post-energy restriction increases in hunger and appetite (hyperphagia) and increases in metabolic efficiency (suppressed REE) (Leibel, Rosenbaum, Hirsch, 1995). Maintenance of fat stores in a state of depletion are likely to provide a stronger hunger impulse and an increased likelihood for fat repletion overshoot (Dulloo, Jacquet, Girardier, 1997).

3. Aerobic fitness and physical activity

The health benefits of physical activity have long been recognized (Blair, et al., 1992). Conversely, physical inactivity has been identified in the etiology of numerous illnesses including cardiovascular disease, (Powell, et al., 1987), cancer (Kohl, LaPorte, Blair, 1988; Severson, et al., 1989), type II diabetes (Helmrick, et al., 1991), and osteoporosis (Snow-Harter, Marcus, 1991). It is widely accepted that regular physical activity is an important health behaviour to maximize quality of life. One of the most well documented effects of regular physical activity is a higher level of physical fitness and health (Blair, et al., 1992).

3.1 Measurement of aerobic fitness

Aerobic capacity, as measured by maximal oxygen uptake, has long been identified as one of the fundamental components of physical fitness (Berthouze, et al., 1995). The measurement of aerobic fitness can be determined by a variety of work tasks that activate large muscle groups provided the exercise is of sufficient intensity and duration to engage maximal aerobic energy transfer (McArdle, Katch, Katch, 1991). During an aerobic fitness test, the point at which

oxygen consumption plateaus and shows no further increase (or increases only slightly) with an additional work load is called the maximal oxygen consumption, maximal oxygen uptake, maximal aerobic power, max VO₂, or VO_{2max} (McArdle, Katch, Katch, 1991). It is generally assumed that VO_{2max} represents an individual's capacity for the aerobic resynthesis of ATP. Additional work is accomplished only by the energy transfer reactions of glycolysis with the resulting formation of lactic acid. Under these conditions, an individual soon becomes exhausted and unable to continue exercising.

VO_{2max} provides important information on the capacity of the long-term energy system. The attainment of VO_{2max} requires integration of the ventilatory, cardiovascular, and neuromuscular systems; this gives maximal oxygen uptake significant physiologic as well as metabolic relevance (Mitchell, et al., 1958). However, VO_{2max} is not the only determinant of aerobic work capacity. Other factors, especially those at the muscular level, such as the number of capillaries, enzymes, and fiber types, exert a strong influence on the capacity to sustain high levels of aerobic exercise (Holloszy, Coyle, 1984).

The usual exercise forms during maximal oxygen uptake assessment include treadmill, bench stepping, or cycling. However, the max VO_2 has also been measured during swimming, rowing, ice skating, and arm-crank exercise (Bonen, et al., 1980; Carey, et al., 1974; Ferguson, et al., 1969; Sawka, 1986). Ideally, performance of these tests should be independent of strength, speed, body size, and skill, with the exception of specialized tests such as swimming, rowing, and ice skating (McArdle, Katch, Katch, 1991).

3.2 Cycle ergometer to measure maximal oxygen consumption

Maximal oxygen uptake tests are either performed continuously (with no rest between work increments) or discontinuously (with the subject resting several minutes between work periods) (McArdle, Katch, Pechar, 1973). There is ample evidence to suggest that max VO₂ is the same whether measured with a continuous or discontinuous protocol (Maksud, Coutts, 1971; McArdle, Katch, Pechar, 1973). Continuous tests usually take 10 to 15 minutes, while

discontinuous tests average 65 minutes in length. Max VO₂ can usually be reached with a continuous exercise protocol where exercise intensity is increased progressively in 15-60 second intervals for a total test time of 5 minutes (Fairshter, et al., 1983).

To determine oxygen consumption during cycle ergometry, the Douglas bag method is commonly used. Special headgear is worn to which a two-way, high-velocity, low-resistance breathing valve is attached. Ambient air is inhaled through one side of the valve while expired air moves out the other side and passes directly through a gas meter that measures the volume of expired air (McArdle, Katch, Katch, 1991). This air is sampled for its oxygen and carbon dioxide composition.

Modern approaches make use of computer technology and microelectronic instrumentation to collect, measure, and compute respiratory and metabolic information (Norton, 1980). A computer is interfaced with three measuring devices: (1) an automated system that continuously samples expired air, (2) a flow meter for measuring the volume of expired air, and (3) rapid electronic oxygen and carbon dioxide gas analyzers for measuring the fractional concentration of gases in the expired air sample (McArdle, Katch, Katch, 1991). Computerized systems provide ease of operation and speed of data analysis, but they are also expensive and require on-going maintenance.

Equations have also been developed to predict the oxygen cost of cycle ergometry. The applications of such predictive equations are limited to specific populations. Researchers determined that oxygen consumption during cycle ergometry is nonlinearly related to work rate and pedal rate and that females use less oxygen for a particular work rate (Londeree, et al., 1997). The American College of Sports Medicine (ACSM) prediction equation significantly underestimated oxygen consumption in obese women (Andersen, Wadden, 1995). Data from this experiment suggest the Latin equation is more accurate than the ACSM formula for estimating oxygen consumption during loaded cycling in obese women, however, this may not apply to unloaded cycling. Greiwe, et al. (1995) found that the ACSM formula significantly overestimated VO_{2max} and

recommended that this formulae not be used when an accurate assessment of VO_{2max} if required. Predictive equations would more suitable to determining fitness of large numbers of participants when conducting complete fitness tests would not be practical or time efficient.

3.3 Variation in VO_{2max}

Regardless of the sophistication of a particular automated system, the output data are only as good as the accuracy and validity of the equipment. In large part, this depends on careful and frequent calibration of the electronic equipment using previously established standards (McArdle, Katch, Katch, 1991). The use of electronic gas analyzers may actually be more precise than earlier chemical methods. Gas analyzers may show a smaller variance for repeated estimates of the same gas sample (Howley, Bassett, Welch, 1995). The maximal error in the gas fraction is estimated to be <2% of the VO₂.

Measurement error can contribute to variation in VO_{2max} testing. Including liberal cut-off values in the determination of a subject's true VO_{2max} and unreliable measurements of gas volume (or flow) and expired gas fractions are important considerations (Howley, Bassett, Welch, 1995). Variations in the barometric pressure, the gas temperature, and the water vapor pressure of the gas have little effect on measurement error in VO_{2max} (Howley, Bassett, Welch, 1995). The use of the Douglas bag method produces VO_{2max} estimates that are relatively insensitive to small errors in these variables (Howley, Bassett, Welch, 1995).

In general, the error associated with the technical aspects of VO_{2max} measurement is less than 10% of the total error of measurement (Katch, Sady, Freedson, 1982). Researchers have typically found that the coefficients of variation are <3% for repeated measurements on a subject performing submaximal steady state exercise (Welch, Pedersen, 1981).

3.4 Measurement of physical activity

Physical activity is a behaviour that has physiological consequences

including increased energy expenditure and elevated levels of cardiorespiratory function (Pate, 1993). Improved physical fitness is often a consequence of chronic participation in exercise and/or habitual physical activity (Pate, 1993). When studying the relationship between physical activity and health, it is necessary to consider not only the total energy expenditure during a certain day or week, but the pattern of habitual physical activity over an extended period of time (Baecke, Burema, Frijters, 1982)

As with aerobic fitness, there are several methods used to assess physical activity. These have included direct observation, self-report measures, motion sensors (Pate, 1993), and heart rate monitors (Saris, et al., 1977). The most widely used procedure for measurement of physical activity is self-report in the form of self-administered questionnaires, interviewer-administered questionnaires, and physical activity diaries. These instruments have been designed to elicit information on physical activity during the recent past or to elicit a report of usual or habitual activity. The major advantages of the self-report measure are minimal time requirements and low cost. These measures can easily be used with large numbers of subjects and, consequently, are often used in epidemiological research (Pate, 1993). Disadvantages of self-report measures include limitations associated with recall and subjectivity in response to the instrument (Pate, 1993).

3.5 Baecke questionnaire

The Baecke questionnaire is a self-administered questionnaire assessing the habitual physical activity of adults (Baecke, Burema, Frijters, 1982). The questionnaire was developed for use across various socio-economic classes in the general population. This assessment tool measures three indices of habitual physical activity: work, sport, and leisure-time.

3.6 Variation and scoring using the Baecke questionnaire

The test-retest reliability of the indices of physical activity, measured over an interval of one to three months, was between 0.78 and 0.90 for the work and sport indices, and 0.74 and 0.86 for the leisure-time index (Jacobs, et al., 1993; Baecke, Burema, Frijters, 1982). Data from epidemiological research has shown the Baecke questionnaire to be valid and well within the range of values found in other studies (Pols, et al., 1995). Each section consists of several questions scored on a five-point Likert scale, ranging from never to always or very often. Scoring of this questionnaire is described elsewhere (Baecke, Burema, Frijters, 1982).

3.7 Aerobic fitness and physical activity of chronic dieters

Support and motivation are of vital importance for women wanting to successfully lose and maintain weight loss (Kayman, Bruvold, Stern, 1990). Physical activity and dietary modification are commonly used strategies, but long-term success at maintaining weight loss is rare. Chronic dieters may adopt prescriptive exercise routines to alter body shape and weight (aesthetics) while the health benefits of physical activity are seen as less of a motivator (Brownell, Steen, 1992; Sandri, 1993).

Exercise intensity may influence changes in body composition. Grediagin, et al., (1995) examined this hypothesis by measuring body composition before and after an exercise intervention among twelve untrained, overweight, but weight-stable women randomly assigned to a high-intensity (80% VO_{2max}) or low-intensity (50% VO_{2max}) exercise group. There were no significant between-group differences in weight change, percent body fat, fat mass, sum of skinfolds, or sum of circumference measurements. However, the high intensity group gained more than twice as much LBM as the low intensity group (2.0 vs. 0.8 kg). These changes in body composition were determined by hydrodensitometry. Although both groups lost similar amounts of body fat, circumference measurements detected that the relative distribution of fat loss was different between groups. The low intensity group experienced a 38% greater loss of circumference measures (waist, hips) than the high intensity group. This study suggests that body composition changes, particularly loss of fat mass, were more related to total energy expended than to achieving high intensity exercise.

The results of a recent study suggest that excessive exercise may be a contributing factor to the development and maintenance of disordered eating (Davis, et al., 1994b). However, relatively little is known about the relationship between physical activity and restrained eating among non-athletes (Tepper, Trail, Shaffer, 1996). In college and community-based samples of adults who were restrained or unrestrained eaters, differences were observed in the activity patterns between men and women. However, no gender differences were identified based on dietary restraint (Tepper, Trail, Shaffer, 1996; Klesges, Isbell, Klesges, 1992). Furthermore, these studies were unable to detect differences in activity between groups of non-athletes with respect to dietary restraint. In contrast, Kanarek and colleagues (1995) reported a positive correlation between restraint and exercise frequency in college women. Therefore, the relationships between exercise and dietary restraint require further research before stronger conclusions can be made.

Dieting without exercise results in loss of fat and lean tissue (Garrow, 1986) which is also correlated with decreases in REE (Thompson, Manore, Thomas, 1996; Wadden, et al., 1997; Phinney, et al., 1988; Welle, et al., 1984). Lean tissue was preserved during a program of strength training in obese dieting subjects, but a decrease in REE could not be prevented (Geliebter, et al., 1997). Wadden, et al., (1997) found that both aerobic and strength training did not preserve FFM better than dieting alone in obese women even after 48 weeks of training. Exercise alone has been shown to increase REE for several hours after the exercise is finished (Mole, 1990). Active skeletal muscle is the major contributor to increased REE after exercise, but coupled and uncoupled phosphorylation of mitochondria in skeletal muscle may also play a role (Mole, 1990). It is clear that exercise type, frequency, intensity, and duration must also be considered in determining the effect of exercise on REE.

4. Biochemical indices

Energy metabolism is regulated by a complex relationship between physiological and neuroendocrine factors. Energy balance, or homeostatic

control, occurs when the energy from food intake matches the energy released as heat. It is generally believed that the regulation of food intake operates through negative feedback control from the periphery (Strubbe, 1994). Signals from the periphery report to the central nervous system on the current energy flux of the body. Of the many neuroendocrine factors that regulate energy metabolism, this review will focus on the effects of insulin, leptin, and thyroid hormones.

4.1 Glucose and insulin

Glucose and lipids are the major sources of energy for the body and metabolic control is largely under the influence of the anabolic hormone insulin (Cooney, Storlien, 1994). Normally, insulin is released from the pancreatic β -cells in response to an increase in plasma glucose seen after a carbohydrate meal is consumed. Specific amino acids (arginine) along with sensory stimuli associated with food intake can also act as insulin secretagogues (Holmes, et al., 1989).

Glucose transport across the plasma membrane occurs by facilitated diffusion with glucose transporter proteins (Cooney, Storlien, 1994). Insulin acts to stimulate the uptake of glucose by increasing the translocation of specific glucose transporters to the plasma membrane (Rodnick, et al., 1992). The mechanism by which insulin exerts this effect is not entirely clear, but it does appear that many of the actions of insulin are mediated by protein phosphorylation (Cooney, Storlien, 1994). Additional physiological effects of increased plasma insulin include the conversion of glucose to glycogen (Dent, et al., 1990), glucose to carbon dioxide (Cooney, et al., 1993), and glucose to fatty acids (Buechler, et al., 1984). Insulin also inhibits gluconeogenesis by specific enzymatic alterations (O'Brien, Granner, 1990; Pilkis, 1990).

Measurement of insulin sensitivity (S_i) in humans is an important metabolic determinant of health. Decreased S_i (insulin resistance) is associated with diabetes mellitus, ischemic heart disease, and hypertension (Daly, et al., 1997). S_i is difficult to assess because of the varied physiologic actions of insulin and because of the cross-reactivity of radioimmunoassays developed to

measure insulin (Temple, et al., 1990). Measurement of fasting insulin concentrations is the most basic method of assessing S_I and it may be of particular value in screening large numbers of subjects (Daly, et al., 1997). Laasko (1993) found a strong correlation between fasting insulin concentrations and insulin levels during a hyperinsulinemic clamp, but correlations were weaker in subjects with impaired glucose tolerance. The euglycemic clamp technique is considered the criterion method for assessing S_I (DeFronzo, Tobin, Andres, 1979) as it allows for the determination of peripheral and hepatic insulin sensitivities (Steele, 1959).

Impaired glucose tolerance (IGT) is indicated by fasting plasma glucose levels intermediate between those considered normal and those indicative of diabetes (National Diabetes Data Group, 1979). IGT is associated with certain conditions and syndromes such as: (1) pancreatic disease, (2) hormonal abnormalities (catecholamines), (3) drug or chemical induced conditions, (4) insulin receptor abnormalities, (5) certain genetic conditions. In some subjects, IGT may represent a preliminary stage in the development of non-insulin dependent diabetes mellitus (NIDDM) or insulin dependent diabetes mellitus (IDDM) (Ferrannini, 1997; Davidson, 1995). However, research is inconclusive about the contribution of IGT to the co-morbidities of diabetes. The majority of persons with IGT remain in this class for many years or return to normal glucose tolerance after a period of time (Home, 1997). IGT remains a consideration for the determination of metabolic fitness.

4.2 Leptin

Leptin is a 16 kDa plasma protein synthesized in adipose tissue. It has been identified as the mutant gene product that leads to obesity in ob/ob mice (Pelleymounter, et al., 1995; Zhang, et al., 1994). Mice inheriting a copy of the *ob/ob* gene from both parents exhibit marked obesity, hyperphagia, glucose intolerance, insulin resistance, low energy expenditure, and sterility (Chehab, Lim, Lu, 1996). Injection of exogenous recombinant leptin into these mice reverses the metabolic abnormalities (Halaas, et al., 1995).

In humans, plasma leptin levels correlate well with body weight (Considine, et al., 1996), BMI, and subcutaneous fat, but not visceral fat (Takahashi, et al., 1996). In addition, expression of leptin messenger ribonucleic acid (mRNA) in adipose tissue is greater in obese than in lean individuals (Hamilton, et al., 1995). The increased leptin mRNA found in obese individuals may explain their abundance of plasma leptin.

It has been postulated that leptin is part of the complex feedback mechanism that regulates body fat levels. This inherent control of body fat is commonly termed the set point, lipostat, or leptinstat (Caro, et al., 1996). When actual adipose mass deviates from the internal standard, compensatory efforts are initiated to minimize the change and return the body to its status quo (Bennett, 1995). It is unknown how the body compares the actual amount of body fat with the internal set point standard and then minimizes the difference between the two. However, it is clear is that the set point is regulated by both genetics and environment (diet, physical activity, stress); two key determinants of behaviour that are well known to regulate body weight and body composition (Caro, et al., 1996).

After leptin is secreted from the adipocytes, the hormone interacts with hypothalamic receptors and decreases the amount of neuropeptide Y (NPY) production (Schwartz, et al., 1996). NPY regulates energy homeostasis by stimulating food intake and lowering energy expenditure (Stephens, et al., 1995). Suppression of NPY by leptin may involve activation of the sympathetic nervous system by increasing energy expenditure via the β_3 -adrenergic receptor in adipose tissue (Collins, et al., 1996). If this is the case, leptin levels may be associated with REE. Still, more information is required to determine if this relationship is evident among different sub-populations (including female chronic dieters).

4.3 Thyroid hormones

The thyroid gland is the site of thyroid hormone synthesis. This gland is composed of many acini, spherical in shape and surrounded by a single layer of

thyroid cells. The acini, or follicles, are filled with colloid in which thyroid hormones are formed. The thyroid cells actively collect iodine to synthesize the thyroid hormones. Sixty micrograms of iodine must be trapped daily to maintain an adequate supply of the thyroid hormones (Clugston, Hetzel, 1994). A detailed review of thyroid hormone production is described elsewhere (Taurog, 1986).

The two main forms of thyroid hormone are thyroxine (T_4) and triiodothyronine (T_3) . The plasma concentration of T_4 is nearly 50 times that of T_3 , and therefore is considered to be the major thyroid hormone, despite the fact that T_3 is many times more effective at influencing metabolism on an equal molar basis (Groff, Gropper, Hunt, 1995b). In the circulation, T_4 and T_3 are associated with transport proteins and are distributed to target cells in peripheral tissues. Thyroid-binding globulin (TBG) has the smallest capacity, but the greatest affinity for T_4 and T_3 . Albumin and transthyretin (prealbumin) also transport thyroid hormones. A very small fraction of T_4 and T_3 is not bound to transport proteins in the blood and exists in a 'free' form. The free form is available to cell receptors and is considered the most "hormonally active" (has the greatest influence on effector sites). Several tissues including the liver, kidney, brain, pituitary, and brown adipose tissue can deiodinate T_4 to generate T_3 and rT_3 (the inactive form of T_3). Most T_3 in the blood has been synthesized in the liver from T_4 .

The biological effects of thyroid hormones are demonstrated by increased mRNA and protein synthesis after hormone receptor attachment (Groff, Gropper, Hunt, 1995b). Other possible mechanisms of thyroid hormone action include modulation of Na $^+$ /K $^+$ -ATPase transport systems, of β -adrenergic receptor sensitivity, and of neurotransmitters (Groff, Gropper, Hunt, 1995b).

Thyroid hormones influence many aspects of energy metabolism. Most of the metabolic effects that are mediated by β -adrenergic receptors are induced by a stimulation of membrane adenylate cyclase which causes an increase in production of intracellular cyclic AMP (Andersson, 1982). The intracellular signaling that influences metabolic rate is unknown, but it does not appear to be mediated through T₃. Welle et al., (1991) reported that the reduction in REE with a β -adrenergic receptor blocker is independent of changes in T₃ concentrations.

Thyroid hormones have a direct effect on metabolism including the stimulation of basal metabolic rate, oxygen consumption, and heat production (Groff, Gropper, Hunt, 1995b). Thyroid hormones also seem to affect insulin action mainly in the liver (Sandler, et al., 1983) although the cellular mechanism is poorly understood.

4.4 The metabolic/hormonal profile of chronic dieters

Research has demonstrated that insulin, leptin, and thyroid hormones influence various aspects of metabolism in chronic dieters, including energy expenditure and body composition. Furthermore, there exist inter-relationships between each of the aforementioned hormones such that they may work in conjunction with each other to influence metabolism (for example, insulin and leptin). These hormonal relationships are extremely complex and continue to remain inadequately conceived in spite of the latest scientific scrutiny.

There appears to be a strong relationship between abdominal fat and insulin metabolism in obese individuals. Until recently, the relationship between abdominal fat and insulin resistance in non-obese women was unknown. Carey, et al., (1996) demonstrated that DXA central fat measurements (which included visceral and some subcutaneous fat) had significant metabolic associations (insulin resistance), independent of total and non-abdominal adiposity. In fact, centrally distributed abdominal fat may have greater effects on insulin sensitivity in the non-obese than in the obese (Carey, et al., 1996).

Weight cycling induced by feeding alternating amounts of a modified high fat diet has been shown to increase blood insulin concentrations that results in insulin resistance (Lu, et al., 1995). Female Wistar rats were fed alternating amounts of a modified high fat diet (similar fat content as high fat chow, but with double the amount of protein, vitamin, and minerals) and 50% of a modified high fat diet (to induce weight loss). Using three cycles of weight change, weight gain among the cycled animals was not different when compared to animals only consuming a high fat control diet. However, animals fed the modified fat diets were heavier and fatter after three cycles of weight loss and regain than animals

consuming the low fat control diet (p<0.001). Associated with increased fat mass in the weight-cycled rats was an elevation of adipose tissue lipoprotein lipase. Although the weight-cycled rats were not heavier or fatter than the non-weight cycled rats consuming the high fat control diet, they exhibited increased blood insulin concentrations and insulin resistance compared to non-weight cycled controls. This study provided an animal model for the enhanced predisposition to diabetes in association with weight cycling. Research has been inconclusive with respect to the metabolic effects of weight cycling in humans (National Task Force on the Prevention and Treatment of Obesity, 1994).

A reduction of 10% in body weight is associated with a 53% reduction in serum leptin (Considine, et al., 1995). This finding suggests that the regulation of leptin is dependent on more than the size of the adipose tissue depot. Caloric intake may be a more sensitive leptin regulator. Progressive weight loss during hypocaloric dieting is accompanied by a decline in circulating leptin levels and adipose tissue mRNA expression (Kolaczynski, et al., 1996a); however, plasma leptin levels then increase once isocaloric diets are initiated to maintain the reduced body weight (Maffei, et al., 1995).

Because leptin appears to be involved in both conserving energy during periods of food deprivation and preventing obesity during periods of energy excess, the relationship between insulin and leptin has been investigated (Cusin, et al., 1995; Saladin, et al., 1995; Kolacynski, et al., 1996).

Body weight preservation under conditions of extreme exercise and restrained eating has been extensively explored. Loucks and Heath (1994) found associated reductions in T₃ hormone levels with energy deficiency in exercising women. These results demonstrate that the energy cost of aerobic exercise can induce 'low T₃ syndrome' in women. This effect may be prevented by increasing the dietary intake to compensate for the energy cost of exercise (Loucks, Heath, 1994; Loucks, Callister, 1993). Therefore, energy balance is required to sustain normal T₃ levels.

Spontaneous hyperthyroidism is a clinical situation that frequently impairs glucose tolerance after both oral and intravenous glucose and after mixed meals

(Ikeda, et al., 1990; Yamada, et al., 1991). Gonzalo, et al. (1996), demonstrated that glucose tolerance was not significantly affected by hyperthyroid in normal weight women. However, moderately overweight women (mean BMI = 28.0 ± 0.8 kg/m²) presented with a clear state of insulin resistance (whether they were hyperthyroid or not). A possible explanation for these findings may be that obesity produces an impairment of both non-oxidative and oxidative glucose metabolism (Bonadonna, DeFronzo, 1992). Therefore, the disturbed non-oxidative pathway present in hyperthyroid patients can not be compensated by an increase in oxidative glucose metabolism if excess weight exists (Gonzalo, et al., 1996). The complex relationship between thyroid hormones and carbohydrate metabolism warrants further investigation, especially among female chronic dieters.

5. Dietary intake

Accurate assessment of dietary intake in free-living humans remains one of the most basic and fundamental problems in the study of nutrition (Wolper, Heshka, Heymsfield, 1995). A variety of assessment techniques are utilized to quantify dietary intake for the study of energy balance.

5.1 Measuring dietary intake

Researchers must choose among existing techniques with careful attention to the balance of desirable and undesirable qualities of each method as they relate to specific study goals (Wolper, Heshka, Heymsfield, 1995). Ideally, any technique used to measure food intake should not in any way interfere with the subject's dietary habits and thus alter the parameter being measured (Westerterp, 1994). The standard methods of determining dietary intake are: (1) indirect determination based on group consumption data, inspection of family budgets, larder inventories, agricultural production, (2) estimation by recall of food consumed over the last day, week, month, or even longer, and (3) direct measurement and recording of food intake as eaten (Westerterp, 1994). These standard methods can be classified into two major groups: (1) quantitative daily

consumption methods (recalls and records), and (2) retrospective methods (dietary histories and food frequency questionnaire (FFQ)) (Gibson, 1990). Quantitative methods are designed to measure the quantity of food consumed over a defined period. The assessment of usual intake is particularly critical when relationships between habitual diet and biological parameters are assessed (Gibson, 1990). Retrospective methods obtain information on the patterns of food use during a longer, less precisely defined time period. Such methods are most frequently used to assess usual intakes of food or specific classes of foods. With modification, these methods can provide data on usual nutrient intakes (Gibson, 1990).

5.2 Food frequency questionnaire

A FFQ is designed to obtain qualitative, descriptive information about usual food consumption patterns (Gibson, 1990). The questionnaire consists of a list of foods as well as a set of frequency-of-use response categories (Gibson, 1990). The list of foods may focus on specific groups of foods, particular foods, or foods consumed periodically in association with special events/seasons (Anderson, 1986). In general, FFQ are inexpensive, quick, and easy to standardize. The data for the FFQ may be obtained by a standardized interview or self-administered questionnaire, both taking approximately fifteen to thirty minutes to complete. The FFQ imposes fewer burdens on respondents than most of the other dietary assessment methods. It is often used by epidemiologists studying associations between long-term dietary habits and disease (Hirayama, 1981), although its use for estimating usual food intakes has been demonstrated as well (van Staveren, et al., 1986). It has been suggested that underreporting of intakes may be associated with increased dietary restraint (Sawaya, et al., 1996). FFQ may reduce the likelihood of underreporting due to the retrospective nature of the dietary assessment (Gibson, 1990), although this bias is inherent in all dietary assessment methods.

5.3 Measuring dietary intake with FFQ

The FFQ typically consists of precoded forms that incorporate a selected list of essential foods arranged in food groups of comparable nutrient content (Wolper, Heshka, Heysmfield, 1995). Questions are posed relating to portion sizes and frequency of consumption; specific amounts are generally described in common household measurements. Individuals are asked to indicate how often they eat each food during a specified period of time in addition to answering questions about daily consumption (Wolper, Heshka, Heysmfield, 1995). The data from the questionnaire are often used to rank subjects into broad categories of low, medium, or high intakes of certain foods, usually based on tertiles (Gibson, 1990). Food scores can be calculated from FFQ based on the frequency of consumption of certain food groups (Gibson, 1990). These scores can be examined in relation to psychosocial influences (level of education, income), personal demographics (age, weight, marital status), season, and geographic distribution. The use of the FFQ to quantify usual intake produces semiquantitative food frequency data. If this semiquantitative data is desired, nutrient scores for each person can be computed by multiplying the relative frequency that each food item is consumed by the nutrient content of the average portion size specified (Gibson, 1990). The nutrient content is obtained from appropriate food composition data (Russel-Briefel, Caggiula, Kuller, 1985).

5.4 Variation in FFQ

Studies on the precision of the FFQ are limited. However, one research group found that after a three month lapse, 90% of the responses on the repeated FFQ did not differ from the original measurement (Acheson, Doll, 1964). Research from Mullen, et al., (1984) demonstrated that a large proportion of individuals could accurately estimate their food intake using a FFQ. In another study, after a one year lapse between self-administered FFQ, correlation coefficients ranged from 0.49 for total vitamin A to 0.71 for sucrose (Willett, et al., 1985). Sawaya, et al., (1996) reported that FFQ was the only dietary assessment method that correlated significantly with total energy expenditure in

comparison to weighed 7-day food intake and 24-hour food recall among normal weight young women (BMI 20.9 \pm 1.9 kg/m² and age 25.2 \pm 3.5 years). The previous assumption that FFQ typically overestimate (Zulkifli, Yu, 1992) usual food intake appears to be incorrect with respect to these data.

Other errors associated with FFQ include inter- and intra-variability, subject age, gender, day-of-the-week effects, seasonal effects, and training effects (Gibson, 1990). These types of errors can be minimized if the initial design of the dietary assessment protocol is maintained. Measurement errors in dietary assessment include respondent bias, interviewer bias, respondent memory lapse, incorrect estimation, supplement usage, coding/computation errors, and 'flat slope syndrome' (heavier individuals under-predict dietary intake and underweight individuals over-predict dietary intake) (Gibson, 1990). Errors associated with determining nutrient content of food from food composition data include inadequate sampling protocols, inappropriate analytical methods, lack of standardized conversion factors, inconsistencies in terminology, incorrect description of individual food items, and inconsistencies from genetic, environmental, food preparation, and processing factors (Gibson, 1990).

There is no ideal method for assessing food or nutrient intakes. The method of choice depends on the objectives of the study (Gibson, 1990). None of the current methods are devoid of systematic errors or prevent individuals from making alterations in their food habits.

5.5 Dietary intake of chronic dieters

Because of the potential lower energy intakes, chronic dieters are recognized as 'at risk' for marginal malnutrition and fatigue. Chronic dieters tended to consume foods in an erratic pattern characterized by restrictive phases with intakes of 1000 calories of less (Grodner, 1992). It would be challenging to meet all nutrient recommendations for most age and gender categories at this level of energy intake. Nutrients of particular concern include iron, zinc, and calcium. Fatigue has been associated with low iron and low energy intakes. Low energy intakes may trigger a reduced metabolism that would complicate further

weight loss. Long-term marginal intakes of calcium were shown to be a risk factor for the development of osteoporosis (Whitney, Cataldo, Rolfes, 1991).

Research studies have demonstrated that restrained eaters consume up to 400 fewer calories per day than unrestrained eaters (Laessle, et al., 1989a; Tuschl, et al., 1990b). Restrained eaters may consume fewer portions of oils, margarines, butter, and high fat dairy products, but greater amounts of low-fat dairy products (Tuschl, et al., 1990a). Other researchers have provided additional evidence which showed restrained eaters consumed less fat and more carbohydrate than unrestrained eaters (Tepper, Trail, Shaffer, 1996).

Reduced dietary fat intakes have been shown to decrease REE and leptin values. Havel, et al., (1996) studied 8 lean/normal weight and 7 overweight/obese women during a self-selected, very low fat diet (<15% of energy from fat) for eight months. REE decreased by 7.7 ± 2.3% in women losing more than 7% body weight but did not change in women who lost less than 7% body weight. These decreases in REE were significantly and positively correlated with the change of BMI, % body fat, and plasma leptin. Overweight women experienced a greater absolute and proportional decrease in plasma leptin after the low fat diet. For example, a 10% decrease in adiposity in overweight women led to a 34% decrease in plasma leptin, whereas the same proportional change in adiposity only decreased plasma leptin by 13% in lean women. This type of diet is similar to the dietary practices of female chronic dieters as highly restrained eating is associated with decreased fat intake (van Strien, et al., 1986). Expectations are that chronic dieters engaging in long-term dietary fat restriction will experience similar decreases in plasma leptin and REE, which may contribute to the strong tendency for weight regain in these individuals.

6. Dietary restraint

Several psychological theories attribute obesity, eating disorders, weight reduction, and weight maintenance to dietary restraint. The construct of dietary restraint is generally described as a person's tendency to eat less than desired.

Common among restrained eaters is the behaviour of eating in response to emotions or external stimuli regardless of the internal state of hunger or satiety (Bruch, 1961; Schachter, Goldman, Gordon, 1968). In both psychosomatic and externality theory, an individual's misperception of his/her internal state prior to eating is considered to be a causal factor in the development of obesity (Robbins, Fray, 1980). Individuals may react to being overweight by consciously restricting food intake irrespective of whether they are emotional eaters (Rodin, 1978). However, a high degree of externality or a strong tendency to emotional eating does not necessarily lead to an overweight physique (van Strien, et al., 1986). Since dietary restraint is commonly used to manipulate body weight or fight against a predetermined set point (Cash, Henry, 1995), a high degree of emotional and external eating can be found in all weight categories.

6.1 Measurement of dietary restraint

There are three main dietary restraint assessment questionnaires used for research purposes (Stunkard, 1981; Polivy, Herman, Howard, 1988; van Strien, et al., 1986). Herman and Mack (1975) developed a five-item scale to measure chronic dieting. After subsequent revisions, the present form of the Restrained Eating Scale (RS) was published (Herman, Polivy, 1980). Stunkard and Messick (1985) built on the work of Herman and Polivy by adapting the RS to include several facets of eating behaviour. Three factors emerged from the factor analysis (cognitive control of eating, disinhibition, and susceptibility to hunger), thus the scale originally appeared as the Three-Factor Eating Questionnaire (Stunkard, Messick, 1985). The scale is presently known as the Eating Inventory (Stunkard, Messick, 1988).

6.2 Dutch eating behaviour questionnaire (DEBQ)

van Strien, et al., (1986) noted that the RS was multi-factorial because it contained both a weight fluctuation factor and a concern for dieting factor. Although many theoretical and empirical works on eating behaviour and dieting addressed restrained eating, emotionally triggered eating, and eating in

response to external or internal hunger cues, the measures of eating behaviour did not always make clear distinctions among these constructs (Gorman, Allison, 1995). Thus, the Dutch Eating Behaviour Questionnaire (DEBQ) was developed. The initial item pool for the DEBQ consisted of 100 items with some items derived from previous research (van Strien, et al., 1986) and others coming from Pudel's latent obesity scale (Pudel, et al., 1975). The most current version of the DEBQ consists of 33-items and is used to assess three separate factors of eating behaviour: restrained eating, emotional eating, and external eating.

6.3 Variation in DEBQ

The items of the DEBQ were derived through repeated applications of factor and item analysis, so it is not surprising the scale's internal consistency, as measured by Cronbach's α , is quite high (usually > 0.90) (Gorman, Allison, 1995). Allison, et al., (1992) found the test-retest reliability to be 0.92 over a 2 week period. The derived solutions appear to be stable across gender and relative weight categories (van Strien, et al., 1986).

It appears that the DEBQ may come closest to a pure dietary restraint scale (Gorman, Allison, 1995). The questionnaire has a single-factor structure and has high communality with other restraint scales, especially with subscales that address the cognitive aspects of restraint (Gorman, Allison, 1995). Additionally, the scale was constructed so that its items reflect restraint rather than disinhibition, hunger, or weight fluctuation (Gorman, Allison, 1995). It has been suggested that the DEBQ selects for dieters who vary in their levels of success at food restriction, but without specifying resulting weight fluctuations or excessive eating behaviour when dieting attempts do not succeed (Ogden, 1993).

6.4 Dietary restraint in chronic dieters

When the DEBQ was used to differentiate between dieters who describe themselves as successful and those who do not, the failed dieters had the highest scores and the successful dieters had the lowest (Ogden, 1993). This result implies that dietary restraint is associated with unsuccessful dieting. Furthermore, obese individuals had significantly higher scores on restrained eating than normal weight subjects (van Strien, et al., 1986). This finding suggests that obese individuals consider exercising dietary restraint to be an acceptable behaviour.

When comparing restraint scores between groups of dieters ranging from Weight Watcher participants, individuals with anorexia, and individuals with bulimia, no differences were detected (Wardle, 1987). However, all scores were above average when compared to normal weight, non-dieters (p<0.05). Based on these results, the DEBQ may not identify differences between the eating styles characterized by disordered eaters.

C. FACTORS INFLUENCING BODY IMAGE OF CHRONIC DIETERS

Body image disturbance, especially among females, has become so prevalent that it is commonly termed a normative discontent (Cash, Pruzinsky, 1990; Fallon et al., 1994; Rodin et al., 1984). In a recent national survey, over one-half of women polled held an unfavorable view of their appearance (Garner, 1997). When comparing these results to earlier data from 1972 (Berscheid, Walster, Bohrnstedt, 1973), 1985 (Cash, Winstead, Janda, 1986) and 1995 (Cash, Henry, 1995), it is apparent that the body images of women have become increasingly negative (Garner, 1997).

1. Developmental theories

There are several theories that attempt to explain the complex process of body image formation. Each explanation encompasses a variety of developmental, psychological and social constructs yielding three common themes: (1) sociocultural pressure, (2) comparisons between self and others, and (3) individual attitudes about the importance of appearance.

Some cultures (particularly Western cultures) encourage the 'thin body cult', an appearance consistent with extreme thinness (Slade, 1994). This places increased pressure on vulnerable individuals (young women) to achieve a body

size that may be inconsistent with genetic determinants of body shape and health (Wilfley, Rodin, 1995; Brownell KD, 1991). An unattainable beauty ideal is manifested and sustained through a variety of media, fashion, and weight-loss industries (Stice, Shaw, 1994; Silverstein, Perdue, Peterson, 1986).

A woman's tendency to compare her own body to that of other individuals is strongly related to overall levels of body dissatisfaction (Heinberg, Thompson, 1992; Striegel-Moore, McAvay, Rodin, 1986; Thompson, Heinberg, Tantleff, 1991). Moreover, ideals often reflect distorted beliefs about what the other gender truly finds attractive (Fallon, Rozin, 1985). Jacobi and Cash (1994) indicated that most individuals are less extreme and more flexible in their heterosexual standards of appearance than either gender assumes.

Certain activating events such as comparison, looking in the mirror, exercising, or weighing can initiate appearance schematic processing (Cash, 1996). Appearance schematic individuals are those concerned with defining 'self' solely through physical appearance. If an individual is susceptible to appearance schema, the activating events can lead to body image dissatisfaction.

2. Measurement of body image

The key to a successful measurement tool is that it is easy to administer, interpret, and is psychometrically sound. There are four main types of body image assessment measures including generic dissatisfaction, perception, subjective, and behaviour indices. These tools can take the form of questionnaires or figural stimuli. Limitations to assessment strategies take the form of methodological issues including experimenter instructions, actual size of the subject, and sample validation. Many of the extant questionnaires are limited because of their development and validation on a narrow range of subject samples (female, Caucasian, college students). The results from these measures are difficult to generalize to males or individuals of various ages, ethnicities, and educational backgrounds.

2.1 Multidimensional body-self relations questionnaire (MBSRQ)

The MBSRQ is a 69-item self-report inventory for the assessment of self-attitudinal aspects of the body-image construct (Cash, 1994). The instrument was developed to take into account cognitive and behavioural as well as affective components of body image. The difference between the MBSRQ and other extant body image assessment tools is that the MBSRQ samples three attitudinal dimensions: affect, cognition, and behaviour related to three *somatic domains*: appearance, fitness, and health/illness (Brown, Cash, Mikulka, 1990). This three by three conceptual matrix yields nine multi-item subscales. Since its development, the MBSRQ has evolved through several empirical studies and, over time, researchers reduced the nine basic subscales to six (Table 2).

Table 2: MBSRQ 3 X 2 conceptual matrix

	Appearance	Fitness	Health
Evaluation	Appearance Evaluation	Fitness Evaluation	Health Evaluation
Orientation	Appearance Orientation	Fitness Orientation	Health Orientation

According to Cash, Winstead, and Janda (1986), evaluation scales reflect how good or bad one feels about each of the three somatic domains: appearance, fitness, and health; while orientation scales measure how personally important the various aspects of body image are and how actively a person does things to maintain or improve his or her body's appearance, fitness, and health. A final factor subscale was added to the MBSRQ (Illness Orientation) before the instrument was cross validated in a factor-analytic study of the original database (Brown, Cash, Mikulka, 1990).

In addition to its seven factor subscales, the MBSRQ includes three subscales: (a) The Body-Areas Satisfaction Scale (BASS) approaches body-image evaluation as satisfaction-dissatisfaction with discrete body features. (b) The Overweight Preoccupation Scale assesses fat anxiety, weight vigilance, dieting, and eating restraint. (c) The Self-Classified Weight Scale assesses self-appraisals of weight from underweight to overweight (Cash, 1994).

Considerable work has been done on the factor analysis of the MBSRQ.

The results from this work has shown the MBSRQ to be reliable and valid (Brown, Cash, Mikulka, 1990). The MBSRQ compares both genders on body image dimensions.

3. Body image of chronic dieters

The results from two large national surveys indicate that 24% of men and 40% of women are currently dieting and that the prevalence of dieting is greater than the prevalence of obesity (Horm, Anderson, 1993: Serdula, et al., 1993). Thus, it appears that many nonobese individuals (primarily women) are dieting. The dieting mind-set typically includes preoccupation with shape and weight, perceived deprivation, and dysfunctional beliefs about food and exercise (Lowe, 1993). Mossavar-Rahmani, et al., (1996) found that the more inaccurate the body size estimation, the greater the likelihood of dieting. Thus, dieting and body image cognitions, affects, and behaviours are fundamentally linked.

The psychological mechanisms involved in the maintenance of dieting behaviours are particularly insidious (Foreyt, Goodrick, 1993). Chronic dieters disrupt their natural physiological homeostasis and thus induce a variety of associated psychological responses. Before starting a very-low-calorie-diet, overweight individuals overestimated their body size, but post-treatment the same individuals underestimated their body size (O'Neil, Jarrell, 1992). This misconception could predispose an individual for future disappointment in terms of relapse and decreased self-worth when cognitions are largely discrepant from actual body sizes.

Repeated failures to control weight might reduce one's feeling of self-efficacy (Foreyt et al., 1995). Furthermore, reduction in self-efficacy due to weight fluctuation may add to feelings of depression (Rosen, Gross, Vara, 1987; Foreyt et al., 1995)

Unlike nondieting individuals, restrained eaters eat more after a fattening preload (milkshake) than after no preload or a preload they perceive to be dietacceptable (salad) even if it contains the same amount of calories as the fattening preload (Herman, Polivy, Esses, 1987; Knight, Boland, 1989; Wing et

al., 1995). Once the preload 'breaks the diets' of the restrained eaters, they feel free to indulge in the forbidden foods that are provided. Dieters will likely exhibit weight obsession, poor self-image, disordered eating patterns, poor nutrition, and disordered lifestyles, often marked by excessive or inadequate exercise (Omichinski, Harrison, 1995). These behaviours are associated with cyclic patterns of restraint, overeating, guilt, and further dietary restraint. Patterns of restraint will continue although body weight may not change *per se*.

Dieting has been termed the 'outside-to-inside' approach to change body image and it is one of the most widely practiced body image remedies among North Americans today (Rosen, 1996). This assertion most often pertains to the portion of women dieting to lose weight who are not obese. In terms of body image disturbance, dieting serves to undermine the multidimensional body image construct; perception, cognition, affect, and behaviour. In addition, a negative body image can potentiate depression (Noles, Cash, Winstead, 1985), social anxiety, sexual difficulties, and low self-esteem (Cash, Pruzinsky, 1990).

D. SUMMARY OF LITERATURE

From the research presented, it appears that chronic dieting has effects on REE, body composition, aerobic fitness, biochemical indices, dietary intake, dietary restraint, and body image. Energy homeostasis appears to be a common link between all of these variables. Whether each variable responds parallel to a change in energy expenditure is not known. Thus, women with either a high or a low REE were investigated to further assess the relationships between energy metabolism and associated metabolic variables.

CHAPTER THREE EXPERIMENTAL DESIGN AND METHODOLOGY

A. EXPERIMENTAL DESIGN

1. Initial research

Prior to the present study, an initial study was conducted which involved measuring REE of female chronic dieters. The purpose of the study was to examine if specific lifestyle factors were associated with a reduction in REE in women who had a long history of dieting. A reduced REE was defined as (≤85% of the Mifflin-St. Jeor prediction equation (Mifflin, et al., 1990).

All of the participants in the present study (n=32) had to meet the criteria for chronic dieting syndrome (Grodner, 1992). Of the total initial sample (n=172), 60% of the women thought that they would have a low REE. In actuality, only a small subset of women (n=30; 17.4%) had a reduced REE. Preliminary analysis showed that this group had: (1) started dieting at a younger age, (2) had reached a higher maximum weight as an adult, and (3) had dieted more frequently last year than the remainder of the participants.

From the initial study, it became apparent that all chronic dieters could not be categorized has having a reduced REE. Although there are many similarities between female chronic dieters with either high or low REE, no studies have been conducted to compare the physiological differences between these two groups. The present study was designed to investigate the physiological and psychological influences of two groups with high or low REE to better understand the profile of chronic dieting syndrome.

2. Research design

The present study was conducted at the University of Alberta, Department of Agricultural, Food, and Nutritional Science, Edmonton, Alberta, Canada. The research was designed as an observational study comparing two groups of female chronic dieters with high or low REE. Chronic dieters were chosen

because few research studies have examined their physiological characteristics.

Subjects were recruited from the sample of chronic dieters in the initial study. Participation in the present study was based on eligibility criteria including women, aged 25-49 years, with predicted REE either ≤85% or >100% of predicted. Initially, the age range was limited to 25 to 49 years to reduce the variability in sample characteristics and to allow for comparison of dietary intake to the corresponding Recommended Nutrient Intake (RNI) category of the same age range. However, a subsequent decrease in the lower limit from 25 to 21 years of age was made to increase subject enrollment. This modification allowed for one additional participant. Additional eligibility criteria included no chronic illness, non-smoking, and no long-term use of medications that are known to affect carbohydrate metabolism or metabolic rate. Participants were screened as to oral contraceptive pill use and no differences in pill use were detected between high and low REE groups.

A total number of 32 participants were initially proposed for the study. The sample size calculation (see Appendix A) estimated that 16 subjects were required to detect significant differences between groups in terms of lean body mass. Appropriately, an equal number of participants comprised the high and low REE groups. These participants represented an estimated 18.6% of the initial study sample.

B. METHODOLOGY

The methodology used in the comparison of chronic dieters on the basis of physiological and psychological factors is outlined below.

1. Anthropometric measurements

Height was measured once at baseline, without shoes, and was determined to the nearest 0.1 cm using a stadiometer. Weight was documented during both REE testing periods and during the VO_{2max} fitness test. Weight was measured using a medical balance beam scale (Healthometer, Continental Scale Corporation, Bridgeview, IL). Additional indirect measurement of body

weight was available from total body composition determination using DXA (referring to the sum of lean body mass, fat mass, and bone mass to represent total body weight). Weight was recorded to the nearest 0.1 kg and participants were weighed in light clothing.

2. Resting energy expenditure

Technically, the rate of oxygen consumption is calculated from minute ventilation (the rate of inspiration (VI) and expiration (VE)), but it is very difficult to measure both VI and VE accurately. Therefore, the Haldane transformation is used; it assumes that nitrogen (N_2) is the same in the inspired and expired gases. In other words there is no net uptake of N_2 (SensorMedics Corporation, 1995). The actual calculation of oxygen consumption (VO_2) is made using the Haldane formula displayed below:

$$VO_2 = \frac{(1-FEO_2-FECO_2)}{1-FIO_2} * (FIO_2-FEO_2) * VI$$

The rate of carbon dioxide production is calculated in a similar manner using VE, FECO₂ and FICO₂.

Standard equations programmed into the indirect calorimeter software convert respiratory gases to REE. The Weir equation is commonly used for this purpose. The complete Weir equation requires the entry of a 24-hour urinary nitrogen measurement that accounts for the incomplete breakdown of protein when metabolized in the body (nitrogen is excreted as a by-product of protein metabolism). However, the difference in the REE before and after this adjustment is minimal (<2%) and is usually omitted (SensorMedics Corporation, 1995; Cunningham, 1990; Westenskow, Schipke, Raymond, 1988). The complete Weir equation is shown below:

REE = $3.94 [VO_2 (mL/min)] + 1.06 [VCO_2 (mL/min)] \times 2.17 [UN (g/24 hrs)]$ The now classic Weir equation remains the standard procedure for the calculation of energy expenditure (Cunningham, 1990).

REE was measured by indirect calorimetry using a metabolic cart (VMax 29N, SensorMedics, Yorba Linda, CA). All measurements were obtained at the Department of Agricultural, Food, and Nutritional Science, Metabolic Testing

Lab, Room 4-04. Participants had their REE measured twice during the follicular phase of the participants' menstrual cycle to confirm their placement in either the high or low REE group. These two measurements were completed approximately 6 months apart. The average of the two measurements was used as the final value for comparison. Participants were excluded from the study if their weight or REE fluctuated significantly over the course of two measurements.

Participants drove to the Metabolic Testing Lab to have their REE tested. Tests were completed in the morning as all participants were in a fasted state. The entire test protocol was explained to the participants before the test began. To reduce the amount of variation involved in measuring REE, the following standard protocol was followed: (1) The participant was in a fasted state (overnight fast of 12 hours). (2) The participant was rested, in a supine position or a semi-supine position for at least 30 minutes before the start of the test. (3) The testing environment remained thermoneutral for the duration of the test. (4) The participant avoided use of stimulants including caffeine, tobacco, or medication before the test. (5) The participant refrained from intense physical activity for a minimum of 24 hours before the test. (6) The participant remained awake, but relaxed during the entire test. (7) The participant refrained from voluntary skeletal muscle activity throughout the test. (SensorMedics Corporation, 1995).

There is conflicting research as to the influence of menstrual cycle on REE. Weststrate's (1993) findings indicate that the phase of menstrual cycle does not significantly influence REE, however another researcher has found higher values during the luteal phase (Webb, 1986). Thus, the research performed in our laboratory requires women to be in the follicular phase (days one to seven) of their cycle during the measurement of REE (McCargar, et al., 1996b).

The metabolic cart was calibrated against a reference mixture of oxygen and carbon dioxide gas before each test. REE measurements were performed while participants rested in the supine position in a darkened and quiet room following a 30-minute rest period. Oxygen consumption (VO₂) and carbon dioxide

production (VCO₂) were measured from breath samples collected from an overhead transparent canopy system. For the duration of the test, participants remained awake, but motionless.

To calculate REE, a minimum of 15 minutes of steady state measurements were averaged. This typically required a total test period of 20 to 40 minutes. A respiratory quotient (RQ) of 0.85 or less was considered to reflect a fasted subject. REE was calculated based on the Weir equation (Weir, 1949) which was adapted for use without the collection of urinary nitrogen. In the present study, the following equation was used to calculate actual REE:

REE (kcal/day) =
$$3.9 [VO_2 (mL/min)] + 1.1 [VCO_2 (mL/min)] \times 1.41$$

To determine whether the participant had a high or a low REE, actual REE was compared to expected REE. The expected REE for these participants was calculated using the Mifflin-St. Jeor equation (Mifflin, et al., 1990). The Mifflin-St. Jeor equation was determined to accurately predict the REE of healthy adults. If the actual REE was equal to or greater than the expected REE, the participant was placed in the high REE group. If the actual was at least 15% less than the expected REE, the participant was placed in the low REE group. The following equation was used to calculate predicted REE:

REE (kcal/day) =
$$10 [weight (kg)] + 6.25 [height (cm)] - 5 [age (yrs)] - 161$$

The measured value for REE was expressed as kcal/kg body weight, and kcal/kg LBM to adjust REE for differences in total body mass and LBM.

3. Body composition

Body composition measurements were performed on one occasion, at the beginning of the study after the REE measurements were completed. In every case, body composition was measured at the same location (Medical Imaging Consultants, College Plaza, Edmonton, Alberta) by a trained technician using standardized procedures. Participants were not pregnant at the time of the DXA measurement.

DXA technology assess body composition using a three-compartment model (assesses LBM, fat mass, and bone mass) DXA uses an x-ray tube and a

K-edge filter to generate and direct two energy levels of photons through tissue. Attenuation of the photon beam results in an exponential decrease in flux by absorption in tissue outside and over the bone, similar to dual-photon absorptiometry (Pierson, et al., 1991; Peppler, Mazess, 1981). Different absorptiometers may use slightly different x-ray pulse patterns: constant vs. alternating (Pritchard et al., 1993). The radiation dose for a single whole-body scan has been established by the manufacturer as 0.5 mrem (Jensen, et al., 1993) which is equivalent to approximately one-tenth of the radiation dose delivered during a standard chest x-ray.

Based on software calibrations, the ratio of soft tissue attenuation (R_{ST}) at the two energy levels is used to partition soft tissue into fat and lean compartments (Aloia, et al., 1995). Fat free mass can be calculated in two ways: (1) as fat mass subtracted from body weight or (2) as fat mass subtracted from total soft tissue mass (Aloia, et al., 1995). Some researchers combine lean soft tissue mass with body bone mineral to calculate fat free mass (Jensen, et al., 1993; Aloia, et al., 1995).

Inherent differences exist between LBM and fat-free mass (FFM). LBM represents tissue that may contain deposits of adipose tissue, but these amounts are not quantifiable. FFM is tissue separate and distinct from adipose tissue. In this respect, DXA is able to distinguish between FFM and adipose tissue within an acceptable range (Pritchard, et al., 1992).

With the participant lying supine, wearing a hospital gown, and with all metal objects removed from their person (no jewellery, no bra), a series of transverse scans were made from head to toe. Scans were done with a standardized transverse scan speed of five cm/sec giving a total scan time of approximately three minutes.

Percent body fat was calculated by dividing fat mass (kg) by total body mass (kg). Total body mass calculated by the summation of lean body mass, fat mass, and bone mineral mass using DXA was also used for comparison to body weight (kg) measured using a medical balance beam scale.

Body composition was estimated for several defined regions outlined in

the Hologic software program (Version 8.10). These regions included: (1) left and right arm (delineated through the left and right shoulders), (2) left and right leg (defined as the tissue below the oblique lines passing through the hip joints), (3) trunk (delineated by an upper horizontal border below the chin), (4) vertical borders lateral to the ribs, (5) and a lower border formed by the oblique lines passing through the hip joints, and head (all tissue above the trunk and shoulders). The analysis provided the subject's mass in grams of bone mineral content (BMC), body fat, lean, lean plus BMC, sum of total body tissues, and percentage fat. In addition to whole body composition, Hologic software provided body surface area (cm²), bone mineral density (g/cm²), T-score, and Z-score as indicators of bone density relative to age and peak bone mass of a reference population (Hologic Corporation, 1995).

To quantify fat distribution, two additional regions were added to the DXA analysis (Appendix B). The central abdominal region (R1) was referenced from Carey, et al., (1996) who used DXA and computed tomography to assess fat distribution and to determine the association between insulin resistance and abdominal fat in 22 healthy women with varying degrees of glucose tolerance. R1 and R2 were determined after the participant's image was 'scanned' into the Hologic program. To exclude some subcutaneous fat, the lateral border of the abdominal regions were aligned with the outer edge of the rib cage, excluding ~30% of the abdominal subcutaneous fat. The superior border passed just above the L2 vertebrae and the inferior border passed just below the L4 vertebrae, so the L2-L4 region was encompassed. This was an area shown by MRI to contain a relatively high visceral and low subcutaneous fat content (Ross, et al., 1993).

The gluteal region (R2) was referenced from Ley, Lees, and Stevenson (1992) who used DXA to investigate sex- and menopause-associated changes in body fat distribution in 131 healthy women (61 premenopausal and 70 postmenopausal). The R2 region was positioned so that its superior border was at the highest points of the inner pelvis and its sides lateral to the lower body tissue. The inferior border passed through the mid-point of the femur. This

process was standardized and completed by the DXA operator for every participant.

4. Aerobic fitness and physical activity

The VO_{2max} test required a continuous 6- to 12-minute supermaximal effort that usually consisted of increments in effort (graded exercise) to the point where the subject was no longer able to continue exercising. Some researchers have termed this 'end-point' exhaustion. It should be kept in mind, however, that it was the participant who, for whatever reason, elected to stop the test. The decision to stop the fitness test was often influenced by a variety of psychological or motivational factors that might not necessarily reflect physiological strain. Practical experience has shown that high motivation and a relatively large anaerobic output were generally required to demonstrate a plateau in oxygen consumption during the max VO₂ test. A common complaint of subjects on both the continuous and discontinuous bicycle tests was a feeling of intense local discomfort in the thigh muscles during heavy work. Many subjects stated that this was the major factor limiting their ability to perform further work on the ergometer. Research has demonstrated that a subject's anxiety to maximal exercise testing was dependent on pre-exercise anxiety levels; however, this response was transitory and followed by positive mood shifts 10 to 15 minutes following such tests (O'Connor, et al., 1995).

Historically, the achievement of maximal oxygen uptake has been based on objective criteria such as a leveling off of oxygen uptake with an increase in work rate, high levels of lactic acid in the blood in the minutes following the exercise test, elevated respiratory exchange ratio, and achievement of some percentage of an age-adjusted estimate of maximal heart rate (Howley, Bassett, Welch, 1995). When the generally accepted criteria for the attainment of max VO₂ are not met, or the test performance appears limited by local factors rather than central circulatory dynamics, the term *peak* VO₂ is usually used. Peak VO₂ refers to the highest value of oxygen consumption measured per minute during the test (Geliebter, et al., 1997; McArdle, Katch, Katch, 1991).

Participants were required to complete a PAR-Q (Canadian Standardized Test of Fitness, 1986) prior to the fitness test to screen for possible medical risks or injuries that would prevent the participant from completing the fitness test. Those answering "no" to all seven questions on the PAR-Q were allowed to proceed with the fitness test. Participants answering "yes" to one or more of the questions were screened by the researcher and allowed to proceed with the test with extreme caution. All aerobic fitness testing was completed in the Exercise Physiology Lab (Physical Education building, University of Alberta, Edmonton, AB) by a trained exercise physiologist from the Faculty of Physical Education using standardized procedures.

Participants were prepared for the cycle ergometer test with detailed verbal instructions and were subsequently fitted with a nose-clip and mouthpiece. After a 5-minute warm-up at minimal workload (1.0 kiloponds), the cycle resistance was increased by 0.5 kiloponds every two minutes until the max VO₂ was attained or the subject stopped the test (peak VO₂). Heart rate (Polar Pacer) and flywheel revolutions were monitored and recorded every minute. Oxygen consumption data was recorded by computer every 30 seconds. Aerobic power was recorded as oxygen consumed per minute (L O₂/min) and oxygen consumed per minute per kg body weight (mL O₂/min/kg).

At the completion of the test, participants were encouraged to pedal at reduced resistance until heart rate returned to a normal (pre-exercise) level. The participants then dismounted the cycle and completed the cool down with the provision of adequate fluids.

Usual physical activity patterns were estimated using the modified Baecke Questionnaire (Appendix C) (Pols, et al., 1995). Participants were asked questions pertaining to work, sport, and leisure time activities. Responses were indicated on a Likert scale, ranging from never to always or very often. Sample questionnaire statements included "At work I sit" and "During leisure time I play sport".

A published scoring guide was used to score the questionnaire (Pols, et al., 1995). All questions were assigned a value between one and five depending

on the participant's response. To assign a value to question nine, ("Do you participate in team sports?"), each sport chosen was rated by determining its "intensity", "time", and "proportion". Intensity was estimated by converting the chosen sport to that sport's approximate energy costs (kcal/min/kg). Energy costs between 0.022 and 0.114 kcal/min/kg were given a score of 0.76, 0.115 and 0.205 kcal/min/kg were given a score of 1.26, and over 0.206 kcal/min/kg were given a score of 1.76. The scores of 0.76, 1.26, and 1.76 were then assigned a value of 1, 3, or 5 respectively. These values were multiplied by "time" and "proportion" to determine the total score for question nine. This value (question nine) was then used to calculate the total physical activity index. The total activity index was a sum of the three subscales (work, sport, leisure); scores closer to 15 indicated participants were very active, while scores closer to zero indicated participants were less active.

5. Biochemical indices

Several biochemical indices were measured including insulin, glucose, leptin, T₄, T₃, and r-T₃. Collection occurred during the course of a 2-hour oral glucose tolerance test (OGTT). All blood collection was conducted at a public collection site (Dynacare-Kasper Medical Laboratory, Edmonton, AB) by a trained technician. Participants were met at this location by the researcher who facilitated the blood collection.

Participants arrived at the collection site in the morning after an overnight fast (nothing to eat or drink except water for 12 hours) and were informed of the blood collection procedure. The baseline sample occurred with the drawing of one, 10-mL tube of whole blood. This tube contained blood for the sampling of glucose, insulin, leptin, and thyroid hormones. After this collection, participants were asked to eat 150-g of white bread (75-g carbohydrate, 12-g protein, 0.5-g fat) (Wolever, Bolognesi, 1996). The bread was cut into bite-sized pieces and the weight was standardized before consumption. At the start of consumption of the bread, time zero was indicated as stated in the recommended OGTT procedure manual (National Diabetes Data Group, 1979). Participants were encouraged to

eat the bread within 10-15 minutes.

At time 30-, 60-, and 90-minutes, a 5-mL sample of whole blood was drawn. All samples were centrifuged, separated, and aliquoted into small, labelled vials prior to transport to the University lab for storage. At the final timepoint, 120-minutes after the start of the test meal, a final 10-mL sample of whole blood was drawn. All blood draws were completed using separate needle insertions – an indwelling cannula was not used. The blood collection procedure was completed in approximately 2.5 hours. During the time between blood draws, participants were asked to complete four questionnaires.

Vials containing samples for glucose, leptin, and thyroid hormone analysis were transported on ice in a biohazard container in accordance with biohazard transport regulations. Upon arrival to the University lab, each 5-mL vial of serum was divided evenly into 1.5-mL Eppendorf tubes for storage at -20°C until further analysis was conducted. This protocol was approved through a biosafety registry in accordance with University biosafety regulations. The tubes containing serum samples for insulin were forwarded to the Capital Health Authority Laboratory, University Hospital lab site for further analysis. All tubes were labelled with participant code, participant initials, sample type, and date of collection. Serum was analyzed for insulin and glucose levels at each of the five time points, leptin was determined at fasting and 120-minutes, and thyroid hormones were determined at fasting only.

Glucose levels were determined with a quantitative, enzymatic diagnostic assay (Sigma Diagnostics). Glucose was oxidized to gluconic acid and hydrogen peroxide with a glucose oxidase catalyst. Hydrogen peroxide reacted with 4-aminoantipyrine and p-hydroxybenzene in the presence of peroxidase to form a quinoneimine dye. The intensity of the color was directly proportional to the glucose concentration in the sample.

The protocol for the above glucose assay was as follows. Seven standard glucose samples (0.005 mL per sample) of known concentration were pipetted in duplicate into a 96 well, non-coated, flat bottomed plate. A positive displacement pipette was used to place 5 μ L of unknown sample in the corner of each

subsequent well. After the entire plate was filled with both standard and samples, 200 µL of the Trinder reagent was added to every well using a multi-tip pipette. The Trinder reagent was added quickly to insure a uniform reaction time. The entire plate was placed on a flat mixer to insure adequate disbursement of sample and reagent. Incubation lasted 20 minutes at ambient temperature (approximately 20°C). Each plate was read by a spectrophotometer to determine absorbance. Absorbance values were converted to glucose concentrations (mmol/L) using a predetermined formula.

Insulin levels were determined using a double antibody enzyme linked immuno-sorbent assay (ELISA) (Boehringer Mannheim Immunodiagnostics, Amsterdam, UK). The kit was designed to measure human insulin in serum or plasma treated with EDTA, fluoride, or heparin. The ELISA method involved adding the sample (0.1 mL), which contained an unknown amount of insulin, to plastic coated tubes coated with mono-clonal anti insulin antibodies. All samples were completed in duplicate. An incubation solution (1.0 mL), which contained the anti-insulin POD conjugate, was also added to the tubes. Tubes were incubated for 120 ± 10 minutes at 20-25 °C. Immediately following the incubation, excess enzyme linked antibody was separated from the tubes with a washing solution (Enzymun-Test® washing solution). The washing solution was aspirated within 3-15 minutes. The following step involved the addition of 1.0 mL substrate-chromogen solution to tubes containing standard solutions, controls, and samples. Tubes were incubated for 60 ± 10 minutes and then transferred to cuvettes for absorbance determinations. A standard curve was developed on which the insulin concentrations could be plotted. Insulin concentration was reported as μU/mL, which was converted to nmol/L by multiplying by a factor of 0.007.

Leptin concentrations were determined using a human radioimmunoassay kit (Linco Research, St. Charles, MO) designed to measure human leptin in plasma or serum. The double antibody method involved incubating the sample (contains an unknown amount of leptin) or the standard (contains a known

amount of leptin) with I¹²⁵ tagged leptin and an antibody that is highly specific to the hormone being measured (AB1). After an unspecific antibody (AB2) was added (which binded to the AB1/label/cold leptin complex to form a pellet), the free label was aspirated to waste. The pellet was counted in a gamma counter to determine levels of radioactivity then converted to leptin concentration (ng/mL).

The protocol for the above leptin assay was as follows. Assay buffer was pipetted into non-specific binding tubes, reference tubes, and sample tubes. Standards and quality controls were pipetted into the first ten sample tubes. The unknown samples were pipetted into the remaining sample tubes. Each sample tube was duplicated. I¹²⁵-Leptin was added to all tubes. Leptin antibody was added to every tube except the two totals and the two non-specific binding tubes. All tubes were vortexed, covered, and incubated overnight (18-24 hours) at 4°C. After incubation, precipitating reagent was added to all tubes except totals. All tubes were vortexed and incubated for 20 minutes at 4°C. Sample tubes were centrifuged for 30 minutes at 3000 rpm, decanted, and drained. Each tube was counted in a gamma counter to measure levels of 'hot' antibody associated with the leptin complex. The counts were converted to ng/mL by a predetermined formula.

Thyroid hormone (T_4, T_3) levels were determined by a solid-phase radioimmunoassay (Diagnostic Products Corporation, Los Angeles, CA – T_4 and T_3 and Biodata Diagnostics, Rome, Italy – r- T_3) as described for leptin. The protocol for the thyroid hormone assay is as follows. Uncoated tubes were used for total counts and non-specific binding. Standards, quality controls, and samples were added to coated tubes. I^{125} -labelled thyroid hormone was added to every tube. Tubes were vortexed and incubated in a heated water bath (T_4 and T_3 only; r- T_3 incubated at room temperature). Tubes were decanted thoroughly (except totals) until all visible moisture was removed. All tubes were counted in a gamma counter. The counts were then converted to thyroid concentration (nmol/L). Quality controls were provided by CON6 Multivalent Control Module (Diagnostic Products Corporation, Los Angeles, CA) an assayed, human serum-

based tri-level control containing over 25 constituents commonly measured by immunoassay.

Normative fasting values were provided for all biochemical indices (Appendix D).

6. Dietary intake

Energy intake was obtained using a FFQ (Appendix E). This information was typically collected during the blood collection period. All questionnaires were reviewed with each participant to clarify answers and to obtain missing information, where necessary. FFQ were numerically coded and independently analyzed for dietary intake, excluding any vitamin or mineral supplements or herbal preparations. Analysis of the dietary intake was completed by a summer student who was blinded to the identity of each participant. Permission for use of the FFQ was granted by Dr. Bright-See of Brescia College, London, ON.

The FFQs were analyzed using a computerized nutrition software program (The Food Processor®, Version 6.0, ESHA Research, Salem, OR). This software used a database of over 10,000 foods from Canadian and USDA databases and includes brand name items, fast foods, and most common items.

Dietary intake was quantified by creating a master food list that provided a template for each participant. The master food list was based directly on categories from the FFQ. Food codes were selected from the data bank and weight values were assigned to each of the three allowable servings (small, medium, large). Individual participant responses were based on frequency of consumption over the previous six months. These responses were subsequently calculated into daily amounts. A serving size key was used to assist in coding the FFQ. The food lists were analyzed for energy intake (kcal/day), carbohydrate, fat, protein, total fibre (g/day), soluble fibre (g/day), daily servings of fruit, vegetables, and breads/cereals, P:S ratio, omega-3 fatty acids (g/day), and omega-6 fatty acids (g/day).

7. Dietary restraint

The ten-item restrained eating subscale (DEBQ) was used to determine deliberate weight control (Appendix F). All items were presented in a Likert scale form with the following categories: never, seldom, sometimes, often, very often, and "not relevant". The "not relevant" category was added to items with a conditional format because some participants might never eat too much or never become heavier. For example: "When you have put on weight do you eat less than you usually do?" and "When you have eaten too much, do you eat less than usual the following day?" Participants were asked to complete this questionnaire during the blood collection period. All ten questions were assigned a mark between one and five depending on the participant's response. Final scores were obtained by totaling the questionnaire items and dividing by ten. Final scores closer to five indicated high restraint, while final scores closer to one indicated low restraint. If "not relevant" was chosen (question #1 and/or 6), the question was omitted and this response was not included in the final score.

8. Body image

The Multidimensional Body-Self Relations Questionnaire (MBSRQ) was used to assess body image (Appendix G). Permission for use was granted by Dr. Thomas Cash of Old Dominion University, Norfolk, VA (original author). Scoring for the ten subscales of the MBSRQ was also provided by Dr. Cash (1994) in the form of a manual that included the population norms and reliabilities for males and females. Scoring formulas were also provided by the original author (Appendix H). Norms for the MBSRQ were derived from US national survey data (Cash, et al., 1985) based on 1070 female responses.

The following interpretive descriptions of each of the MBSRQ subscales are included in this manuscript as indicated in the scoring manual: Appearance evaluation measured feelings of physical attractiveness or unattractiveness, satisfaction or dissatisfaction with one's looks. High scorers felt mostly positive and satisfied with their appearance. Appearance orientation measured the extent of investment in one's appearance. High scorers placed importance on how they

look, paid attention to their appearance, and engaged in extensive "grooming behaviours" to manage their appearance. Low scorers were apathetic about their physical appearance and didn't expend much energy or time to "look good". Fitness evaluation described feelings of being physically fit or unfit. High scorers regarded themselves as physically fit, "in shape", or athletically active and competent. Low scorers felt physically unfit, "out of shape", or athletically unskilled. Fitness orientation assessed extent of investment in being physically fit or athletically competent. High scorers valued fitness and were actively involved in activities to enhance or maintain their fitness. Low scorers did not value physical fitness and did not regularly incorporate exercise activities into their lifestyle. Health evaluation described feelings of physical health and/or the freedom from physical illness. High scorers felt their bodies were in good health. Low scorers felt unhealthy or experienced bodily symptoms of illness or vulnerability to illness. Health orientation characterized the extent of investment in a physically healthy lifestyle. High scorers were "health conscious" and tried to lead a healthy lifestyle. Low scorers were apathetic about their health. Illness orientation assessed the extent of reactivity to being or becoming ill. High scorers were alert to personal symptoms of physical illness and were apt to seek medical attention. Low scorers were not especially alert to reactive to symptoms of illness. Body areas satisfaction scale (similar to appearance evaluation) measured satisfaction or dissatisfaction with discrete aspects of one's appearance. High scorers were generally content with most areas of their body. Low scorers were unhappy with the size or appearance of several areas of their body. Self-classified weight reflected how one perceived and labelled one's weight (very underweight to very overweight). Overweight preoccupation assessed a construct of fat anxiety, weight vigilance, dieting, and eating restraint. High scorers were extremely anxious about body fat and weight and particularly restrained with respect to dietary intake.

At the completion of the data collection and analysis, all participants received a comprehensive summary of individual and group results (Appendix I). Participants were encouraged to contact the researcher for clarification of results

either by telephone or in person. At the completion of the project, all participants received an invitation to attend an evening presentation to discuss the results of the study.

9. Ethical approval

The present study received ethical approval from The University of Alberta, Faculty of Agriculture, Forestry, and Home Economics Human Ethics Review Committee. The amended Certificate of Approval from the University of Alberta is provided in Appendix J. Participants provided informed consent before participation in the study (Appendix K).

10. Statistical analysis of the data

Statistical analyses were calculated using the software program, Statistical Package for the Social Sciences (SPSS, Version 7.5). All data were entered into SPSS files from a standardized data form at the time of data collection. Prior to analysis, all entries were verified by comparing the recorded data in each file to a printed copy of the data files entered into SPSS.

All results were presented as mean ± standard deviation. All significant p-values (<0.05) were indicated in bold type. Frequencies and Q-Q plots were used to test the data for normality around the mean (data not shown). To test the hypotheses, independent t-tests were used to compare REE, body composition, physical fitness, biochemical variables, dietary intake, dietary restraint, and body image data of each group (high vs. low REE). Repeated measures analysis of variance (ANOVA) were also conducted on data with more than one time point (glucose, insulin, and leptin). Correlations were completed to test for associations between specific variables and included as raw data in the appendices (Appendix L). All statistical tests were performed to detect differences at p<0.05 level of significance, a priori (using two-tailed values).

CHAPTER FOUR RESULTS

1. Participant recruitment

Potential participants were screened from a pre-existing subject pool. All participants were either told of the study in person or contacted by telephone. During 16 weeks of recruitment, 74 participants were individually screened for eligibility based on age and initial REE. Forty-two participants were excluded for the following reasons: failing to meet second REE criteria (n=15), taking medications (n=4), unable to commit to project (n=17), smoking (n=5), and recent hypothyroid diagnosis (n=1). Once the research project commenced, two participants decided not to continue due to conflicting commitments. Thirty women completed the entire research project (15 per group).

2. Participant characteristics

Table 3 represents a summary of the demographic, anthropometric, and energy expenditure data of both groups.

Table 3: Characteristics of female chronic dieters

VARIABLE	HIGH REE	LOW REE	p-value
Age (years)	39.3 ± 5.5^{a}	39.5 ± 7.9	0.936
Height (cm)	162.0 ± 5.2	162.3 ± 6.0	0.900
Weight (kg)	84.9 ± 17.3	82.9 ± 26.5	0.800
BMI (kg/m²)	32.0 ± 5.8	31.1 ± 8.9	0.750
Measured REE (kcal/day)	1610 ± 195	1228 ± 264	0.0001
Predicted REE ^b (kcal/day)	1507 ± 205	1488 ± 316	0.845
% Predicted ^c	107.1 ± 5.9	82.5 ± 2.8	<0.00001
REE ^d /kg body weight (kcal/day/kg)	19.3 ± 2.0	15.2 ± 1.7	<0.00001
REE/kg LBM (kcal/day/kg LBM)	35.9 ± 2.3	30.7 ± 2.8	<0.00001
RQ (VCO ₂ /VO ₂)	0.79 ± 0.04	0.81 ± 0.04	0.103

^a Mean ± standard deviation

^b Using Mifflin equation (Mifflin, et al., 1990)

^{°%} predicted = measured REE/predicted REE

^d Using measured REE

Age, height, weight, BMI, predicted REE, and RQ did not differ between the two groups of female chronic dieters. Women in the high REE group had significantly higher measured REE, % predicted REE, REE/kg body weight, and REE/kg LBM. BMI values ranged from 22.1 to 49.8 kg/m²; 20 participants had a BMI of 27.3 kg/m² or more and were considered obese. The average BMI for all participants was 31.6 ± 7.4 kg/m².

There was no statistical difference between groups with respect to oral contraceptive use. The high REE group had 3 participants taking oral contraceptives and the low REE group had 1 participant using oral contraceptives and 3 using hormone replacement therapy. When controlling for pill use, no differences in REE were observed.

When individual groups were compared with respect to their measured and predicted REE, the low group had significantly lower REE (p<0.0001) and the high group had significantly higher REE (p=0.0003) than predicted by the Mifflin St. Jeor equation (Mifflin, et al., 1990).

Participant weight changes were determined from the first REE to the second REE; no significant differences between groups were noted. The weight changes for each group were as follows: high REE, -0.69 ± 3.64 kg; low REE, 0.71 ± 3.08 kg (p=0.264).

3. Body composition

Table 4 represents body composition characteristics of both groups.

Table 4: Body composition characteristics of female chronic dieters

VARIABLE	HIGH REE	LOW REE	p-value
LBM (kg)	44.8 ± 4.8^{a}	39.8 ± 5.9	0.017
Fat mass (kg)	34.7 ± 12.2	37.8 ± 18.4	0.600
Bone mass (kg)	2.2 ± 0.3	2.1 ± 0.3	0.404
% LBM	55.9 ± 6.9	51.9 ± 8.7	0.180
% body fat	41.4 ± 7.3	45.3 ± 9.2	0.204
Bone density (g/cm²)	1.12 ± 0.09	1.09 ± 0.09	0.356

a Mean ± standard deviation

There were no differences between high and low REE groups of female chronic dieters with respect to fat mass, bone mass, % LBM, % body fat, and bone density. However, the high REE group had greater LBM.

In addition to the above measures of body composition, two regions of the body were compared between participants (Table 5). These regions were analyzed in terms of fat content and ratio of regional fat to total body fat. These data were compared to waist-to-hip ratio (WHR), an additional measure of fat distribution. This comparison demonstrated that individuals with high REE had higher ratios of abdominal:gluteal fat and waist:hip circumference.

Table 5: Fat distribution by pre-determined regions

VARIABLE	HIGH REE	LOW REE	p-value
R1ª Fat mass (kg)	3.0 ± 1.3 ^b	2.8 ± 1.4	0.722
R1/total body fat	0.09 ± 0.02	0.07 ± 0.01	0.064
R2° Fat mass (kg)	8.3 ± 2.9	9.8 ± 4.6	0.286
R2/total body fat	0.24 ± 0.03	0.26 ± 0.62	0.052
R1Fat mass/R2 Fat mass	0.37 ± 0.10	0.29 ± 0.07	0.019
WHR ^d	0.81 ± 0.04 ^e	0.76 ± 0.06	0.021

^a R1 = region 1 (abdominal region) as determined from: Carey, et al., 1996.

Groups did not differ with respect to abdominal or gluteal fat mass when expressed in absolute or relative (to total body fat) terms. However, groups did differ when abdominal and gluteal fat mass was expressed as a ratio and when fat distribution was compared using WHR.

^b Mean ± standard deviation

^c R2 = region 2 (gluteal region) as determined from: Ley, et al., 1992.

^d WHR = Waist to hip ratio determined from circumference measurements

^e Missing data from 3 participants (n=12 for high REE group)

4. Aerobic fitness and physical activity

Table 6 represents a summary of aerobic fitness and physical activity characteristics.

Table 6: Aerobic fitness and physical activity of participants

VARIABLE	HIGH REE	LOW REE®	p-value
Absolute VO _{2max} (L O ₂ /min)	2.34 ± 0.43 ^b	2.11 ± 0.46	0.181
Relative VO _{2max} (mL O ₂ /min/kg body wt)	28.13 ± 7.13	26.66 ± 5.42	0.540
Relative VO _{2max} (mL O ₂ /min/kg LBM)	52.39 ± 8.70	53.70 ± 6.53	0.653
Avg Relative VO _{2max} (mL O ₂ /min/kg body wt)	32	.0°	n/a
Physical activity score ^d	7.27 ± 1.22	7.02 ± 1.26	0.579
Work index ^e	2.52 ± 0.70	2.69 ± 1.00	0.602
Sports index ^e	2.13 ± 0.92	1.61 ± 0.63	0.081
Leisure time index ^e	2.62 ± 0.50	2.72 ± 0.42	0.557

a Missing one case (n=14 for low REE group)

The two groups did not differ with respect to absolute VO_{2max} , relative VO_{2max}/kg body weight, relative VO_{2max}/LBM , or physical activity score. Both groups were less physically fit than the average Canadian woman, with the low REE group significantly less fit (p<0.01). On average, the physical activity score of chronic dieters was 7.14 ± 1.22 (out of a possible score of 15). The range of physical activity scores was 4.32 to 9.29. Although physical activity scores did not differ between high and low REE groups, there was a trend for high REE participants to engage in more sports activities (p=0.081).

Most of the participants (n=24/29; 82.8%) did not attain the specified criteria for VO_{2max} . Of those who reached a plateau in O_2 consumption during the test, three were from the high REE group and two were from the low REE group. One individual from the low REE group did not complete the aerobic fitness test.

^b Mean ± standard deviation

^c Average value for women aged 30-39 years (Canadian Standardized Tests of Fitness, 1986)

^d From Baecke questionnaire, scored on a 15-point Likert scale (score of 15 indicates high physical activity)
^e From Baecke questionnaire: three activity indices which add together to comprise the total physical activity score, scored on a 5-point Likert scale (score of 5 for each index indicates high physical activity)

Participants were divided into two groups based on aerobic fitness level and compared in terms of relevant metabolic parameters (Table 7). The "high fit" or "low fit" designation depended on absolute fitness scores relative to the group fitness median of 2.20 L O₂/min.

Table 7: Comparisons between high fit and low fit female chronic dieters

VARIABLE	HIGH FIT ^{a,b}	LOW FIT	p-value
Weight (kg)	92.4 ± 24.6°	73.8 ± 13.3	0.017
REE (kcal/day)	1592.3 ± 250.1	1248.0 ± 255.2	0.001
REE/kg body wt (kcal/day/kg)	17.8 ± 2.9	17.0 ± 2.5	0.441
REE/kg LBM (kcal/day/kg)	34.9 ± 2.4	31.9 ± 4.3	0.031
Body fat (kg)	40.8 ± 18.3	31.0 ± 10.6	0.085
LBM (kg)	45.5 ± 5.17	39.0 ± 4.7	0.001
R1 fat mass (kg)	3.1 ± 1.3	2.6 ± 1.2	0.327
R1 LBM (kg)	5.4 ± 0.7	4.7 ± 0.9	0.030
R2 fat mass (kg)	9.9 ± 4.5	7.7 ± 2.4	0.116
R2 LBM (kg)	11.4 ± 2.3	9.0 ± 1.0	0.002
Rel. VO _{2max} (mL O ₂ /min/kg LBM)	57.5 ± 6.5	48.8 ± 6.2	0.001
Physical Activity Scored	7.40 ± 1.47	6.95 ± 0.98	0.341
Glucose AUC (mmol·min/L)	140.5 ± 57.2	199.1 ± 118.6	0.102
Insulin AUC (nmol·min/L)	23.3 ± 12.5	32.0 ± 21.3	0.191
Fasting Leptin (ng/mL)	29.7 ± 13.5	23.4 ± 11.5	0.183
T ₄ (nmol/L)	114.8 ± 29.1	96.0 ± 14.7	0.042
T ₃ (nmol/L)	2.3 ± 0.4	2.1 ± 0.6	0.289
r-T ₃ (nmol/L)	0.22 ± 0.05	0.19 ± 0.03	0.054
Energy Intake (kcal/day)	2538.9 ± 774.4	1834.2 ± 385.0	0.006
Dietary Restraint Score	2.86 ± 0.49	3.45 ± 0.92	0.044

^a Participants were divided into two fitness groups by the median absolute fitness score of 2.20 L O₂/min

The high fit group had significantly higher values than the low fit group for body weight, REE/day, REE/kg LBM, LBM, R1 LBM, R2 LBM, fitness/LBM, T₄, and energy intake. Furthermore, the high fit group had a significantly lower

^b Missing one value (n=14 for high fit group, n=15 for low fit group)

^c Mean ± standard deviation

^d From Baecke questionnaire, scored on a 15-point Likert scale (score of 15 indicates high activity)

dietary restraint score than the low fit group.

5. Biochemical indices

Glucose and insulin

There were no differences between the groups in terms of the average time required to consume the OGTT meal (high REE group, 16.7 ± 6.3 minutes; low REE group, 20.5 ± 6.6 minutes) (p=0.124). Participants did not report difficulty with consuming the test meal.

The summary of glucose and insulin values during the OGTT for both groups is presented in Table 8. Time points during the OGTT include fasting (10 minutes before ingestion of test meal), 30-, 60-, 90-, and 120-minutes (after start of test meal). Participants were asked to provide these samples without an indwelling cannula as the risk of infection was greater with this procedure. A summary of total glucose and insulin area under the curve (AUC) is included as determined by the incremental model (Wolever, Jenkins, 1986).

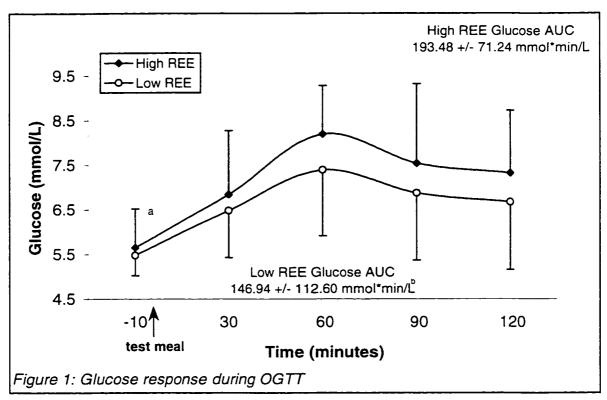
Table 8: Glucose and insulin values during OGTT

VARIABLE	HIGH REE	LOW REE	p-value
Fasting glucose (mmol/L)	5.7 ± 0.9 ^a	5.5 ± 0.5	0.494
Fasting insulin (nmol/L)	0.1 ± 0.1	0.1 ± 0.1	0.061
30-Min glucose (mmol/L)	6.8 ± 1.4	6.5 ± 1.1	0.437
30-Min insulin (nmol/L)	0.3 ± 0.1	0.2 ± .0.1	0.040
60-Min glucose (mmol/L)	8.2 ± 1.1	7.4 ± 1.5	0.103
60-Min insulin (nmol/L)	0.5 ± 0.3	0.3 ± 0.1	0.013
90-Min glucose (mmol/L)	7.5 ± 1.8	6.9 ± 1.5	0.267
90-Min insulin (nmol/L)	0.6 ± 0.3	0.3 ± 0.1	0.009
120-Min glucose (mmol/L)	7.3 ± 1.4	6.7 ± 1.5	0.236
120-Min insulin (nmol/L)	0.5 ± 0.3	0.3 ± 0.1	0.011
Glucose AUC (mmol·min/L)	193.5 ± 71.2	146.9 ± 112.6	0.187
Insulin AUC (nmol·min/L)	36.6 ± 20.2	18.8 ± 7.9	0.005

^a Mean ± standard deviation

There were no significant differences between the two groups in terms of fasting glucose, fasting insulin, 30-min glucose, 60-min glucose, 90-min glucose,

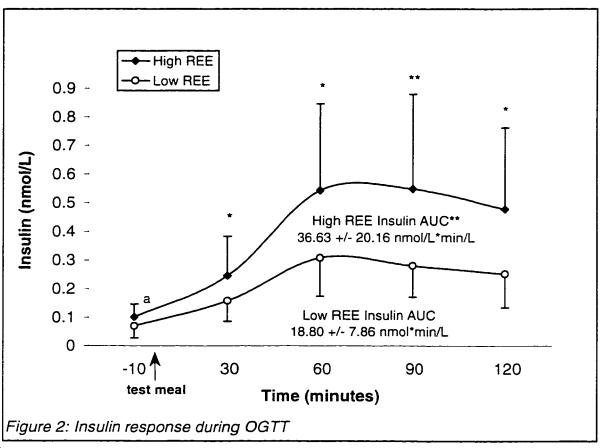
120-min glucose, and glucose AUC. The groups differed significantly in terms of 30-min insulin, 60-min insulin, 90-min insulin, 120-min insulin, and insulin AUC. The high REE group had higher levels of 30-min, 60-min, and 90-min serum insulin as well as higher insulin AUC. Refer to Figures 1 and 2 for group curves.



^a Mean ± standard deviation

Two individuals from the high REE group met the criteria for impaired glucose tolerance (IGT) as determined from the National Diabetes Data Group (1979) classification guidelines. Removal of these data did not alter the significance of these results, so these data were included for both Figure 1 and 2. Glucose results were confirmed by a non-significant group by time interaction using a repeated measures ANOVA (p=0.950).

No significant difference between groups



^a Mean ± standard deviation

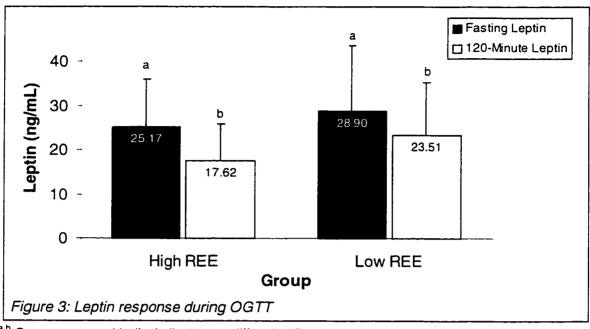
When insulin AUC was compared to previous data from healthy, normal weight participants who completed a similar OGTT (using 75-g carbohydrate from 150-g white bread) (Wolever, Bolognesi, 1996), the high REE group was not statistically different from the comparison's group value of 31.0 ± 14.3 (p=0.298), but the low REE group was significantly lower (p<0.0001). Therefore, the insulin AUC of the high REE group was significantly higher than the low REE group, but similar to previous results in normal weight individuals. These results were also analyzed by repeated measures ANOVA. The group by time interaction was not significant (p=0.188).

^{*} p<0.05

^{**} p<0.01

Leptin

The summary of serum leptin values at fasting and 120 minutes post 75-g carbohydrate test meal for all participants in both groups is presented in Figure 3.



a.b Group means with dissimilar letters differ significantly (p<0.001)

There were no differences between the two groups in terms of fasting and 120-min leptin values (p=0.437 and p=0.124, respectively). However, there were differences *within* the two groups in terms of leptin response. In all cases and in both groups, leptin decreased significantly from fasting to 120-min levels (p<0.001). These results were confirmed with a non-significant group by time interaction using a repeated measures ANOVA (p=0.708).

Thyroid

The summary of fasting serum T_4 , T_3 , and $r-T_3$ is presented in Table 9.

Table 9: Thyroid hormones at fasting

VARIABLE	HIGH REE	LOW REE	p-value
T ₄ (nmol/L)	115.3 ± 27.5 ^a	93.9 ± 14.2	0.014
T ₃ (nmol/L)	2.3 ± 0.5	2.2 ± 0.6	0.491
r-T ₃ (nmol/L)	0.22 ± 0.05	0.18 ± 0.01	0.019

^a Mean ± standard deviation

High and low REE groups did not differ significantly in terms of T_3 , but differences existed between the two groups in terms of T_4 and r- T_3 . The high REE group had significantly higher levels of fasting serum T_4 and r- T_3 .

6. Dietary intake

Table 10 summarizes the dietary intake of both groups. Dietary intake was determined using Food Processor®, ESHA Research, Salem, OR.

Table 10: Dietary intake of female chronic dieters

VARIABLE	HIGH REE	LOW REE	p-value
Energy (kcal/day)	2313.4 ± 759.4^{a}	2026.3 ± 585.0	0.256
Fat (g/day)	96.4 ± 40.7	73.6 ± 34.2	0.109
% Fat	37.2 ± 7.7	32.0 ± 8.7	0.098
Carbohydrate (g/day)	269.0 ± 89.0	258.1 ± 71.3	0.714
% Carbohydrate	46.8 ± 7.4	51.8 ± 9.1	0.108
Protein (g/day)	103.2 ± 37.3	92.2 ± 39.0	0.438
% Protein	17.9 ± 3.2	18.0 ± 4.1	0.970

^a Mean ± standard deviation

There were no differences between groups in terms of dietary intake.

Other dietary factors were compared between the two groups (Table 11). These factors included daily servings of fruits, vegetables, and breads/cereals, polyunsaturated:saturated fat ratio (P:S ratio), omega-3 fatty acids, omega-6 fatty acids, total fibre, and soluble fibre.

Table 11: Other dietary factors of chronic dieters

VARIABLE	HIGH REE	LOW REE	p-value
Servings of Fruits/day	3.8 ± 2.3^{a}	3.6 ± 2.3	0.828
Servings of Vegetables/day	2.3 ± 1.8	1.3 ± 1.0	0.086
Servings of Breads & Cereals/day	8.9 ± 3.2	8.0 ± 3.4	0.470
P:S Ratio	0.5 ± 0.	0.5 ± 0.2	0.585
Omega-3 Fatty Acids (g/day)	1.8 ±0.9	1.2 ± 0.5	0.075
Omega-6 Fatty Acids (g/day)	12.0 ± 4.5	10.4 ± 5.2	0.382
Fibre (g/day)	26.0 ± 12.1	25.1 ± 6.8	0.804
Soluble Fibre (g/day)	7.1 ± 3.5	6.6 ± 2.1	0.673

^a Mean ± standard deviation

As indicated in Table 11, no differences were observed when the dietary intakes of both groups were analyzed in this manner.

7. Dietary restraint

Differences were observed between the two groups in terms of dietary restraint. The high REE group (2.81 \pm 0.52) had lower restraint scores (**p=0.011**) than the low REE group (3.52 \pm 0.84).

8. Body image

The body image scores of female chronic dieters are presented in Table 12. Body image was evaluated using the 10 subscales of the MBSRQ. These subscales included appearance, fitness, and health (evaluation and orientation), illness orientation, body areas satisfaction, overweight preoccupation, and self-classified weight. These subscales were scored on a 5-point Likert scale. High evaluation and orientation scores reflected positive body image attributes. High Body Areas Satisfaction Scores reflected positive body satisfaction. High

overweight preoccupation and self-classified weight reflected negative body image attributes.

Table 12: MBSRQ body image subscales of female chronic dieters

VARIABLE ^a	HIGH REE	LOW REE	p-value
Appearance Evaluation	2.30 ± 0.88 ^b	2.63 ± 1.03	0.362
Appearance Orientation	3.74 ± 0.47	3.79 ± 0.44	0.740
Fitness Evaluation	3.49 ± 0.96	3.15 ± 0.77	0.289
Fitness Orientation	3.25 ± 1.07	3.34 ± 0.64	0.782
Health Evaluation	3.53 ± 0.60	3.68 ± 0.86	0.601
Health Orientation	3.60 ± 0.59	3.81 ± 0.68	0.377
Illness Orientation	3.12 ± 0.70	2.91 ± 0.82	0.450
Body Areas Satisfaction Scale	2.60 ± 0.62	2.85 ± 0.62	0.280
Overweight Preoccupation	3.15 ± 0.63	3.00 ± 0.68	0.535
Self-Classified Weight	4.27 ± 0.62	4.30 ± 0.68	0.889

^a All measures were scored on a 5-point Likert scale whereby a score of 5 denotes positive body image evaluations and orientations (appearance, fitness, health, illness), positive body satisfaction (BASS), high overweight preoccupation, and high self-classified weight (both of which are negative body image attributes).

There were no significant differences between groups with respect to all 10 body image subscales.

^b Mean ± standard deviation

Body image scores of female chronic dieters were compared to gendermatched norms (Table 13).

Table 13: Body image scores of chronic dieters compared to reference norms

3.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2			
VARIABLE ⁸	REF	HIGH & LOW REE	p-value
Appearance	3.36	2.47 ± 0.96^{c}	<0.0001
Evaluation			
Appearance	3.91	3.77 ± 0.45	0.090
Orientation	<u></u>		
Fitness	3.48	3.32 ± 0.87	0.316
Evaluation			
Fitness	3.20	3.30 ± 0.87	0.550
Orientation			
Health	3.86	3.60 ± 0.73	0.068
Evaluation			
Health	3.75	3.70 ± 0.63	0.695
Orientation			
Iliness	3.21	3.01 ± 0.76	0.166
Orientation			
Body Areas	3.23	2.73 ± 0.62	0.0001
Satisfaction Scale			
Overweight	3.03	3.08 ± 0.65	0.706
Preoccupation			
Self-Classified	3.57	4.28 ± 0.64	<0.0001
Weight			

^a All measures were scored on a 5-point Likert scale whereby a score of 5 denotes positive body image evaluations and orientations (appearance, fitness, health, illness), positive body satisfaction (BASS), high overweight preoccupation, and high self-classified weight (both of which are negative body image attributes).

Chronic dieters (both high and low REE) had significantly lower appearance evaluation, lower body satisfaction, and higher self-classified weight than the reference population. No other differences between body image reference data and high or low REE groups existed.

^b REF = Reference norms for MBSRQ subscales (Cash, 1994; received from author, used with permission)

^c Mean ± standard deviation

9. Summary of results with reference to the study hypotheses

<u>Hypothesis 1:</u> Female chronic dieters with a high REE will have more favourable values for metabolic variables including higher LBM, higher aerobic fitness, higher physical activity, normal biochemical indices (glucose, insulin, leptin, thyroid), and higher dietary intake (energy, fat, carbohydrate, protein) compared to female chronic dieters with a low REE.

Within this hypothesis, several variables achieved significance. A portion of this hypothesis was accepted as the high REE group had higher LBM (p=0.017), insulin AUC (p=0.005), T4 (p=0.014), and r-T3 (p=0.019) than the low REE group. The remainder of this hypothesis was rejected since there were no group differences with respect to aerobic fitness, physical activity, fasting glucose, glucose AUC, fasting insulin, fasting leptin, 120-minute leptin, T₃, and dietary intake. There were significant intragroup differences as leptin decreased between baseline and 2-hours (p<0.001) during the OGTT.

<u>Hypothesis 2:</u> Female chronic dieters with a high REE will have lower levels of android fat distribution and dietary restraint compared to female chronic dieters with a low REE.

One half of this hypothesis was rejected as the high REE group had higher levels of abdominal:gluteal fat distribution and WHR (p=0.019 and p=0.021, respectively). The other half of this hypothesis was accepted as the high REE group had lower dietary restraint scores (p=0.011).

<u>Hypothesis 3:</u> Female chronic dieters will have lower body image scores compared to age- and gender-matched reference norms.

This hypothesis was accepted for three specific subscales of the MBSRQ. Chronic dieters had lower appearance evaluation scores (p<0.05), lower BASS (p<0.05), and higher self-classified weight (p=0.001). There were no other body image differences between the research groups and the reference population.

CHAPTER FIVE DISCUSSION

A. MAJOR FINDINGS

A number of distinctive conclusions have arisen from this study. The most meaningful finding was that female chronic dieters with a high REE had higher LBM compared to the low REE group. The second major finding was that the high REE group was insulin resistant in relation to the low REE group. Insulin resistance was characterized by higher insulin values over all OGTT time points (30-, 60-, 90-, and 120-minutes) except fasting. High REE insulin AUC values were almost twice those of the low REE group. The third major finding was that the high REE group had a higher ratio of abdominal:gluteal body fat than the low REE group. The fourth major finding was that the high REE group had higher fasting serum levels of T₄ and r-T₃ than the low REE group. The final major finding was that the high REE group demonstrated lower levels of dietary restraint than the low REE group. These results suggest that metabolic differences exist between these two groups of female chronic dieters.

B. OTHER FINDINGS

Other significant results were observed from this research. Female chronic dieters with a high REE had similar aerobic fitness and physical activity scores as dieters with a low REE. However, the two groups did differ in terms of aerobic fitness when compared to average Canadian fitness levels for women aged 30-39 years, the low REE group being significantly lower (Canadian Standardized Test of Fitness, 1986). Mean fasting and 120-minute serum leptin values were not different between groups, but within groups, leptin decreased significantly during this time period. All participants exhibited a decrease in leptin from fasting to 120-minutes regardless of whether they had high or low REE. Although several variables were significantly different between the groups, some variables were similar. For example, there were no group differences in terms of general

dietary intake variables (total energy, fat, protein, or carbohydrate) or other nutrients (fibre, servings of fruits, vegetables, breads, P:S ratio, omega-3, and omega-6 fatty acids). As well, there were no differences between groups in terms circulating T₃ and body image. However, chronic dieters scored significantly different on several body image subscales in relation to reference norms. Chronic dieters were more concerned about their appearance, more dissatisfied with their bodies, and rated their weight higher. These findings, together with the other major findings, establish that these chronic dieters possess distinct physiological and psychological characteristics.

1. Participant recruitment

The number of women completing the study was the same as specified in the research protocol (sample size calculation, Appendix A). Only two participants dropped out of the study once it had commenced. Sample size was based on the number of participants necessary to see differences between groups in terms of LBM. Losing two participants did not alter the significance of the results; LBM was still different between groups. Considering the invasiveness of the research protocol (especially the fitness testing and OGTT), maintaining a sample size of 30 participants demonstrated their commitment to the study. One participant refused the fitness test as a condition of her consent to the study. This was allowed considering aerobic fitness was only one component of the research protocol and the participant was in the low REE group. Recruitment for the low REE group was more time consuming than for the high REE group. This confirms what is already known about the number of chronic dieters with low REE. In a recent study, only 17.4% of female chronic dieters measured had a lowered REE (McCargar, McBurney, 1996). As this was the first such experiment of this design, participant recruitment was considered adequate to demonstrate the hypothesized differences in LBM.

2. Participant characteristics

The only characteristic that differed significantly between groups was measured (actual) REE. This was an important strength of this study. Minimizing

differences between groups was essential to reduce confounding variables. As such, this sample was a non-randomized convenience sample.

Although oral contraceptive use was not different between groups, progestin has been shown to have contrainsulin effects and is responsible for the deterioration in carbohydrate metabolism seen in genetically susceptible women during prolonged exercise and at rest (Bale, Davies, 1983; Bale, Nelson, 1985). These studies represent only a small number of women and oral contraceptive pills, so further inquiry is necessary to determine the effects of pill use among chronic dieters.

Participants were considerably heavier than what is determined desirable from BMI standards (healthy BMI range is between 20 and 27.3 kg/m²) (National Institutes of Health Consensus Development Panel, 1995). The average BMI for all participants was 31.6 ± 7.4 kg/m² (high REE, 32.0 ± 5.8 kg/m²; low REE, 31.1 ± 8.9 kg/m²). There has been recent debate about the relevance of BMI in determining health risk. Physical fitness, not BMI, has been suggested to be a better predictor of mortality or morbidity, even among overweight individuals (Blair, et al., 1993). As such, participants' BMI values were used as a descriptive measure only.

To determine that participants were not engaging in weight loss dieting during the time of the study, body weight was compared during the first and second REE and participants were strongly encouraged to stop dieting during the course of the study. There were no statistically significant differences reported, although weight change did occur between the first and the second REE. Since the participants were characterized as chronic dieters, it was perhaps unrealistic to believe that all weight loss practices would cease during the research protocol. This was a possible limitation of the research design. Weight change over time would be important to try to control for in future studies with this population.

3. Body composition

Assessment of body composition in this study revealed a significant difference of LBM between the two research groups; the high REE group had

greater amounts of LBM than the low REE group. It was hypothesized that LBM would be different between groups as LBM is the most metabolically active tissue and accounts for 50 to 80% of variation in REE (Ravussin, Bogardus, 1989; Bouchard, et al., 1989; Sims, Danforth, 1987; Ravussin, et al., 1982; Bogardus, et al., 1986; Astrup, et al., 1992). Other influences on REE include age, gender, and genetics (Rice, et al., 1996). Genetic differences could explain higher LBM among female chronic dieters with higher REE (Bouchard, et al., 1989). Another possible explanation for this difference includes variations in time spent weight training as weight training is known to cause lean tissue hypertrophy.

Although the level of LBM differed between both groups, no other differences were detected with respect to fat mass, bone mass, or total body weight when measured using DXA. The only body compartment that DXA does not assess directly is total body water (DXA assumes a total body water content of 73.2%). If body water content was controlled for, additional body composition differences may have emerged between groups.

The ratio of abdominal:gluteal fat mass was also different between the two groups. High REE chronic dieters had higher ratios of abdominal:gluteal fat. Absolute amounts of abdominal and gluteal fat were not different between groups, however, specific trends were noted in regional fat mass relative to total body fat. High REE chronic dieters tended to have more abdominal fat (8.5% vs. 7.5%) and less gluteal fat (24% vs. 26%) as percentages of total body fat than low REE women. The measures of WHR support DXA findings in that the high REE group had higher values for WHR.

Correlations strengthened these findings by demonstrating that REE/kg LBM was positively associated with abdominal fat mass, percent abdominal fat mass, and ratio of abdominal:gluteal fat (Appendix L, Table 14). Although these differences in the ratio abdominal:gluteal fat distribution were not anticipated, an increased abdominal fat mass may be the result of genetic predisposition, increased androgen hormonal activity, increased dietary fat intake, decreased physical activity, or altered rates of lipolysis (Krotkiewski, 1988). It is possible that any one or a combination of these factors could explain the findings in the high

REE group.

Abdominal fatness is associated with increased prevalence of glucose intolerance, insulin resistance, elevated blood pressure, and elevated blood lipids in both males and females (Pouliot, et al., 1994). It has been suggested that the abdominal or android fat pattern may represent an increase in the size and/or number of more metabolically active intra-abdominal fat cells (Kissebah, et al., 1982). These fat cells release free fatty acids directly into the portal circulation which might interfere with insulin clearance in the liver, thus affecting various metabolic processes (Krotkiewski, 1988). Therefore, it is postulated that the insulin resistance observed in the high REE group may be the result of increased abdominal fatness.

As Manore, et al., (1991) reported, dieters were significantly heavier and fatter than non-dieting controls, but fat-free mass was similar between groups. It is suggested that repeated bouts of dieting may alter body composition by increasing the efficiency of food utilization (Brownell, 1987). On average, the participants in the present study were heavier and fatter than reference standards (McArdle, Katch, Katch, 1991). However, it has not been determined whether women diet because they are heavier or if they become heavier because they diet.

DXA has been found to under-estimate and over-estimate body fat mass (Milliken, Going, Lohman, 1996; Aloia, et al., 1995; Snead, Birge, Kohrt, 1993; Pritchard, et al., 1993) as compared to hydrodensitometry. However, recent developments in DXA technology may allow for better estimations of whole body composition (Kohrt, 1998). Body composition options for the Hologic QDR 4500A system have been revised to improve the calibration of percent body fat (Hologic Release Notes, 1998). Independent cross-calibration studies between the Hologic QDR 4500 and hydrodensitometry have provided similar results (Wegner, et al., 1993; Snead, Birge, Khort, 1993). The reanalysis of these data indicated that DXA percent body fat was equivalent to hydrodensitometry percent body fat to within ±2% over the entire range of percent body fat from extremely lean (~5% body fat) to extremely obese (~50% body fat) participants. It is

important to realize that for each manufacturer of DXA technology (Lunar, Norlund, Hologic), results may vary according to the instrument model, mode of data collection (pencil beam vs. array beam), and software used to analyze the data (Kohrt, 1995). All of these parameters must be considered before determining the validity of DXA for body composition assessment. It appears that the corrections made to the Hologic QDR 4500A series support its application in assessing whole body composition.

4. Aerobic fitness and physical activity

There were no differences noted in terms of aerobic fitness or physical activity between high and low REE groups. When participants were grouped according to aerobic fitness level, the high fit group possessed greater LBM, a characteristic commonly associated with fitness. This finding is supported by correlational analysis when absolute fitness is compared to LBM (Appendix L, Table 17).

Specific attributes, also associated with fitness levels in this study, would not be considered desirable from a chronic dieter's perspective (higher body weight, higher dietary intake, and lower dietary restraint). One of the primary goals of a chronic dieter is to achieve weight loss through dietary restriction and/or restraint. It appears that low fit individuals closely match the description of chronic dieters and possess attributes commonly associated with restriction of dietary intake including low REE, low LBM, low dietary intake, and high dietary restraint.

Physical activity did not correlate significantly with aerobic fitness as shown in previous research (Baecke, et al., 1982) (Appendix L, Table 18). Even when participants were grouped by aerobic fitness, activity levels did not reach significance (Table 7). The assessment tool used to quantify physical activity (Baecke questionnaire with three indices each scored on a 5-point Likert scale for a total score out of 15) was not as sensitive as the measurement of aerobic fitness (cycle ergometer test to a plateau in O₂ consumption). Genetics may also account for variations between aerobic fitness and physical activity (Bourchard,

et al., 1992). These differences could not be detected with the methods used in this research.

The majority of participants (n=24/29, 82.8%) did not reach maximal VO₂ uptake criteria (plateau in O₂ consumption). This finding suggests factors other than physical exhaustion may have influenced aerobic fitness levels (lack of motivation, high degree of anxiety, unfamiliarity with equipment). Inexperience with exercise to exhaustion may preclude most participants from reaching maximal VO₂ uptake, resulting in artificially low fitness scores (Howley, Bassett, Welch, 1995). However, both groups contained similar numbers of participants reaching peak VO₂, thus providing a reasonable means for comparison between groups.

For each of the indices of physical activity (work, sports, and leisure), mean group scores did not vary from reference data. Compared to mean scores for 55 Dutch women aged 20-70 years (7.5 ± 1.1) , the participants in the present study were similar in physical activity scores (7.14 ± 1.2) (Pols, et al., 1995; Baecke, Burema, Frijters, 1982). These findings suggest that healthy Dutch women do not engage in more (or less) physical activity than Canadian female chronic dieters.

Although the study participants as a group were considerably less fit than the average Canadian women aged 30-39 years (Canadian Fitness Standards Testing, 1986), this did not appear to influence REE. As well, physical activity levels were not associated with variations in REE. These results are similar to those reported in the literature (Albanes, et al., 1990; Cauley, et al., 1987).

5. Biochemical indices

The OGTT provided unanticipated results in terms of insulin response. The high REE group exhibited insulin resistance compared to the low REE group. The high REE group produced almost double the amount of insulin for the same glucose load (150-g white bread, 75-g carbohydrate). These findings may be explained by impaired glucose metabolism in the high REE group. Increased abdominal fat is associated with decreased insulin sensitivity as assessed with a

euglycemic hyperinsulinemic clamp technique and an OGTT (Kissebah, et al., 1982; Carey, et al., 1996). This literature demonstrated a negative and significant relationship (r=-0.89, p=<0.001) between central abdominal fat and insulin sensitivity in normal- (BMI <25 kg/m²) and overweight (BMI ≥25 kg/m²) women with varying degrees of glucose tolerance (normal to non-insulin dependent diabetes mellitus). Insulin resistance may be of equal or greater prevalence in thin women as in heavy women depending on levels of central abdominal fat. In addition, increased abdominal fat was associated with impaired insulin sensitivity and with reduced glycogen synthesis (measured by non-oxidative glucose disposal) (Carey, et al., 1996). This finding is consistent with the reduced responsiveness of muscle glycogen synthase in women with android obesity (Evans, Murray, Kissebah, 1984). Increased abdominal fat in the high REE group could be explained by these alterations in enzyme function, although this variable was not measured in the present study.

Correlations between REE/kg LBM and all insulin time points (fasting, 30-, 60-, 90-, 120-minute, and insulin AUC) were reported as positive and significant (Appendix L, Table 15). This strengthens the finding of group insulin differences between high and low REE participants. Also correlating negatively with insulin 90-, 120-minute and insulin AUC was relative VO_{2max} (mL O₂/min/kg LBM) (Appendix L, Table 18). Higher degrees of fitness/kg of metabolically active tissue tend to yield lower insulin values during an OGTT. It appears that lower REE, lower ratios of abdominal:gluteal fat mass, and higher levels of aerobic fitness may be associated with normal insulin response during an OGTT.

Although the amount of insulin produced by the high REE group was considerably higher than the low REE group, these results were not considered clinically significant. Levels of insulin production for a similar 2 hour OGTT performed with seven normal weight students (three female, four male; mean BMI $23.8 \pm 1.0 \text{ kg/m}^2$) were similar to those produced by the high REE group ($31.0 \pm 14.3 \text{ vs } 36.6 \pm 20.2$, respectively) (Wolever, Bolognesi, 1996). These data were also confirmed with non-significant group by time interaction as determined from repeated measures ANOVA. These data suggest that although the pattern

of change over time was similar between groups, the differences did not reach clinical significance. Variations with respect to insulin AUC are indicative of group differences, regardless of clinical significance.

Leptin values decreased 120-minutes post-prandially in all participants. This response may be explained by diurnal variations in leptin rather than a direct response to feeding. In a study by Sinha, et al., (1996), leptin levels decreased from approximately 0300 hours to 0930 hours in lean, obese, and obese non-insulin dependent diabetic participants irrespective of 0800 hour breakfast. In the present study, all leptin samples were drawn in the early morning. It appears that the decrease in serum leptin levels after breakfast is the continuation of decline in nighttime acrophase (Laughlin, Yen, 1997; Weigle, et al., 1997; Sinha, et al., 1996).

High and low REE groups did not differ with respect to fasting or 120-minute leptin, but fasting leptin did correlate positively with REE/kg LBM (Appendix L, Table 15). This finding was supported by recent scientific literature that states leptin is an important regulator of energy expenditure mostly through the inhibition of neuropeptide Y and the stimulation of the sympathetic nervous system (Stephens, et al., 1995; Giacobino, 1996). However, data from Kennedy, et al., (1997) suggested that leptin may regulate body weight in humans predominantly by affecting behavioural mechanisms and satiety, rather than REE. More research is necessary to clarify the complex relationships between leptin and REE.

Fasting leptin and fasting insulin were significantly correlated. However, by partial regression analysis, plasma insulin was not significantly correlated with plasma leptin independent of BMI or percent body fat. These findings were supported by the research of Havel, et al., (1996) who measured fasting leptin and insulin levels in 38 normal- and overweight/obese women (as defined by BMI <27.3 or >27.3 kg/m², respectively). Fasting insulin and fasting leptin levels in their study were positively correlated (r=0.61, p<0.0001), but not after accounting for differences in BMI and percent body fat. It is unclear whether this relationship between insulin and leptin is the result of the coexisting adiposity or due to the

regulation of leptin by insulin. The latter suggestion is particularly interesting since fasting insulin did not correlate with plasma leptin independent of adiposity.

Kennedy, et al., (1997) and Nicklas, et al., (1997) found leptin levels increased three times more rapidly with progressively increasing percent body fat in women than in men over the same range of percent body fat. These data suggest that women require higher leptin levels than men to achieve similar biological end points. As such women could perhaps be described as leptin resistant, relative to men. Leptin resistance may provide a partial explanation of why women have a greater percent body fat than men and why women find it more difficult to sustain a diet-induced weight loss (Kennedy, et al., 1997).

Kennedy, et al., (1997) determined that women experience hyperleptinemia (20% increase over baseline values) during the final 60 minutes of a 180 minute induced hyperinsulinemic state (euglycemic hyperinsulinemic clamp). Kolaczynski, et al., (1996b) demonstrated hyperleptinemia after 48 hours during a prolonged (64-72 hours) euglycemic hyperinsulinemic clamp protocol. However, they only tested eight women. Hyperinsulinemia was associated with higher WHR and abdominal fat mass in women (Kissebah, et al., 1982; Carey, et al., 1996). The condition of hyperleptinemia in the present study was observed only in terms of high fasting values of leptin (n=10 with fasting leptin >31.8 ng/mL). Fasting insulin values of hyperleptinemic participants were significantly higher than those with normal-to-low values of leptin (n=10 with fasting leptin <19.8 ng/mL). As well, fasting insulin and fasting leptin values were significantly correlated between all participants (n=30). Total insulin AUC was correlated with the ratio of abdominal:gluteal fat mass even after controlling for BMI and percent body fat. However, fasting leptin was not correlated to the ratio of abdominal:gluteal fat, but was significantly correlated to total abdominal fat. Based on these findings and others (Kennedy, et al., 1997), leptin is believed to regulate overall body fat content, which can then be directed by predominant sex hormones (free testosterone) (Evans, et al., 1983) to an upper body (abdominal) distribution. Relative insulin resistance as a function of abdominal body fat distribution would then explain the association between insulin and higher leptin

concentrations. As such, insulin resistance would not be the direct consequence of hyperleptinemia but, rather, the by-product of accumulated abdominal fat that occurs in the setting of progressive generalized obesity as observed (on average) in the high REE group of the present study.

As reported in the literature, changes in thyroid status are controlled by dietary intake (Mathieson, et al., 1986; O'Brian, et al., 1980). Conversion of T_4 to r- T_3 (5'-monodeiodination of T_4) increases with caloric restriction, carbohydrate restriction, and prolonged exercise (O'Connell, et al., 1978). Differences observed in thyroid status between the two groups of chronic dieters provide evidence for increased amounts of T_4 being converted to r- T_3 as observed during thyroid hormone economy (Loucks, et al., 1992; van der Heyden, et al., 1986). T_4 and r- T_3 were also positively correlated with relative measures of REE (kcal/day/kg LBM) (Appendix L, Table 15). As the high REE group had greater amounts of T_4 and r- T_3 , it would be expected they were engaging in greater caloric restriction or prolonged exercise, relative to the low REE group. This theory was not upheld by the supporting data in the present study.

As a percentage of total energy, the high REE group tended to consume less carbohydrate. Low carbohydrate intakes are associated with increased r-T3, which may provide evidence to support the r-T3 differences between groups. Although there were significant differences between groups, all thyroid values (group means) were within normal ranges (Appendix D). The finding that all thyroid hormone results were within normal ranges diminishes the clinical significance of group differences.

Chomard, et al. (1985) reported that 44 obese women (stable body weight and no dietary restrictions, BMI=34.2 \pm 0.9 kg/m²) did not differ with respect to T₄ and r-T₃ status when compared to normal weight controls. However, T₃ levels were significantly lower in obese women when compared to normal weight women. These differences were attributed to non-significant differences in nutrient intake (obese women tended to eat less carbohydrate and more fat than normal weight women). Nonetheless, these findings do not support the differences seen between groups in the present study.

Loucks and Heath (1994) observed thyroid hormone changes (suppression of T_3 and elevation of r- T_3) associated with decreasing caloric intake and increasing energy expenditure (energy drain theory). Changes in T_4 were not observed during this protocol. Loucks and Heath (1994) suggested that these changes could be prevented by repleting energy to 25 kcal/kg LBM/day regardless of differences in physical activity. However, the present study does not support the energy drain theory since both high and low REE groups consumed similar amounts of dietary energy (kcal) per LBM (high REE group, 51.65 kcal/kg LBM/day; low REE group, 50.88 kcal/kg LBM/day).

Unexpectedly, measures of T_3 were not different between groups, nor were they correlated with REE (absolute or relative). T_3 is the most hormonally active of the three major thyroid hormones, but it appears that variations in energy expenditure are not related to T_3 in female chronic dieters.

6. Dietary intake

There were no differences between groups in terms of dietary intake. However, it is possible that there were differences in dietary intake between high and low REE groups in the present study that were not detected using the FFQ. Alternatively, the use of a 3- or 7-day diet records could have encouraged respondent bias (participants alter dietary intake to reflect more desirable intakes). Preoccupation with food and its nutritional composition are common characteristics of chronic dieters. Using the FFQ helped to shift the focus from actively recording food intake to passively recording previous intakes. Several participants remarked while completing the FFQ that they did not realize how much of a certain food they ate until considering it in the context of the FFQ. This realization may not have been possible with a food record or 24-hour recall.

Slightly higher levels of fat intake may have contributed to differences in body composition of the high REE group if energy intake exceeded energy expenditure (positive energy balance). Several investigators found that the mean intakes of restrained women to be 1900-2000 kcal/day as compared to 2300 kcal/day in unrestrained, normal weight women (Laessle, et al., 1989a; Tuschl, et

al., 1990b). These estimates agreed with measures of 24-hr energy expenditure in restrained and unrestrained women using the doubly labelled water technique (Tuschl, et al., 1990b). Based on these findings, the high REE group would be considered unrestrained as their average energy intake was estimated to be approximately 2300 kcal/day. The low REE group consumed approximately 2000 kcal/day, which would confirm their classification as "restrained eaters". This difference may also be a reflection of a greater energy requirement for women with a high REE. More detailed energy balance analyses (doubly labelled water) would have to be conducted to help explain these variations.

7. Dietary restraint

As expected, participants with high REE scored lower on the dietary restraint scale (they were less restrained). This finding is substantiated by Platte, et al., (1996) who observed reduced REE in restrained eaters even after controlling for LBM. Restraint was assessed using the Three-Factor Eating Questionnaire (Stunkard, Messick, 1985). The significantly lower REE in restrained eaters could be a cause or a consequence of their eating behaviour. If the lower REE is the result of genetic predisposition, the restrained eating style can be thought of as a behavioural adaptation used to prevent gaining weight or becoming overweight.

Low restraint was indicated in the high REE group by a tendency to consume more fat than the low REE group. It appears that within a group of chronic dieters there exist sub-groups of restrained eaters. Thus chronic dieting, in and of itself, may not predict high dietary restraint.

High dietary restraint correlated with decreased fat intake, but not with decreased energy intake (Appendix L, Table 20). Other researchers have found similar results. van Strien, et al., (1986) and Tepper, Trail, Shaffer, (1996) found that dietary restraint correlated negatively with dietary fat intake using the same dietary assessment tool (FFQ; Tepper, Trail, Shaffer, 1996) which suggested that restrained eaters restrict dietary fat, but not total energy. There was a trend for unrestrained eaters (high REE group) to consume more fat than restrained

eaters (low REE group) in the present study. In an effort to reduce fat intake, highly restrained chronic dieters may choose low-fat foods more often. Thus, fat intake would decrease, but total energy intake may stay the same (or even increase). This may explain the differences observed between chronic dieters with respect to dietary fat intake and dietary restraint.

In an effort to explain why the low REE group exhibited higher dietary restraint scores than the high REE group, success at dieting has been examined. Ogden (1993) observed that unsuccessful dieters scored higher on dietary restraint scales than successful dieters. Thus, it appears that high restraint may be an indicator of failed attempts at dieting. As demonstrated by the initial research conducted by McCargar and McBurney (1996), chronic dieters with lowered REE also started dieting at a younger age, had reached a higher maximum weight as an adult, and had dieted more frequently in the previous year than the remainder of the participants.

Classification of restrained and non-restrained participants was completed to determine if chronic dieters were more or less restrained than reference norms. According to the literature, the average dietary restraint score for obese women (similar in weight to present study participants) was 3.0. Three participants with a high REE and nine participants with a low REE scored over 3.0 on the restraint scale. On average, high REE participants would be considered non-restrained eaters (mean dietary restraint score of 2.81 ± 0.52), while low REE participants would be considered restrained eaters (mean dietary restraint score of 3.52 ± 0.84). The average values for low REE participants were not dissimilar from failed dieters (Ogden, 1993) or individuals attending Weight Watchers (Wardle, 1987), but the average dietary restraint scores for high REE participants were significantly different from these groups (p<0.002).

Dietary restraint was positively correlated with three subscales of the MBSRQ. Appearance orientation, health orientation, and overweight preoccupation were associated with high restraint scores (Appendix L, Table 20). This infers that the more intent the individual is in maintaining their appearance and/or health, the more preoccupied they are with their body weight and the

more effort is put into restricting dietary intake. Bezner, Adams, and Steinhardt (1997) found that lower physical self-esteem was associated with restrained eating and body dissatisfaction in 386 women of 35.5 ± 12.5 years of age. Bezner's (1997) findings added another perspective regarding dietary restraint, body image, and self-esteem; the lower the self-esteem, the higher the dietary restraint, and the higher the body weight preoccupation. Cultural messages that tend to provide a constant reminder of failure to achieve the ideal body shape often contribute to a lowered physical self-esteem (Wiseman, et al., 1993; Brownell, 1991). These same messages may be responsible for the dietary restraint practices among female chronic dieters in the present study.

8. Body image

As there were no differences between high and low REE groups on the basis of body image, it appears that chronic dieters are relatively similar in this regard. However, several subscales of the MBSRQ correlated significantly with REE when compared with correlational analysis. These findings were not expected and could not be explained by mean group differences. Only when compared to reference norms do differences in body image appear, and then, only for three specific subscales of the MBSRQ. Chronic dieters were more concerned about their appearance, less satisfied with their bodies, and rated their weight heavier than reference norms.

Self-classified weight was higher than reported for reference norms, but actual weight was also higher than that for reference norms. Reference data was obtained from US national surveys based on 1070 females (Cash, et al., 1986). Participants were divided into actual weight categories based on self-reported height and weight. Respondents within 20% of ideal body weight (Metropolitan Life Insurance Tables) were considered normal weight. Using these criteria, 14% of the women were overweight (>20% of ideal body weight). Using the same criteria with the present research participants, 80% were considered overweight (marginally to morbidly). This suggests that the present research participants were accurate in assessing their self-classified weights.

Self-classified weight correlated positively with actual weight, BMI, body fat, and percent body fat all at. Brodie and Slade (1988) reported similar results among 100 women aged 30.8 \pm 9.38 years, 164.9 \pm 5.5 cm in height and 63.7 \pm kg in weight. Percent body fat was assessed by hydrodensitometry, and electrical impedance. These measures of body composition were highly correlated (p<0.01) and yielded similar average indices of percent body fat. Participants in this study reported higher body dissatisfaction with higher percent body fat (r=0.25, 0.24, and 0.32 with hydrodensitometry, electrical impedance, and skinfolds, respectively; p<0.05). Dieting correlated positively with BMI (p<0.01) suggesting that elevated body weight is positively related to self-reported binge-eating, desire to diet, and general body dissatisfaction (Brodie, Slade, 1988; Rosen, et al., 1987; Davis, et al., 1994a). The average BMI for the present study was $31.6 \pm 7.4 \text{ kg/m}^2$, higher than that reported by Brodie and Slade (1988) as influencing body image evaluation in their participants. Negative feelings towards appearance and high levels of body dissatisfaction noted in the present study were also related to a higher BMI. A high BMI along with high percent body fat appear to be motivators to initiate and maintain a weight-loss diet.

Other dieting catalysts include concerns about shape and weight, including fears about being or becoming fat (Brownell, Rodin, 1994; Cash, et al., 1986). Similar concerns were expressed by the participants in the present study as determined by their negative appearance evaluations. A majority of the study participants (70%; 21/30) indicated a negative appearance evaluation (scoring in the unfavourable direction relative to a 3.0, the neutral midpoint, on the 1-5 point response scale). In a 1994 body image survey, Cash and Henry (1995) reported 47.9% of American women scored less than three for appearance evaluation. The prevalence of negative body image among women had increased from 30% in the 1985 body image survey (Cash, Winstead, Janda, 1986) to 48% in the 1994 survey (Cash, Henry, 1995). It appears that the body images of the participants in the present study are more negative than either of the previously noted population surveys indicate.

CHAPTER SIX CONCLUSION AND RECOMMENDATIONS

A. CONCLUSION

The present research provided evidence of differences in metabolic variables between two groups of female chronic dieters. Of the differences between the two groups, those relating to body composition (LBM) were most predictive of REE. The differences in LBM were correlated with differences in aerobic fitness, but the groups did not differ with respect to aerobic fitness or activity. The research also revealed that high REE was associated with insulin resistance compared to low REE. Chronic dieters with a high REE produced almost 100% more insulin for the same glucose load as chronic dieters with a low REE. Insulin resistance is positively correlated with increased abdominal fat mass and this was shown to be true for women in the high REE group. Differences in leptin were likely due to normal diurnal variations more than the influence of feeding or REE. Higher T₄ and r-T₃ values in the high REE group supported the association of thyroid production and metabolism, but these differences have been noted in energy restricting situations of which the high REE group was not. More research is required to elucidate the complex interactions of insulin, leptin, and thyroid with energy metabolism. No differences were discovered between groups in terms of dietary intake, but there was a trend for the high REE group to consume more fat. Differences in dietary restraint were noticed between groups with the high REE group being less restrained. This may partially explain lowered REE in chronic dieters as dietary restraint has been shown to reduce metabolic rate. High dietary restraint was associated with negative body image. The more invested a chronic dieter was in their appearance and their health, the more preoccupied they were with their body weight and the more effort was dedicated to restricting dietary intake. Among a group of female chronic dieters, it appears there exists a subgroup of women with lowered REE, LBM, metabolic hormones, and increased dietary restraint. As well, body image was affected on several levels by chronic dieting. These results

must be interpreted within the context of the present research design, but it is hoped these findings would be applicable in a general sense to female chronic dieters as a population.

B. RECOMMENDATIONS

1. Limitations of the study

The main limitation of this study was the reliance on self-report measures for the determination of dietary intake and physical activity. If more sensitive measures were utilized, differences may have become statistically significant which, in turn, would have allowed for more power in the interpretation of the results. The second limitation of this study was the limited biochemical assessment of chronic dieters. Enhanced interpretation of biochemical mechanisms may have resulted if catecholamine levels had been assessed. Catecholamines are strongly associated with both energy metabolism and insulin resistance and may have provided another explanation of the existing differences between groups. The third limitation to this research was the observational design. Results will have limited application to the population of chronic dieters in general and associations between variables do not imply causation. The final limitation of this study was the possible confounding of continued dieting during the measurement periods. Although weight change from the first to second determination of REE was not statistically different, the high REE group tended to lose weight while the low REE group tended to gain weight. Repeated measures of REE during the entire course of the study may have provided more detailed monitoring of the influences of dieting on REE. These changes could be implemented in future research projects of this nature.

2. Future research

Several participants could be characterized as highly fit. Chronic dieters with high aerobic fitness levels were heavier, leaner, and consumed more energy. It would be prudent for researchers to determine the associations between fitness, body composition, and chronic dieting as previous research has

demonstrated reduced LBM and REE with diet and exercise regimes.

Adding another group of non-dieting controls (two additional groups; obese and lean) to the present research design may improve the application of the findings to distinguish between energy metabolism of dieters and non-dieters. This may prove challenging as the prevalence of dieting is increasing among women. Specifically, understanding the psychological differences between chronic dieters and non-dieters would provide additional information that could be used to assist dieters adopt a more balanced approach to health and wellness.

Body image therapy would benefit the present participants as the knowledge of their metabolic health did little to alleviate their desire to lose weight. Interventions would be useful to determine if body image therapy would reduce the characteristics that typify chronic dieters (for example, persistent overconcern with body weight and shape, restriction of food choices). This type of program could be offered by trained group facilitators as a means of helping women achieve freedom from chronic dieting.

3. Recommendations for chronic dieters

Based on the research findings, chronic dieters should be encouraged to move away from dietary restraint as a means of self-acceptance and enhanced wellness. Sociocultural evidence suggests that there is ever increasing pressure on women to be thin. This pressure drives women to want to be thinner than what might be realistically achieved or required for good health. As health professionals, our goal is to empower women to achieve and maintain a healthy perspective with respect to body weight and shape. Individuals posses unique determinants of health influenced by genetic, physiological, social, and psychological factors. All of these factors must be considered in the nutrition counselling context.

The following is a summary of recommendations to chronic dieters based on the observations of the present study and previous reviews (Manore, 1996; Gaesser, 1996; Burgard, Lyons, 1994; Oberrieder, et al., 1995).

a) Share attitudes that foster healthy eating styles. Determine the most

appropriate and realistic changes (if any) to the individual's eating style depending on their schedule, preferred foods, preparation resources, and finances. Encourage responsibility, enjoyment, and normalization of all eating activities. Chronic dieters should be encouraged to identify reachable goals.

- b) Emphasize personal health and well being, not weight. Measure progress in health by improvements in fitness, glucose tolerance, insulin sensitivity, blood pressure, blood lipids, stress management, and decreased dietary restraint. Underscore that the body cannot be shaped and molded at will and that the pursuit of an unrealistic body shape increases the risk for eating disorders, promotes preoccupation with food, and may have undesirable physiological consequences.
- c) Put the role of biology and weight regulation in perspective. Genetics control a major proportion of physiological variables (REE, LBM, weight, and height). Focus on improving environmental influences to weight regulation such as nutritious eating, active lifestyle, and positive self-image.
- d) Encourage chronic dieters to attain a realistic proportion of their body size. Some women do not have an accurate view of their bodies. Integrate nutrition counselling and body image awareness. Recognize personal boundaries when counselling on body image and use other professional resources to augment positive nutrition messages (psychological counselling).
- e) Promote regular daily activity that can be embraced for a lifetime. Emphasize that physical activity should be valued, not as a weight-loss method, but for its contribution to enhanced mental and physical health. Discuss barriers to physical activity and encourage the development and maintenance of a supportive social network to overcome those barriers.

As nutrition professionals, it is essential for dietitians to provide sound nutrition messages to chronic dieters, regardless of their weight, so they may enjoy all of the health benefits of nutrition. A continued emphasis on health, completely separate from a focus on dietary restraint, may provide the support chronic dieters require in order to adopt more beneficial lifestyle practices.

LITERATURE CITED

- Acheson ED, Doll R. Dietary factors in carcinoma of the stomach: A study of 100 cases and 200 controls. Gut 1964;5:126-131.
- Albanes D, Conway JM, Taylor PR, Moe PW, Judd J. Validation and comparison of eight physical activity questionnaires. Epidemiology 1990;1:65-71.
- Allison DB. Handbook of assessment methods for eating behaviours and weightrelated problems: Methods of theory and research. Thousand Oaks: Sage Publications, 1995.
- Allison DB, Kalinsky LB, Gorman BS. The comparative psychometric properties of three measures of dietary restraint. Psychological Assessment 1992;4:391-398.
- Aloia JF, Vaswani A, Ma R, Flaster E. Comparative study of body composition by dual-energy x-ray absorptiometry. Journal of Nuclear Medicine 1995;36:1392-1397.
- Altabe M, Thompson JK. Body image changes during early adulthood. International Journal of Eating Disorders 1993;13:323-328.
- Andersen RE, Wadden TA. Validation of a cycle ergometry equation for predicting steady-rate VO2 in obese women. Medicine and Science in Sports and Exercise 1995;27:1457-1460.
- Anderson SA. Guidelines for use of dietary intake data. Life Sciences Research Office, Federation of American Societies for Experimental Biology, Bethesda, Maryland, 1986.
- Andersson KE. Drugs blocking adrenoreceptors. Acta Medica Scandinavia 1982;665:9-17.
- Arciero PJ, Goran MI, Poehlman ET. Resting metabolic rate is lower in women compared to men. Journal of Applied Physiology 1993;75:2514-2520.
- Astrup A, Buemann B, Christensen NJ, Madsen J, Gluud C, Bennett P, Svenstrup B. The contribution of body composition, substrates, and hormones to the variability in energy expenditure and substrate utilization in premenopausal women. Journal of Clinical Endocrinology and Metabolism 1992;74:279-286.
- Baecke J, Burema A, Frijters JER. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. American Journal of Clinical Nutrition 1982;36:936-942.
- Bale P, Davies J. Effects of menstruation and contraceptive pill on the performance of physical education students. British Journal of Sports Medicine 1983;17:46-50.
- Bale P, Nelson G. The effects of menstruation on performance of swimmers. Australian Journal of Science and Medicine in Sport 1985;Mar:19-22.

- Beals KA, Manore MM. The prevalence and consequences of sub-clinical eating disorders in female athletes. International Journal of Sport Nutrition 1994;4:175-195.
- Beck-Nielson H. Clinical disorders of insulin resistance. In Alberti KGMM, DeFronzo RA, Keen H, Zimmet P, eds. International textbook of diabetes mellitus. John Wiley & Sons, 1992:631-550.
- Bennett WI. Beyond overeating. New England Journal of Medicine 1995;332:673-674.
- Ben-Porat M, Sideman S, Bursztein S. Energy metabolism rate equation for fasting and post-absorptive subjects. American Journal of Physiology 1983;244:R764-9.
- Berke EM, Gardner AW, Goran MI, Poehlman ET. Resting metabolic rate and the influence of the pre-testing environment. American Journal of Clinical Nutrition 1992;55:626-629.
- Berthouze SE, Minaire PM, Castells J, Busso T, Vico L, Lacour JR. Relationship between mean habitual daily energy expenditure and maximal oxygen uptake. Medicine and Science in Sports and Exercise 1995;27:1170-1179.
- Bezner JR, Adams TB, Steinhardt MA. Relationship of body dissatisfaction to physical health and wellness. American Journal of Health Behaviour 1997;21:147-155.
- Blackburn GL, Wilson GT, Kanders BS, Stein LJ, Lavin PT, Adler J, Brownell KD. Weight cycling: the experience of human dieters. American Journal of Clinical Nutrition 1989;49:1105-1109.
- Blair SN, Kohl HW, Gordon NF, Paffenbarger RS. How much physical activity is good for health? Annual Review of Public Health 1992;13:99-126.
- Blair SN, Shaten J, Brownell KD, Collins G, Lissner L. Body weight change, all-cause mortality, and cause-specific mortality in the Multiple Risk Factor Intervention Trial. Annals of Internal Medicine 1993;119:749-757.
- Bogardus C, Lillioja S, Ravussin E, Abbott W, Zawadzki JK, Young A, Knowler WC, Jacobowitz R, Moll PP. Familial dependence of the resting metabolic rate. New England Journal of Medicine 1986;315:96-100.
- Bonadonna RC, DeFronzo RA. Glucose metabolism in obesity and type II diabetes. In: Bjorntorp P, Brodoff BN, eds. Obesity. Philadelphia: JB Lippincott Company, 1992:474-501.
- Bonen A, et al. Maximal oxygen uptake during free, tethered, and flume swimming. Journal of Applied Physiology 1980;48:232
- Bouchard C. Reproducibility of body composition and adipose tissue measurements in humans. In: Roche AF, ed. Body composition assessment in youth and adult. Columbus, OH: Ross Laboratories, 1985:9

- Bouchard C, Dionne FT, Simoneau J-A, Boulay MR. Genetics of aerobic and anaerobic performances. Exercise and Sport Sciences Reviews 1992;20:27-58.
- Bouchard C, Tremblay A, Nadeau A, Despres JP, Theriault G, Boulay MR, Lortie G, Leblanc C, Fournier G. Genetic effects in resting and exercise metabolic rates. Metabolism 1989;38:364-370.
- Brodie DA, Slade PD. The relationship between body image and body-fat in adult women. Psychological Medicine 1988;18:623-631.
- Brown TA, Cash TF, Mikulka PJ. Attitudinal body-image assessment: factor analysis of the Body-Self Relations Questionnaire. Journal of Personality Assessment 1990;55:135-144.
- Brownell KD. Dieting and the search for the perfect body: Where physiology and culture collide. Behaviour Therapy 1991;22:1-12.
- Brownell KD, Rodin J. The dieting maelstrom. Is it possible and advisable to lose weight? American Psychologist 1994;49:781-791.
- Brownell KD, Steen SN. Weight cycling in athletes: effect on behaviour, physiology, and health. In: Brownell KD, Robin J, Wilmore JH, eds. Eating, body weight, and performance in athletes: disorders of modern society. Philadelphia: Lea & Febiger, 1992:159-171.
- Brownell KD, Steen SN, Wilmore JH. Weight regulation practices in athletes; Analysis of metabolic and health effects. Medicine and Science in Sports and Exercise 1987;19:546-556.
- Bruch H. Psychological aspects in overeating and obesity. Psychosomatics 1961;5:269-274.
- Buechler KF, Beynen AC, Geelen MJH. Studies on the assay, activity and sedimentation behaviour of acetyl-CoA carboxylase from isolated hepatocytes incubated with insulin or glucagon. Biochemical Journal 1984;221:869-874.
- Burgard D, Lyons P. Alternatives in obesity treatment: Focusing on health for fat women. In Fallon P, Katzman MA, Wooley SC, eds. Feminist perspectives on eating disorders. New York: Guilford Press, 1994:212-230.
- Canadian Standardized Test of Fitness. 3rd Edition. Fitness and Amateur Sport Canada, 1986;34.
- Carey DG, Jenkins AB, Campbell LV, Freund J, Chisholm DJ. Abdominal fat and insulin resistance in normal and overweight women: direct measurements reveal a strong relationship in subjects at both low and high risk of NIDDM. Diabetes 1996;45:633-638.
- Carey P, Stensland M, Hartley LH. Comparison of oxygen uptake during maximal work on the rowing ergometer. Medicine and Science in Sports and Exercise 1974;6:101

- Caro JF, Sinha MK, Kolaczynski JW, Zhang PL, Considine RV. Leptin: the tale of an obesity gene. Diabetes 1996;42:1455-1462.
- Cash TF, Henry PE. Women's body images: The results of a national survey in the USA. Sex Roles 1995;33:19-28.
- Cash TF, Pruzinsky T. Body images: Development, deviance, and change. New York, NY: Guilford Press, 1990.
- Cash TF. The Multi-dimensional Body-Self Relations questionnaire users manual. Requested from the author 1994;1-8.
- Cash TF. The treatment of body image disturbances. In: Thompson JK, ed. Body image, eating disorders, and obesity. Washington: American Psychological Association, 1996:83-107.
- Cash TF, Winstead BW, Janda LH. The great American shape-up: Body image survey report. Psychology Today 1986;20:30-37.(abst)
- Cash TF, Winstead BW, Janda LH. Your body, yourself: A Psychology Today reader survey. Psychology Today 1985;19:22-26.
- Casperson CJ, Powell KE, Christenson GM. Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. Public Health Reports 1985;100:126
- Cauley JA, LaPorte RE, Sandler RB, Schramm MM, Kriska AM. Comparison of methods to measure physical activity in postmenopausal women. American Journal of Clinical Nutrition 1987;45:14-22.
- Chehab FF, Lim ME, Lu R. Correction of the sterility defect in homozygous obese female mice by treatment with the human recombinant leptin. Nature Genetics 1996;12:318-320.
- Cheney CL, Boushey CJ. Estimating sample size. In: Monsen ER, ed. Research: Successful approaches. Chicago: The American Dietetic Association, 1992:337-346.
- Chomard P, Vernhes G, Autissier N, Debry G. Serum concentrations of total T4, T3, reverse T3, and free T4, T3 in moderately obese patients. Human Nutrition: Clinical Nutrition 1985;39C:371-378.
- Clugston GA, Hetzel BS. Iodine. In: Shils ME, Olson JA, Shike M, eds. Modern nutrition in health and disease. Philadelphia: Lea & Febiger, 1994:252-263.
- Collins S, Kuhn CM, Petro AE, Swick AG, Chrunyk BA, Surwit RS. Role of leptin in fat regulation. Nature 1996;380:677
- Considine RV, Sinha MK, Heiman ML, Kriauciunas A, Stephens TW, Nyce MR, Ohannesian JP, Marco CC, McKee LJ, Bauer TL, Caro JF. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. New England Journal of Medicine 1996;334:292-295.

- Cooney GJ, Denyer GS, Jenkins AB, Storlien LH, Kraegen EW, Caterson ID. In vivo insulin sensitivity of the pyruvate dehydrogenase complex in tissues of the rat. American Journal of Physiology 1993;265:E102-E107.
- Cooney GJ, Storlien LH. Insulin action, thermogenesis and obesity. Bailliere's Clinical Endocrinology and Metabolism 1994;8:481-507.
- Cunningham JJ. Calculation of energy expenditure from indirect calorimetry: assessment of the Weir equation. Nutrition 1990;6:222-223.
- Cusin I, Sainsbury A, Doyle P, Rohner-Jeanrenaud F, Jeanrenaud B. The obgene and insulin. Diabetes 1995;44:1467-1470.
- Daly ME. Vale C. Walker M. Alberti KG. Mathers JC. Dietary carbohydrates and insulin sensitivity: A review of the evidence and clinical implications. American Journal of Clinical Nutrition 1997;66:1072-85.
- Davidson MB. Clinical implications of insulin resistance syndromes. The American Journal of Medicine 1995;99:420-426.
- Davis C, Durnin JVGA, Dionne M, Gurevich M. The influence of body fat content and bone diameter measurements on body dissatisfaction in adult women. International Journal of Eating Disorders 1994a;15:257-263.
- Davis D, Kennedy SH, Ravelski E, Dionne M. The role of physical activity in the development and maintenance of eating disorders. Psychological Medicine 1994b;24:957-967.
- DeFronzo R, Tobin J, Andres R. Glucose clamp technique: a method for quantifying insulin secretion and resistance. American Journal of Physiology 1979;237:E314-E323.
- Dulloo AG, Jacquet J, Girardier L. Auto-regulation of body composition during weight recovery in humans: The Minnesota Experiment revisited. International Journal of Obesity 1996;20:393-405.
- Dulloo AG, Jacquet J, Girardier L. Poststarvation hyperphagia and body fat overshooting in humans: A role for feedback signals from lean and fat tissues. American Journal of Clinical Nutrition 1997;65:717-723.
- Elia M, Livesey G. Theory and validity of indirect calorimetry during net lipid synthesis. American Journal Clinical Nutrition 1988;47:591-607.
- Elliot DL, Goldberg L, Kuehl KS, Bennett WM. Sustained depression of the resting metabolic rate after massive weight loss. American Journal of Clinical Nutrition 1989;49:93-96.
- Emmons L. Predisposing factors differentiating adolescent dieters and nondieters. Journal of the American Dietetic Association 1994;84:725-728, 731.

- Evans DJ, Hoffmann RG, Kalkhoff RG, Kissebah AH. Relationship of androgenic activity to body fat topography, fat cell morphology, and metabolic aberrations in premenopausal women. Journal of Clinical Endocrinology and Medicine 1983;57:304-310.
- Evans DJ, Murray R, Kissebah A. Relationship between skeletal muscle insulin resistance, insulin mediated glucose disposal, and insulin binding: Effects of obesity and body fat topography. Journal of Clinical Investigation 1984;74:1515-1525.
- Fairshter RD, Walters J, Salness K, Fox M, Minh VD, Wilson AF. A comparison of incremental exercise tests during cycle and treadmill ergometry. Medicine and Science in Sports and Exercise 1983;15:549
- Fallon AE, Rozin P. Sex differences in perception of desirable body shape. Journal of Abnormal Psychology 1985;94:102-105.
- Fallon P, Katzman M, Wooley SC. Feminist perspectives on eating disorders. New York, NY: Guilford Press, 1994.
- Ferguson HJ, Marcotte GG, Montpetit RR. A maximal oxygen uptake test during ice skating. Medicine and Science in Sports 1969;1:207
- Ferrannini E. Insulin resistance is central to the burden of diabetes. Diabetes and Metabolism Reviews 1997;13:81-86.
- Ferraro R, Lillioja S, Fontvieille AM, Rising R, Bogardus C, Ravussin E. Lower sedentary metabolic rate in women compared with men. Journal of Clinical Investigation 1992;90:780-784.
- Ferraro R, Ravussin E. Fat mass in predicting resting metabolic rate. American Journal of Clinical Nutrition 1992;56:460-461.
- Foreyt JP, Brunner RL, Goodrick GK, Cutter G, Brownell KD, St. Jeor ST. Psychological correlates of weight fluctuation. International Journal of Eating Disorders 1995;17:263-275.
- Foreyt JP, Goodrick GK. Weight management without dieting. Nutrition Today. 1993;28:4-9.
- Formica C, Atkinson MG, Nyulasi I, et al. Body composition following hemodialysis: studies using dual-energy x-ray absorptiometry and bioelectrical impedance analysis. Osteoporosis 1993;3:192-197.
- Gaesser, G. Big fat lies. New York: Ballantine, 1996:17-29
- Gann N. Minimal error introduced by changing lean body mass water constant. 1997;(personal communication)
- Garrow JS. New possibilities for the treatment of obesity. Bibliotheca Nutritio et Dieta 1986;39:60-67.

- Geliebter A, Maher MM, Gerace L, Gutin B, Heymsfield SB, Hashim SA. Effects of strength or aerobic training on body composition, resting metabolic rate, and peak oxygen consumption in obese dieting subjects. American Journal of Clinical Nutrition 1997;66:557-563.
- Giacobino JP. Role of the β_3 -adrenoreceptor in the control of leptin expression. Hormone and Metabolic Research 1996;28:633-637.
- Gibson R. Food consumption of individuals. In: Gibson R, ed. Principles of nutritional assessment. New York: Oxford University Press, 1990:37-54.
- Gonzalo MA, Grant C, Moreno I, Garcia FJ, Suarez Al, Herrera-Pombo JL, Rovira A. Glucose tolerance, insulin secretion, insulin sensitivity, and glucose effectiveness in normal and overweight hyperthyroid women. Clinical Endocrinology 1996;45:689-697.
- Goran MI, Kaskoun MC, Johnson RK. Determinants of resting energy expenditure in young children. Journal of Pediatrics 1994;125:362-367.
- Gorman BS, Allison DB. Measures of restrained eating. In: Allison DB, ed. Handbook of assessment methods for eating behaviours and weight-related problems: Measures, theory, and research. Thousand Oaks: Sage Publications, 1995:149-184.
- Grande F. Energy expenditure of organs and tissues. In: Kinney JM, ed. Assessment of energy metabolism in health and disease. Columbus, OH: Ross Laboratories, 1980:88-92.
- Grediagin MA, Cody M, Rupp J, Benardot D, Shern R. Exercise intensity does not effect body composition change in untrained, moderately overfat women. Journal of the American Dietetic Association 1995;95:661-665.
- Greiwe JS, Kaminsky LA, Whaley MH, Dwyer GB. Evaluation of the ACSM submaximal ergometer test for estimating VO_{2max}. Medicine and Science in Sports and Exercise 1995;27:1315-1320.
- Grodner M. Forever dieting: chronic dieting syndrome. Journal of Nutrition Education 1992;24:207-210.
- Groff J, Gropper S, Hunt SM. Energy balance and weight control. In: Advanced nutrition and human metabolism. 2nd ed. St. Paul, MN: West Publishing Company, 1995a:466-487.
- Groff J, Gropper S, Hunt SM. Microminerals. In: Advanced nutrition and human metabolism. 2nd ed. St. Paul: West Publishing Company, 1995b:352-419.
- Haarbo J, Gotfredsen J, Hassager C, Christiansen C. Validation of body composition by dual-energy x-ray absorptiometry. Clinical Physiology 1991;11:331-341.
- Halaas JL, Gajiwala KS, Maffei M, Cohen SL, Chait BT, Rabinowitz D, Lalone RL, Burley SK, Friedman JM. Weight-reducing effects of the plasma protein encoded by the obese gene. Science 1995;269:543-546.

- Hamilton BS, Paglia D, Kwan AYM, Deitel M. Increased obese mRNA expression in omental fat cells from massively obese humans. Nature Medicine 1995;1:953-956.
- Hansen NJ, Lohman TG, Going SB, Hall MC, Pamenter RW, Bare LA, Boyden TW, Houtkooper LB. Prediction of body composition in premenopausal females from dual-energy x-ray absorptiometry. Journal of Applied Physiology 1993;75:1637-1641.
- Havel PJ, Kasim-Karakas S, Mueller W, Johnson PR, Gingerich RL, Stern JS. Relationship of plasma leptin to plasma insulin and adiposity in normal weight and overweight women: Effects of dietary fat content and sustained weight loss. Journal of Clinical Endocrinology and Metabolism 1996;81:4406-4413.
- Heinberg LJ, Thompson JK. Social comparison: Gender, target importance ratings, and relation to body image disturbance. Journal of Social and Behaviour Personality 1992;7:335-344.
- Helmrick SP, Ragland DR, Leung RW, Paffenbarger RS. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. New England Journal of Medicine 1991;325:147-152.
- Henry CJK, Hayter J, Rees DG. The constancy of basal metabolic rate in free-living subjects. European Journal Clinical Nutrition 1989;43:727
- Herman CP, Mack D. Restrained and unrestrained eating. Journal of Personality 1975;143:647-660.
- Herman CP, Polivy J, Esses VM. The illusion of counter-regulation. Appetite 1987;9:161-169.
- Herman CP, Polivy J. Restrained eating. In: Stunkard AJ, ed. Obesity. Philadelphia: Saunders, 1980:208-225.
- Hirayama T. Diet and cancer. Nutrition and Cancer 1981;1:67-81.
- Holloszy JO, Coyle EF. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. Journal of Applied Physiology 1984;56:831
- Holmes LJ, Smythe GA, Storlien LH. Monoaminergic activity and the level of the hypothalamus and striatum: Relationship to anticipated feeding and pancreatic insulin responses. Brain Research 1989;496:204-210.
- Hologic Corporation. Hologic QDR® 4500 Technical Manual. Revision B 1995.
- Hologic Release Notes. QDR 4500A and QDR 4500W, Body composition option. # 612-0390 Revision B 1998
- Home PD. Insulin resistance is not central to the burden of diabetes. Diabetes and Metabolism Reviews 1997;13:87-92.
- Horm J, Anderson K. Who in America is trying to lose weight? Annals of Internal Medicine 1993;119:672-676.

- Howley ET, Bassett DR, Welch HG. Criteria for maximal oxygen uptake: review and commentary. Medicine and Science in Sports and Exercise 1995;27:1292-1301.
- Ikeda T, Fujiyama K, Hoshino T, Takeuchi T, Mashiba H, Tominaga M. Oral and intravenous glucose-induced insulin secretion in hyperthyroid patients. Metabolism 1990;50:392-396.
- Jacobi L, Cash TF. In pursuit of the perfect appearance: Discrepancies among self-ideal percepts of multiple physical attributes. Journal of Applied Social Psychology 1994;24:379-396.
- Jacobs DR, Ainsworth BE, Hartman TJ, Leon AS. A simultaneous evaluation of ten commonly used physical activity questionnaires. Medicine and Science in Sports and Exercise 1993;25:81-91.
- Jebb SA, Goldberg GR, Coward WA, Murgatroyd PR, Prentice AM. Effects of weight cycling caused by intermittent dieting on metabolic rate and body composition in obese women. International Journal of Obesity 1991;15:367-374.
- Jensen MD, Kanaley JA, Roust LR, O'Brien PC, Braun JS, Dunn WL, Wahner HW. Assessment of body composition with use of dual-energy x-ray absorptiometry: evaluation and comparison with other methods. Mayo Clinical Proceedings 1993;68:867-873.
- Jequier E, Acheson K, Schutz Y. Assessment of energy expenditure and fuel utilization in man. Annual Reviews of Nutrition 1987;7:187-208.
- Jequier E. Direct and indirect calorimetry in man. In: Garrow JS, Halliday D. eds. Substrate and energy metabolism. London: J. Libbey, 1985;82-92.
- Johansson AG, Forslund A, Sjodin A, Mallmin H, Hambraeus L, Ljunghall S. Determination of body composition: a comparison of dual-energy x-ray absorptiometry and hydrodensitometry. American Journal of Clinical Nutrition 1993;57:323-326.
- Kanarek RB, Ryu M, Przypek J. Preferences for foods with varying levels of salt and fat differ as a function of dietary restraint and exercise but not menstrual cycle. Physiology and Behaviour 1995;57:821-826.
- Katch VL, Sady SS, Freedson P. Biological variability in maximum aerobic power. Medicine and Science in Sports and Exercise 1982;14:21-25.
- Kayman S, Bruvold W, Stern JS. Maintenance and relapse after weight loss in women: Behavioural aspects. American Journal of Clinical Nutrition 1990;52:800-807.
- Kennedy A, Gettys TW, Watson P, Wallace P, Ganaway E, Pan Q, Garvey WT. The metabolic significance of leptin in humans: Gender-based differences in relationship to adiposity, insulin sensitivity, and energy expenditure. Journal of Clinical Endocrinology and Metabolism 1997;82:1293-1300.

- Keys A, Brozek, Henschel A, Mickelsen O, Taylor HL. The biology of human starvation. Minneapolis: University of Minnesota Press, 1950.
- Kinney JM. Energy metabolism. In Fisher JE, ed. Surgical nutrition. Boston: Little Brown and Company, 1983;103-104.
- Kissebah AH, Vydelingum N, Murray R, Evans DJ, Hartz AJ, Kalkhoff RK, Adams PW. Relation of body fat distribution to metabolic complications of obesity. Journal of Clinical Endocrinology and Metabolism 1982;54:254-258.
- Klesges RG, Isbell TR, Klesges LM. Relationship between dietary restraint, intake, physical activity, and body weight: a prospective analysis. Journal of Abnormal Psychology 1992;101:668-674.
- Knight LJ, Boland FJ. Restrained eating: An experimental disentanglement of the disinhibition variables of perceived calories and food type. Journal of Abnormal Psychology 1989;98:412-420.
- Kohl HW, LaPorte RE, Blair SN. Physical activity and cancer: an epidemiological perspective. Sports Medicine 1988;6:222-237.
- Kohrt WM. Body composition by DXA: tried and true? Medicine and Science in Sports and Exercise 1995;27:1349-1353.
- Kohrt WM. Preliminary evidence that DEXA provides an accurate assessment of body composition. Journal of Applied Physiology 1998;84:372-377.
- Kolaczynski JW, Considine RV, Ohannesian JP, Marco C, Opentanova I, Nyce MR, Myint M, Caro JF. Responses of leptin to short-term fasting and refeeding in humans: a link with ketogenesis but not ketones themselves. Diabetes 1996a;45:1511-1515.
- Kolaczynski JW, Nyce MR, Considine RV, Boden G, Nolan JJ, Henry R, Mudaliar SR, Olefsky J, Caro JF. Acute and chronic effect of insulin on leptin production in humans: studies in vivo and in vitro. Diabetes 1996b;45:699-701.
- Krotkiewski M. Can body fat patterning be changed? Acta Medica Scandinavica Supplementum 1988;723:213-223.
- Laasko M. How good a marker is insulin level for insulin resistance? American Journal of Epidemiology 1993;137:959-965.
- Laessle RG, Tuschl RJ, Kotthaus BC, Pirke KM. A comparison of the validity of three scales for the assessment of dietary restraint. Journal of Abnormal Psychology 1989b;98:504-507.
- Laessle RG, Tuschl RJ, Kotthaus BC, Pirke KM. Behavioural and biological correlates of dietary restraint in normal life. Appetite 1989a;12:83-94.
- Laughlin GA, Yen SSC. Hypoleptinemia in women athletes: Absence of a diurnal rhythm with amenorrhea. Journal of Clinical Endocrinology and Metabolism 1997;82:318-321.

- Lawson OJ, Williamson DA, Champagne CM, DeLany JP, Brooks ER, Howat PM, Wozniak PJ, Bray GA, Ryan DH. The association of body weight, dietary intake, and energy expenditure with dietary restraint and disinhibition. Obesity Research 1995;3:153-161.
- Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. New England Journal of Medicine 1995;332:621-628.
- Ley CJ, Lees B, Stevenson JC. Sex- and menopause-associated changes in body-fat distribution. American Journal of Clinical Nutrition 1992;55:950-954.
- Livesey G, Elia M. Estimation of energy expenditure, net carbohydrate utilization, and net fat oxidation and synthesis by indirect calorimetry: evaluation of errors with special reference to the detailed composition of fuels. American Journal of Clinical Nutrition 1988;47:608-28.
- Lohman TG. Dual energy radiography: total body and regional composition. In: Advances in body composition assessment: current issues in exercise science. Illinois: Human Kinetics Publishers, 1992:25-36.
- Londeree BR, Moffitt-Gerstenberger J, Padfield JA, Lottmann D. Oxygen consumption of cycle ergometry is nonlinearly related to work rate and pedal rate. Medicine and Science in Sports and Exercise 1997;29:775-780.
- Loucks AB, Callister R. Induction and prevention of low-T3 syndrome in exercising women. American Journal of Physiology 1993;264:R924-R930.
- Loucks AB, Heath EM. Induction of low-T3 syndrome in exercising women occurs at a threshold of energy availability. American Journal of Physiology 1994;266:R817-R823.
- Loucks AB, Laughlin GA, Mortola JF, Girton L, Nelson JC, Yen SSC. Hypothalamic-pituitary-thyroidal function in eumenorrheic and amenorrheic athletes. Journal of Clinical Endocrinology and Metabolism 1992;65:514-518.
- Lowe MR. The effects of dieting on eating behaviour: A three-factor model. Psychological Bulletin 1993;114:100-121.
- Lu H, Buison A, Uhley V, Jen KLC. Long-term weight cycling in female Wistar rats: effects on metabolism. Obesity Research 1995;3:521-530.
- Maffei M, Halaas JL, Ravussin E, Pratley RE, Lee GH, Zhang Y, Fei H, Kim S, Lalone RL, Ranganathan S, Kern PA, Friedman JM. Leptin levels in human and rodent: measurement of plasma leptin and ob RNA in obese and weight-reduced subjects. Nature Medicine 1995;1:1155-1161.
- Maksud MG, Coutts KD. Comparison of a continuous and discontinuous graded treadmill test for maximal oxygen uptake. Medicine and Science in Sports 1971;3:63-65.
- Manore MM, Berry TE, Skinner JS, Carroll SS. Energy expenditure at rest and during exercise in nonobese female cyclical dieters and in nondieting control subjects. American Journal of Clinical Nutrition 1991;54:41-46.

- Manore MM. Chronic dieting in active women: what are the health consequences? Women's Health Issues 1996;6:332-341.
- Mathieson RA, Walberg JL, Gwazdauskas FX, Hinkle DE, Gregg JM. The effect of varying carbohydrate content of a very-low-caloric diet on resting metabolic rate and thyroid hormones. Metabolism 1986;394:8
- Mazess RB, Barden HS, Hanson JA. Body composition by dual-photon absortpiometry and dual-energy x-ray absorptiometry. In: Yasumura S, ed. Advances in in vivo body composition studies. New York: Plenum Press, 1990:327-432.
- Mazess RB, Bisek J, Trempe J, Pourchot S. Effects of tissue thickness on body composition measurement using dual-energy x-ray absorptiometry. Bone 1992;13:280(abst)
- Mazess RB, Collick B, Trempe J, Barden H, Hanson J. Performance evaluation of dual-energy x-ray bone densitometer. Calcified Tissue International 1989;44:228-232.
- McArdle WD, Katch FI, Katch VL. Exercise physiology: energy, nutrition, and human performance. 3rd ed. Philadelphia: Lea & Febiger, 1991.
- McArdle WD, Katch FI, Pechar GS. Comparison of continuous and discontinuous treadmill and bicycle tests for max VO2. Medicine and Science in Sports 1973;5:156-160.
- McCargar LJ, McBurney RF. Association between specific weight loss factors and resting metabolic rate in women with a history of dieting. Obesity Research 1996;4:47S(abst)
- McCargar LJ, Sale J, Crawford SM. Chronic dieting does not result in a sustained reduction in resting metabolic rate in overweight women. Journal of the American Dietetic Association 1996b;96:1175-1177.
- McCargar LJ, Taunton J, Birmingham L, Pare S, Simmons D. Metabolic and anthropometric changes in female weight cyclers and controls over a one year period. Journal of the American Dietetic Association 1993;93:1025-1030.
- Mifflin MD, St Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO. A new predictive equation for resting energy expenditure in healthy individuals. American Journal of Clinical Nutrition 1990;51:241-247.
- Milliken LA, Going SB, Lohman TG. Effects of variations in regional composition on soft tissue measurements by dual-energy x-ray absorptiometry. International Journal of Obesity and Related Metabolic Disorders 1996;20:677-682.
- Mitchell J. The physiological meaning of the maximal oxygen intake test. Journal of Clinical Investigation 1958;37:538
- Mole PA. Impact of energy intake and exercise on resting metabolic rate. Sports Medicine 1990;10:72-87.

- Morse WI, Soeldner JS. The composition of adipose tissue and the non-adipose body of obese and nonobese man. Metabolism 1963;12:99-107.
- Mortenson GM, Hoerr SL, Garner DM. Predictors of body satisfaction in college women. Journal of the American Dietetic Association 1993;93:1037-1040.
- Mossavar-Rahmani Y, Pelto GH, Ferris AM, Allen LH. Determinants of body size perceptions and dieting behavior in a multiethnic group of hospital staff women. Journal of the American Dietetic Association 1996;96:252-256.
- Mullen BJ, Krantzler NJ, Grivetti LE, Schutz HG, Meiselman HL. Validity of a food frequency questionnaire for the determination of individual food intake. American Journal of Clinical Nutrition 1984;39:136-43.
- National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. Diabetes 1979;28:1039-1057.
- National Institutes of Health. Consensus Development Panel on the Health Implications of Obesity. Annals of Internal Medicine 1995;103:1073-1077.
- National Institutes of Health. Technology Assessment Conference Panel. Methods of voluntary weight loss and control. Annals of Internal Medicine 1993;119:764-770.
- National Task Force on the Prevention and Treatment of Obesity. Weight cycling. Journal of the American Medical Association 1994;272:1196-1202.
- Nicklas BJ, Katzel LI, Ryan AS, Dennis KE, Goldberg AP. Gender differences in the response of plasma leptin concentrations to weight loss in obese older individuals. Obesity Research 1997;5:62-68.
- Noles SW, Cash TF, Winstead BA. Body image, physical attractiveness, and depression. Journal of Consulting & Clinical Psychology 1985;53:88-94.
- Norton AC. Portable equipment for gas exchange. In: Assessment of energy metabolism in health and disease. Columbus: Ross Laboratories, 1980:
- Oberrieder H, Walker R, Monroe D, Adeyanju M. Attitude of dietetics students and registered dietitians toward obesity. Journal of the American Dietetic Association 1995;95:914-916
- O'Brian JT, Bybee DE, Burman KE, Osburne RC, Ksiazek MR, Wartofsky L, Georges LP. Thyroid hormone homeostasis in states of relative caloric deprivation. Metabolism 1980;29:721
- O'Brien RM, Granner DK. PEPCK gene as a model of inhibitory effects of insulin on gene transcription. Diabetes Care 1990;13:327-329.
- O'Connell M, Robbins DC, Horton ES, Sims EAH, Danforth E. Changes in serum concentrations of triiodothyronine and reverse triiodothyronine during prolonged moderate exercise. Journal of Clinical and Endocrinological Metabolism 1978;49:242-245.
- O'Connor PJ, Petruzzello SJ, Kubitz KA, Robinson TL. Anxiety responses to maximal exercise testing. British Journal of Sports Medicine 1995;29:97-102.

- Ogden J. The measurement of restraint: Confounding success and failure? International Journal of Eating Disorders 1993;13:69-76.
- Omichinski L, Harrison KR. Reduction in dieting attitudes and practices after participation in a non-diet lifestyle program. Journal of the Canadian Dietetic Association 1995;56:81-85.
- O'Neil PM, Jarrell MP. Psychological aspects of obesity and very-low-calorie diets. American Journal of Clinical Nutrition 1992;56:185S-189S.
- Pate RR. Physical activity assessment in children and adolescents. Critical Reviews in Food Science and Nutrition 1993;33:321-326.
- Pelleymounter MA, Cullen MJ, Baker MB, Hecht R, Winters D, Boone T, Collins F. Effects of the obese gene product on body weight regulation in ob/ob mice. Science 1995;269:540-543.
- Peppler WW, Mazess RB. Total body bone meneral and lean body mass by dual-photon absorptiometry. Calcified Tissue International 1981;33:353-359.
- Phinney SD, LaGrange BM, O'Connell MO, Danforth E. Effects of aerobic exercise on energy expenditure and nitrogen balance during very low calorie dieting. Metabolism 1988;37:758-765.
- Pierson RN, Wang J, Heymsfield SB, Russell-Aulet, Mazariegos M, Tierney M, Smith R, Thornton JC, Kehayias J, Weber DA, Dilmanian FA. Measuring body fat: calibrating the rulers; Inter-method comparisons in 389 normal Caucasian subjects. American Journal of Physiology 1991;261:E103-E108.
- Pilkis SJ. Hepatic gluconeogenesis/glycolysis: regulation and structure/function relationships of substrate cycle enzymes. Annual Reviews of Nutrition 1990;11:465-515.
- Platte P, Wurmser H, Wade SE, Mercheril A, Pirke KM. Resting metabolic rate and diet-induced thermogenesis in restrained and unrestrained eaters. International Journal of Eating Disorders 1996;20:33-41.
- Polivy J, Herman CP. Breaking the diet habit. New York: Basic Books, 1983.
- Polivy J, Herman CP, Howard KI. Restraint Scale: Assessment of dieting. In: Hersen M, Bellack AS, eds. Dictionary of behavioural assessment techniques. New York: Pergamon, 1988:377-380.
- Pols MA, Peeters PHM, Bueno-de-Mesquita HB, Ocke MC, Wentik CA, Kemper HCG, Collette HJA. Validity and repeatability of a modified Baecke questionnaire on physical activity. International Journal of Epidemiology 1995;24:381-388.
- Pouliot MC, Despres JP, Lemieux S, Moorjani S, Bouchard C, Tremblay A, Nadeau A, Lupien PJ. Waist circumference and abdominal sagittal diameter: Best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. American Journal of Cardiology 1994;73:460-468.

- Powell KE, Thompson PD, Caspersen CJ, Kendrick JS. Physical activity and the incidence of coronary heart disease. Annual Review of Phublic Health 1987;8:253-287.
- Prentice AM, Jebb SA, Goldberg GR, Coward WA, Murgatroyd PR, Poppitt SD, Cole TJ. Effects of weight cycling on body composition. American Journal of Clinical Nutrition 1992;56:209S-216S.
- Pritchard JE, Nowson CA, Strauss BJ, Carlson JS, Kaymakci B, Wark JD. Evaluation of dual energy x-ray absorptiometry as a method of measurement of body fat. European Journal of Clinical Nutrition 1993;47:216-228.
- Pudel V, Metzdorff M, Oetting M. Zur Personlichkeit Adiposer in psychologischen Tests unter Berucksichtigung latent Fettsuchtiger. Zeitschrift für Psycholsomatische Medizin und Psycholanalyse 1975;21:345-361.
- Ravussin E, Bogardus C. A brief overview of human energy metabolism and its relationship to essential obesity. American Journal of Clinical Nutrition 1992;55:242S-245S.
- Ravussin E, Bogardus C. Relationship of genetics, age, and physical fitness to daily energy expenditure and fuel utilization. American Journal of Clinical Nutrition 1989;49:968-975.
- Ravussin E, Burnand B, Schutz Y, Jequier E. Twenty-four-hour energy expenditure and resting metabolic rate in obese, moderately obese, and control subjects. American Journal of Clinical Nutrition 1982;35:566-573.
- Rebuffe-Scrive M, Enk L, Crona N, Lonnroth P, Abrahamsson L, Smith U, Bjorntorp P. Fat cell metabolism in different regions in women. Effect of menstrual cycle, pregnancy, and lactation. Journal of Clinical Investigation 1985;75:1973-1976.
- Rebuffe-Scrive M, Hendler R, Bracero N, Cummings N, McCarthy S, Rodin J. Biobehavioural effects of weight cycling. International Journal of Obesity 1994;18:651-658.
- Rice T, Tremblay A, Deriaz O, Perusse L, Rao DC, Bouchard C. A major gene for resting metabolic rate unassociated with body composition: Results from the Quebec Family Study. Obesity Research 1996;4:441-449.
- Robbins TW, Fray PJ. Stress-induced eating. Fact, fiction, or misuderstanding. Appetite 1980;1:103-133.
- Rodin J. Has the distinction between internal versus external control of feeding out-lived its usefulness? In: Bray GA, ed. Recent advances in obesity research:II, Proceedings of the 2nd International Congress on Obesity. London: John Libbey, 1978:75-85.
- Rodin J, Silberstein L, Striegel-Moore R. Women and weight: A normative discontent. Nebraska Symposium on Motivation. 1984;26:25-42.

- Rodnick KJ, Slot JW, Studelska DR, et al. Immunocytochemical and biochemical studies of GLUT 4 in rat skeletal muscle. Journal of Biological Chemistry 1992;267:6278-6285.
- Rosen JC. Improving body image in obesity. In JK Thompson (Ed). Body image, eating disorders, and obesity. Washington, DC: American Psychological Association, 1996.
- Rosen JC, Gross J, Vara L. Psychological adjustment of adolescents attempting to lose or gain weight. Journal of Consulting and Clinical Psychology 1987;55:742-747.
- Ross R, Shaw K, Martel Y, De Guise J, Avruch L. Adipose tissue distribution measured by magnetic resonance imaging in obese women. American Journal of Clinical Nutrition 1993;57:470-475.
- Russel-Briefel R, Caggiula AW, Kuller LH. A comparison of three dietary methods for estimating vitamin-A intake. American Journal of Epidemiology 1985;122:628-638.
- Saladin R, De Vos P, Guerre-Millo M, Leturque A, Girard J, Staels B, Auwerx J. Transient increase in obese gene expression after food intake of insluin administration. Nature 1995;377:527-529.
- Sandler MP, Robinson RP, Rabin D, Lacy WW, Abumrad NN. The effect of thyroid hormones on gluconeogenesis and forearm metabolism in man. Journal of Clinical Endocrinology and Metabolism 1983;56:479-485.
- Sandri SC. On dancers and diet. International Journal of Sport Nutrition 1993;3:334-342.
- Saris WHM, Snel P, Baecke J, van Waesberghe F, Binkhorst RA. A portable miniature solid-state heart rate recorder for monitoring daily physical activity. Biotelemetry 1977;4:131-140.
- Sawaya AL, Tucker K, Tsay R, Willett W, Saltzman E, Dallal GE, Roberts SB. Evaluation of four methods for determining energy intake in young and older women: Comparison with doubly labeled water measurements of total energy expenditure. American Journal of Clinical Nutrition 1996;63:491-499.
- Sawka MN. Physiology of upper body exercise. Exercise Sport Science Review 1986;14:175
- Schachter S, Goldman R, Gordon A. Effects of fear, food deprivation, and obesity on eating. Journal of Personality and Social Psychology 1968;10:91-97.
- Schoeller DA. Measurement of energy expenditure in free-living humans by using doubly labeled water. Journal of Nutrition 1988;118:1278-1289.
- Schutz Y, Deurenberg P. Energy metabolism: Overview of recent methods used in human studies. Annals of Nutrition Metabolism 1996;40:183-193.

- Schutz Y, Jequier E. Energy needs: assessment and requirements. In: Shils ME. Olson JA, Shike M, eds. Modern nutrition in health and disease. 8th ed. Malvern, PA: Lea & Febiger, 1994:101-111.
- Schutz Y. Terminology, factors, and constants in studies on energy metabolism of humans. In: van Es AJH, ed. Human energy metabolism: physical activity and energy expenditure measurements in epidemiological research based upon direct and indirect calorimetry. Wageningen: Euro-Nut, 1985:153-168.
- Schutz Y. The basis of direct and indirect calorimetry and their potentials. Diabetes Metabolism Reviews 1995;11:383-408.
- Schwartz MW, Baskin DG, Bukowski TR, Kuijper JL, Foster D, Lasser G, Prunkard DE, Porte D, Woods SC, Seeley RJ, Weigle DS. Specificity of leptin action on elevated blood glucose levels and hypothalamic neuropeptide Y gene expression in ob/ob mice. Diabetes 1996;45:531-535.
- Seale J, Miles C, Bodwell CE. Journal Applied Physiology 1989;66:644-653.
- SensorMedics Corporation. Reference manual for Vmax 29N series indirect calorimetry testing. Yorba Linda, CA 1995;15.1-15.15.
- Serdula MK, Collins ME, Williamson DF, Anda RF, Pamuk E, Byers TE. Weight control practices of U.S. adolescents and adults. Annals of Internal Medicine 1993;119:667-671.
- Severson RK, Nomura AMY, Grove JS, Stemmermann GN. A prospective analysis of physical activity and cancer. American Journal of Epidemiology 1989;130:522-529.
- Silverstein B, Perdue L, Peterson B. The role of the mass media in promoting a thin standard of bodily attractiveness for women. Sex Roles 1986;14:519-532.
- Sims EAH, Danforth E. Expenditure and storage of energy in man. Journal of Clinical Investigation 1987;79:1019-1025.
- Sinha MK, Ohannesian JP, Heiman ML, Kriauciuans A, Stephens TW, Magosin S, Marco C, Caro JF. Nocturnal rise of leptin in lean, obese, and non-insulin dependent diabetes mellitus subjects. Journal of Clinical Investigation 1996;97:1344-1347.
- Slade PD. What is body image? Behaviour Research & Therapy 1994;32:497-502.
- Snead DB, Birge SJ, Kohrt WM. Age-related differences in body composition by hydrodensitormery and dual-energy x-ray absorptiometry. Journal of Applied Physiology 1993;74:770-775.
- Snow-Harter C, Marcus R. Exercise, bone mineral density, and osteoporosis. In: Holloszy JO, ed. Exercise and sport sciences reviews. Baltimore: Williams & Wilkins, 1991:351-388.
- Steele R. Influences of glucose loading and injected insulin on hepatic glucose output. Annals of the New York Academy of Science 1959;82:420-430.

- Steen SN, Oppliger RA, Brownell KD. Metabolic effects of repeated weight loss and regain in adolescent wrestlers. Journal of the American Medical Association 1988;260:47-50.
- Stephens TW, Basinski M, Bristow PK, Bue-Valleskey JM, Burgett SG, Craft L, Hale J, Hoffmann J, Hsiung HM, Kriauciunas A, MacKellar W, Rosteck PR, Schoner B, Smith D, Tinsley FC, Zhang X, Helman M.. The role of neuropeptide Y in the anti-obesity action of the obese gene product. Nature 1995;377:530-532.
- Stice E, Shaw HE. Adverse effects of the media-portrayed thin ideal on women and linkages to bulimic symptomatology. Journal of Social and Clinical Pshychology 1994;13:288-308.
- Striegel-Moore RH, McAvay G, Rodin J. Psychological and behavioural correlates of feeling fat in women. International Journal of Eating Disorders 1986;5:935-947.
- Strubbe JH. Regulation of food intake. In: Westerterp-Plantenga MS, Fredrix EWHM, Steffens AB, eds. Food intake and energy expenditure. Boca Raton: CRC Press, 1994:141-154.
- Stunkard AJ, Messick S. The Eating Inventory. San Antonio, TX: Psychological Corporation, 1988.
- Stunkard AJ, Messick S. The three factor eating questionnaire to measure dietary restraint, disinhibition, and hunger. Journal of Psychosomatic Research 1985;29:71-81.
- Stunkard AJ. Restrained eating: What it is and a new scale to measure it. In: Cioffi LAa, James WPT, van Itallie TB, eds. The body weight regulatory system: normal and disturbed mechanisms. New York: Raven, 1981:243-251.
- Takahashi M, Funahashi T, Shimomura I, Miyaoka K, Matsuzawa Y. Plasma leptin levels and body fat distribution. Hormone and Metabolic Research 1996;28:751-752.
- Taurog A. Thyroid iodine metabolism. In: Ingbar SH, Braverman LE, eds. Werner's the thyroid. Philadelphia: Lippincott, 1986:53-97.
- Temple R, Clark P, Nagi D, Schneider A, Yudkin J, Hales C. Radioimmunoassay may overestimate insulin in non-insulin dependent diabetics. Clinical Endocrinology 1990;32:689-693.
- Tepper BJ, Trail AC, Shaffer SE. Diet and physical activity in restrained eaters. Appetite 1996;27:51-64.
- Thompson JK, Heinberg LJ, Tantleff S. The Physical Appearance Comparison Scale (PACS). The Behaviour Therapist 1991;14:174
- Thompson JL, Manore MM, Thomas JR. Effects of diet and diet-plus-exercise programs on resting metabolic rate: a meta-analysis. International Journal of Sport Nutrition 1996;6:41-61.

- Tuschl RJ, Laessle RG, Platte P, Pirke KM. Differences in food-choice frequencies between restrained and unrestrained eaters. Appetite 1990a;14:9-13.
- Tuschl RJ, Platte P, Laessle RG, Stichler W, Pirke KM. Energy expenditure and everyday eating behaviour in healthy young women. American Journal of Clinical Nutrition 1990b;52:81-86.
- van Dale D, Saris WH. Repetitive weight loss and weight regain: Effects on weight reduction, resting metabolic rate, and lipolytic activity before and after exercise and/or diet treatment. American Journal of Clinical Nutrition 1989;49:409-416.
- van der Heyden JTM, Docter R, van Toor H, Wilson JHP, Hennemann G, Krenning EP. Effects of caloric deprivation on thyroid hormone tissue uptake and generation of low-T₃ syndrome. American Journal of Physiology 1986;251:E156-E163.
- van Staveren WA, West CE, Hoffmans MDAF, Bos P, Kardinaal AFM, van Poppel GAFC, Schipper HJ-A, Hautvast JGAJ, Hayes RB. Comparison of contemporaneous and retrospective estimates of food consumption made by a dietary history method. American Journal of Edpidemiology 1986;123:884-893.
- van Strien T, Frijters JER, Bergers GPA, Defares PB. The Dutch eating behaviour questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behaviour. International Journal of Eating Disorders 1986;5:295-315.
- Wadden TA, Bartlett S, Letizia KA, Foster GD, Stunkard AJ, Conill A. Relationship of dieting history to resting metabolic rate, body composition, eating behavior, and subsequent weight loss. American Journal of Clinical Nutrition 1991;56:203S-208S.
- Wadden TA, Foster GD, Stunkard AJ, Conill AM. Effects of weight cycling on the resting energy expenditure and body composition of obese women. International Journal of Eating Disorders 1996;19:5-12.
- Wadden TA, Vogt RA, Andersen RE, Bartlett SJ, Foster GD, Kuehnel RH, Wilk J, Weinstock R, Buckenmeyer P, Berkowitz RI, Steen SN. Exercise in the treatment of obesity: Effects of four interventions on body composition, resting energy expenditure, appetite, and mood. Journal of Consulting and Clinical Psychology 1997;65:269-277.
- Wang J, Pierson RN. Disparate hydration of adipose and lean tissue require a new model for body water distribution in man. Journal of Nutrition 1976;106:1687-1693.
- Wardle J. Eating style: A validation study of the Dutch Eating Behaviour Questionnaire in normal subjects and women with eating disorders. Journal of Psychosomatic Research 1987;31:161-169.
- Webb P. 24-hour energy expenditure and the menstrual cycle. American Journal

- Wegner M, Snow-Harter C, Wilcox A, Guerra A, White K. The accuracy of dual energy x-ray absorptiometry in determining percent body fat: comparison with a multicomponent model. Medicine and Science in Sports and Exercise 1993;26:S202.
- Weigle DS, Duell PB, Connor WE, Steiner RA, Soules MR, Kuijper JL. Effect of fasting, refeeding, and dietary fat restriction on plasma leptin levels. Journal of Clinical Endocrinology and Metabolism 1997;82:561-565.
- Weir JBV. New methods for calculating metabolic rate with special reference to protein metabolism. Journal of Physiology 1949;109:1-9.
- Welch HG, Pedersen PK. Measurement of metabolic rate in hyperoxia. Journal of Applied Physiology 1981;51:725-731.
- Welle SL, Amatruda JM, Forbes GB, Lockwood DH. Resting metabolic rates of obese women after rapid weight loss. Journal of Clinical Endocrinology and Metabolism 1984;59:41-44.
- Welle SL, Schwartz RG, Statt M. Reduced metabolic rate during beta-adrenergic blockage in humans. Metabolism 1991;40:619-622.
- Westenskow DR, Schipke CA, Raymond JL. Calculation of metabolic expenditure and substrate utilization from gas exchange measurements. Journal of Parenteral and Enteral Nutrition 1988;12:20
- Westerterp KR. Energy from food. In: Westerterp-Plantenga MS, Fredrix EWHM, Steffens AB, eds. Food intake and energy expenditure. Boca Raton: CRC Press, 1994:225-234.
- Weststrate JA. Resting metabolic rate and diet-induced thermogenesis: a methodological reappraisal. American Journal of Clinical Nutrition 1993;58:592-601.
- Whitney EN, Cataldo CB, Rolfes SR. Understanding normal and clinical nutrition. 3rd ed. St. Paul, MN: West Publishing, 1991.
- Wilfley DE, Rodin J. Cultural influences on eating disorders. In: Brownell KD, Fairburn CG, eds. Eating disorders and obesity: A comprehensive handbook. New York: Guilford Press, 1995:78-82.
- Willett WC, Sampson L, Stampfer MJ, Rosner B, Bain C, Witschi J, Hennekens CH, Speizer FE. Reproducibility and validity of a semiquantitative food frequency questionnaire. American Journal of Epidemiology 1985;122:51-65.
- Wing RR, Matthews KA, Kuller LH, Smith D, Becker D, Plantinga PL, Meilahn EN. Environmental and familial contributions to insulin levels and change in insulin levels in middle-aged women. Journal of the American Medical Association 1992;268:1890-1895.
- Wing RR, Shiffman S, Drapkin RG, Grilo CM, McDermott M. Moderate versus restrictive diets: Implications for relapse. Behavior Therapy 1995;26:5-24.

- Wiseman CV, Gray JJ, Mosimann JE, Ahrens AH. Cultural expectations of thinness in women: An update. International Journal of Eating Disorders 1993;11:85-89.
- Wolever TMS, Bolognesi C. Source and amount of carbohydrate affect postprandial glucose and insulin in normal subjects. Journal of Nutrition 1996;126:2798-2806.
- Wolever TMS, Jenkins DJA. The use of the glycemic index in predicting the blood glucose response to mixed meals. American Journal of Clinical Nutrition 1986;43:167-172.
- Wolper C, Heshka S, Heymsfield SB. Measuring food intake. In: Allison DB, ed. Handbook of assessment omethods for eating behaviours and weight-related problems: Measures, theory, and research. Thousand Oaks: Sage Publications, 1995:215-240.
- World Health Organization: Energy and protein requirements. Report of a Joint FAO/WHO/UNU Expert Consultation. Technical Report Series 724. Geneva, 1985.
- Yamada T, Shirota T, Aizawa T, Takasu N. Blood glucose, serum thyroid hormones, insulin, C-peptide, and C-peptide/insulin ratio in hyperthyroid patients. Hormone and Metabolic Research 1991;23:504-505.
- Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. Positional cloning of the mouse obese gene and its human homologue. Nature 1994;372:425-432.
- Zulkifli Sn, Yu SM. The food frequency method for dietary assessment. Journal of the American Dietetic Association 1992;92:681-685.

APPENDIX A - SAMPLE SIZE CALCULATION

Estimation of sample size for independent groups was conducted using LBM (continuous variable) as the end-point of interest. Lean body mass is the best single predictor of energy expenditure (Jensen, et al., 1988), REE is linearly related to LBM, and LBM explains the largest variability in REE (Nelson, et al., 1992).

The appropriate calculations for determination of sample size are as follows:

$$n = \frac{(SD_1^2 + SD_2^2)(Z_{1-\beta} + Z_{1-\alpha/2})^2}{(x_2 - x_1)^2}$$

Where values $Z_{1-\beta}$ and $Z_{1-\alpha/2}$ are defined in Table 20.2 (Cheney, Boushey, 1992). The LBM SD₁ and SD₂ for normal weight and overweight women are 9.4 kg and 5.6 kg, respectively. The difference of interest $(x_2 - x_1)$ is 9.1 kg based on research comparing LBM between normal weight and overweight women (Svendsen, Hassager, Christiansen, 1995; Tataranni, Ravussin, 1995). A p-value of 0.05 and β -value of 0.10 were used.

According to the above formula the sample size is:

$$n = \frac{(SD_1^2 + SD_2^2) (Z_{1-\beta} + Z_{1-\alpha/2})^2}{(x_2 - x_1)^2}$$

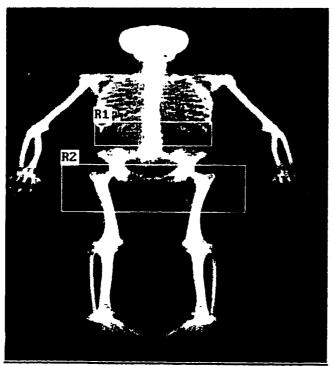
$$n = \frac{(9.4^2 + 5.6^2) (1.28 + 1.96)^2}{(52.6-43.5)^2}$$

$$n = (119.72) (10.4976)$$
82.81

$$n = 15.177$$

APPENDIX B - PRE-DETERMINED DXA REGIONS (R1 AND R2)

MIC COLLEGE PLAZA BONE DENSITY



oMay 4 11:40 1997 [327 x 150] Hologic QDR-4500A (S/N 45026) Whole Body V8.10a:3

204099723 Wed Apr 9 19:58 1997 Name: Comment: I.D.: Sex: F S.S.#: Ethnic: C ZIPCode: Height: CM Operator: NG Weight: kg BirthDate: 05/01/48 48 Age: Physician: MCCARGAR Image not for diagnostic use

C.F.	1.012	0.998	1.000	
Region Area (cm2)		BMC (grams)	BMD (gms/cm2)	
		~~~~		
R1	55.93	67.88	1.214	
R2	196.16	273.05	1.392	
NETAUG	252.10	340.93	1.352	



# MIC COLLEGE PLAZA BONE DENSITY

Hologic QDR-4500A (S/N 45026) Whole Body V8.10a:3 oMay 4 11:40 1997

TBAR1010 F.S. 68.00% 0(10.00)%

204099723 Wed Apr 9 19:58 1997 Name: Comment: I.D.: Sex: F S.S.#: Ethnic: С ZIPCode: Height: CM

Operator: NG Weight: kg BirthDate: 05/01/48 Age: 48 Physician: MCCARGAR

Region	BMC	Fat	Lean	Lean+BMC	Total	% Fat
	(grams)	(grams)	(grams)	(grams)	(grams)	(%)
R1	67.9	914.4	4626.7	4694.6	5608.9	16.3
R2	273.0	4820.1	9092.1	9365.2	14185.3	34.0
NETAUG	340.9	5734.4	13718.8	14059.8	19794.2	29.0

## APPENDIX C - BAECKE QUESTIONNAIRE

1. What is your main occupation?

Please check the box that most accurately answers the following questions:

		NEVER	SELDOM	SOMETIMES	OFTEN	ALWAYS
2.	At work I sit					
3.	At work I stand					
4.	At work I walk					
5.	At work I lift heavy loads					
6.	At work I sweat	$\mathbf{L}_{-}$				
7.	After work I am tired					

Circle the most appropriate response.

8. In comparison with others of my own age, I think my work is physically...

much lig	hter	lighter	as	heavy		heavie	r	much heavier
9. Do you	play a sport?		Yes	No				
If yes:								
Which sport do you play most frequently?								
•	How many h	ours per week?	⁷ <1	1-2	2-3	3-4	>4	
•	How many m	nonths per year	?	<1	1-3	4-6	7-9	>9
If you play ◆	a second sport Which sport							
•	How many h	ours per week?	•	<1	1-2	2-3	3-4	>4
•	How many m	nonths per year	?	<1	1-3	4-6	7-9	>9

Circle the most appropriate response.

10. In comparison with others my own age, I think my physical activity during leisure time is...

much less less the same more much more

Please check the box that most accurately answers the following questions:

	NEVER	SELDOM	SOMETIMES	OFTEN	ALWAYS
11. During leisure time, I sweat					
12. During leisure time, I play					
sport(s)					
13. During leisure time, I watch TV					
14. During leisure time, I walk					1
15. During leisure time, I cycle		1			
16. During leisure time, I work in the					
garden	į				
17. During leisure time, I do do-it-					
yourself activities		<u> </u>			

	How many minut shopping?	ninutes per day do you walk and/or cycle to and from work, school, and									
		<5	5-15	15-30	30-45	>45					
19.	How many hours	do you sleep	o (on average)?								
		<5	6	7	8	>9					

## BAECKE QUESTIONNAIRE, CONTINUED

## Scoring for question #9:

Question 9 = (Intensity₁xTime₁xProportion₁)+ (Intensity₂xTime₂xProportion₂)  
= 0 (given to participants who do not play a sport) (Likert score = 1)  
= 0.01 - < 4 (Likert score = 2)  
= 4 - < 8 (Likert score = 3)  
= 8 - < 12 (Likert score = 4)  
= 
$$\geq$$
 12 (Likert score = 5)

Calculation of the indices of physical activity:

WORK INDEX = 
$$(1 + 2 + 3 + 4 + 5 + 6 + 7 + 8) / 8$$

SPORTS INDEX = (9 + 10 + 11 + 12) / 4

LEISURE TIME INDEX = (13 + 14 + 15 + 16 + 17 + 18 + 19) / 7

TOTAL ACTIVITY INDEX = work index + sports index + leisure time index

## APPENDIX D - NORMAL FASTING LABORATORY VALUES

Variable	Normal	All Participants	High	Low REE	
	Ranges		REE		
Glucose ^a (mmol/L)	4.33 – 6.38	5.57 ± 0.68	5.65 ± 0.87	5.48 ± 0.45	
Insulin ^b (nmol/L)	0.04 - 0.14	0.09 ± 0.05	0.10 ± 0.05	$0.07 \pm 0.04$	
Leptin ^c (ng/mL)	0 – 16.8	27.03 ± 12.87	25.17 ± 10.87	28.90 ± 14.74	
T₄ ^d (nmol/L)	58 -161	104.59 ± 24.12	115.28 ± 27.53	93.89 ± 14.20	
T ₃ ^d (nmol/L)	1.32 – 2.87	2.25 ± 0.53	2.32 ± 0.47	2.18 ± 0.60	
r-T ₃ ^e (nmol/L)	0.14 - 0.54	0.20 ± 0.05	0.22 ± 0.05	0.18 ± 0.01	

From Sigma Diagnostics, St. Louis, MO.
 From Dynacare Kasper Medical Laboratories, Edmonton, AB.
 From Considine RV, et al., NEJM. 1996;334:292-295.
 From Diagnostic Products Corporation, Los Angeles, CA.
 From BioData Diagnostics, Rome, Italy.

#### APPENDIX E - FOOD FREQUENCY QUESTIONNAIRE

#### Food Intake

This part of the survey is designed to determine your usual food intake over the last 6 months. Complete the chart on the next pages including foods and beverages consumed both at home and away from home. Please read the items carefully and take your time filling in the chart.

For every food mark Yes or No. If Yes, indicate the number of times and mark day, week or month. Mark one of the serving sizes.

Here are some examples showing how to complete the chart.

"Sarah drinks 1% milk once a day - about 1 1/2 cups each time"
This is how she would show that on the chart

#### Examples

		Do you have this food or beverage at least once a month?  About how many times per day or week or month?		About how much do you have each time?			
2.	1% milk and beverages made with it	Yes → O No	1	© Day O Week O Month	O 1/2 cup	O I cup	More than 1 cup

"Sarah eats whole wheat bread in a sandwich for lunch about five times a week, two slices each time." She would record her bread this way.

"Sarah only eats roast beef or steak every 3 or 4 months."

She would show that on the food chart like this.

		Do you have this food or beverage at least once a month?	ut how many tim day or week or th?		nuch do you bav	ve each time?
Whi	te or Chocolate Milk	To Drink				
i.	Skim milk and beverage made with it	ges O Yes → O No	 O Day O Week O Month	O 1/2 cup	O 1 cup	O more than I cup
2.	1% milk and beverages made with it	s O Y⇔ → O No	 O Day O Week O Month	O 1/2 cup	O I cup	O more than I cup
3.	2% milk and beverages made with it	o Yes → O No	 O Day O Week O Month	O I/2 cup	O 1 cup	O more than I cup
4.	Whole milk and beverages made with it	O Yes → O No	 O Day O Week O Month	O 1/2 cup	O 1 cup	O more than 1 cup
5.	Milkshakes	O Y⇔ → O No	 O Day O Week O Month	O 1/2 cup	O 1 cup	O more than 1 cup
Chees	se, Yogurt and Eggs					
6.	Hard cheese such as cheddar	O Yes → O No	 O Day O Wæk O Month	O I inch cube	O More than I inch cube	O Less than 1 inch cube
7.	Skim milk cheese such as low fat mozzarella	O Yes → O No	 O Day O Week O Month	O I inch cube	O More than 1 inch cube	O Less than I inch cube
8.	Processed cheese slices (including on sandwiches and hamburgers)	O Yes → O No	 O Day O Week O Month	O I slice	O 2 slices	more than 2 slices
9.	Cottage cheese	O Yes → O No	 O Day O Week O Month	O 1/2 Cup	O More than 1/2 cup	O Less than 1/2 cup
10.	Low fat cottage cheese	O Yes → O No	 O Day O Week O Month	O 1/2 Cup	O More than 1/2 cup	O Less than 1/2 cup

		Do you have this food or beverage at least once a month?	t how many tie ay or week or h?		uch do you bave e	esch time?
11.	Any other cheese and cheese spreads	O Y⇔ → O No	 O Day O Week O Month	O 1 in. cube /1 Tbsp.		O Less than cube/Tbsp.
12.	Yogurt	O Y¤ → O No	 O Day O Week O Month	O small carton	O large carton	O 1/2 cup
13.	Low fat yogurt	O Y⇔ → O No	 O Day O Week O Month	O small carton	O large carton	O 1/2 cup
14.	Eggs	O Yes → O No	 O Day O Week O Month	egg I O	O 2 eggs	O 3 or more eggs
Break	dast Cereals					
15.	Whole grain hot cereals (rolled oats, red river)		 O Day O Week O Month	O 3/4 cup	O more than 3/4 cup	O less than 3/4 cup
16.	Instant hot cereals	O Yes → O No	 O Day O Week O Month	O 3/4 cup	O more than 3/4 cup	O less than 3/4 cup
17.	Cold cereals, no sugar (Shredded Wheat, Corn Flakes, Rice Krispies, Cheerios)	O Yes → O No	 O Day O Week O Month	O 3/4 cup	O more than 3/4 cup	O less than 3/4 cup
18.	Bran type cold cereals (Bran Flakes, All Bran Raisin Bran, etc.)		 O Day O Week O Month	O 3/4 cup	O more than 3/4 cup	O less than 3/4 cup
19.	Sweetened cold cereals (Frosted Flakes, Sugar Smacks)		 O Day O Week O Month	O 3/4 cup	O more than 3/4 cup	O less than 3/4 cup
20.	Granola	O Y⇔ → O No	 O Day O Week O Month	O 3/4 cup	O more than 3/4 cup	O less than 3/4 cup

21.	If you eat cereal						
a) b) c)	Do you usually add su Do you usually add an Which one of the follow	ificial sweetener?		OYes OYes ten on your c	O No O No creal?		
	O Cream/Half & Half	O Whole milk	O 29	6 milk O	1% milk	O Skim milk	
ď)	How much milk do you	add to your cere	<b>a</b> l?				
	O O 1/2 cup 1 cup	O more than 1 cup					
		Do you have this food or beverage at least once a month?		bow many tic ty or week or ?		sch do you have	each time?
Bread	ls, Rolls and Mussins						
22.	Wholewheat or light rye bread and rolls	: O Yes → O No		O Day O Week O Month	O 1-2 sliæs	O 3-4 sliœs	O 5 or more slices
23.	Dark rye, pumpernickel fibre-enriched bread and rolls	O Yes → O No		O Day O Week O Month	O 1-2 slices	O 3-4 slices	O 5 or more slices
24.	White, Italian, French egg, raisin bread and rolls, bagels, hotdog or hamburger bu	O Yes → O No		O Day O Week O Month	O 1-2 slice	O 3-4 slices	O 5 or more slices
25.	Bran or com muffins	O Y≈ → O No		O Day O Week O Month	O 1 muffin	Q 2 muffins	O 3 or more muffins
26.	Any other muffins, such as blueberry, plain chocolate chip	O Yes → O No		O Day O Week O Month	O I muffin	Q 2 muffins	O 3 or more muffins
27.	Pancakes or waffles	O Y⇔ → O No		O Day O Week O Month	o i	2	Q 3 or more

28.	If you eat bread, do	you add	Alwa	ıys U	sually	Sometimes	Rarely/Never
	Butter, margarine or ca	eam cheese	0	•	0	0	0
	Diet margarine or creat	m cheese	0	ı	0	0	0
	Mayonnaise or salad di		0		0	0	0
	Low calorie mayonnais	_	0		0	0	0
	Peanut butter	Peanut butter			0	0	•
	Jelly, jam, honey or other sweet spread		0		0	0	0
	If you eat muffins, do you add		Alwa	ys U:	maily	Sometimes	Rarely/Never
	Butter, margarine or cr	eam cheese	0		0	0	0
	Diet margarine or crear		0		0	0	0
	Mayonnaise or salad dr		0		0	0	0
	Low calorie mayonnaise	_	0		0	0	0
	Peanut butter	_	0		0	0	0
	Jelly, jam, honey or other sweet spread		0		0	0	0
Meat	t, Poultry, Fish and A	Do you have this food or beverage at least once a month?		how many ti y or week or ?		uch do you hav	e each time?
29.	Beef and steak, roasted or stewed	O Y≈ → O No		O Day O Week O Mouth	O 4 ounces	More than 4 ounces	O Less than 4 ounces
30.	Pork and pork chops, roasted or stewed	O Yes → O No		O Day O Week O Month	O 4 ounces	O More than 4 ounces	O Less than 4 ounces
31.	Fried or breaded beef, steak, pork pork and pork chops	O Yes → O No		O Day O Week O Month	O 4 ounces	O More than 4 ounces	O Less than 4 ounces
32.	Liver, any type	O Y⇔ → O No		O Day O Week O Month	O 4 ounces	O More than 4 ounces	O Less than 4 ounces
33.	Chicken, turkey or other poultry, roasted, stewed or barbecued	O Y⇔ → O No		O Day O Week O Month	O 1-2 slices	O 3-4 slices	O 5 or more slices

		Do you have this food or beverage at least once a month?		t how many t ay or week o h?	r	nes About how much do you have each time?			
34.	Fried chicken, nuggets chicken sandwiches	, O Y⇔ → O No		O Day O Week O Month	O 2 piece 6 nugge 1 s'wich	ts 9 nuggets	More than 4 pieces / 9 nuggets		
35.	Fish, canned, fresh, frozen (ex. tuna salmon, sushi)	O Yes → O No		O Day O Week O Month	O 4 ounce	O More than 4 ounces	O Less than 4 ounces		
36.	Fried fish, fried fish sandwiches	O Yes → O No		O Day O Week O Month	O 2 pieces 1 s'wich	•	o more than 9 nuggets		
37.	Hamburgers and cheeseburgers	O Yes → O No		O Day O Week O Month	О 4 очл <i>с</i> е	os more than 4 ounces	O less than 4 ounces		
38.	Wieners, hot dogs	O Yes → O No		O Day O Week O Month	O r <del>e</del> gular	O large/2 r <del>eg</del> ular	O r more than l large/2 reg.		
39.	bacon	O Yes → O No		O Day O Week O Month	O 1-2 slices	O 3-4 slices	O 5 or more slices		
40.	Sausages	O Y⇔ → O No		O Day O Week O Month	O 1-2 links	O 3-4 links	O 1-2 large sausages		
41.	Coldcuts, luncheon meats (bologna, salami, chicken loaf or ham)	O Y⇔ → O No		O Day O Week O Month	O 1-2 slices	O 3-4 slices	O 5 or more slices		
42.	Tofu, soy bean curd	O Yes → O No		O Day O Week O Month	O I/2 cup	O more than 1/2 cup	O less than 1/2 cup		
43.			Al:	ways	Usually	Sometimes I	Rarely/Never		
a) If you eat meat or chicken, do you add gravy?				0	0	0	0		
b) If you eat meat, do you eat the fat?				0	0	0	0		
c) If you eat chicken do you eat the skin?				0	0	0	0		
	ou eat fish, do you have to onnaise with it?	irtar sauce or		0	0	0	0		

		Do you have this food or beverage at least once a month?	it how many tin lay or week or h?	nes About how much do you have each time?			
Mix	ed meat, fish or chicke	n dishes					
44.	Meat and chicken pies	O Yes → O No	 O Day O Week O Month	O 1-2 slices	O 3-4 slices	O 5 or more slices	
45.	Any other mixed dishes made with ground mean chicken and fish		 O Day O Week O Month	O I cup	O more than I cup	O less than I cup	
46.	Spaghetti, lasagna, other pasta with meat-tomato sauce	O Yes → O No	 O Day O Week O Month	O 1 cup	O more than 1 cup	O less than 1 cup	
47.	Macaroni and cheese, other pasta dishes with cheese	O Yes → O No	 O Day O Week O Month	O I cup	O more than I cup	O less than 1 cup	
48.	Pizza	O Yes → O No	 O Day O Week O Month	O 1-2 slices	O 3-4 slices	O 5 or more slices	
49.	Any other pasta or noodles	O Yes → O No	 O Day O Week O Month	O 1 cup	O more than I cup	O less than I cup	
50.	Rice, any type	O Yes → O No	 O Day O Week O Month	O l cup	O more than I cup	O less than I cup	
Soups	5						
51.		O Yes → O No	 O Day O Week O Month	O I cup	O more than 2 cups	O less than 2 cups	
52.	• • •	O Yes → O No	 O Day O Week O Month	O I cup	O more than 2 cups	O less than 2 cups	

		Do you have this food or beverage at least once a month?		bow many tin iy or week or i?	nes  About how much do you have each time?			
Vegetables								
53.	Broccoli	O Y⇔ → O No		O Day O Week O Month	O 1/2 cup	O more than 1/2 cup	O less than 1/2 cup	
54.	Carrots	O Yes → O No		O Day O Week O Month	O 1/2 cup	O more than 1/2 cup	O less than 1/2 cup	
55.	Com	O Yes → O No		O Day O Week O Month	O 1/2 cup small cob	o more than 1/2 cup	O less than 1/2 cup	
56.	Green Peas	O Yes → O No		O Day O Week O Month	O 1/2 cup	o more than 1/2 cup	O less than 1/2 cup	
57.	Greens (spinach, kale, bok choy, leeks)	O Yes → O No		O Day O Week O Month	O 1/2 cup	o more than 1/2 cup	O less than 1/2 cup	
58.	Green beans, string beans, yellow beans	O Yes → O No		O Day O Week O Month	O 1/2 cup	O more than 1/2 cup	O less than 1/2 cup	
59.	Any other beans, peas lentils (lima beans, navy, baked, pork and beans, kidney beans)	O Yes → O No		O Day O Week O Month	O 1/2 cup	O more than 1/2 cup	O less than 1/2 cup	
60.	Potatoes, baked, salad, boiled	O Yes → O No		O Day O Week O Month	O 1 cup	O more than I cup	O less than I cup	
61.	French fries, home fries, pan fried potatoes hash browns	OY⇔ → O No		O Day O Week O Month	O 1/2 cup	O more than 1/2 cup	O less than 1/2 cup	
62.	Squash, all types	O Yes → O No		O Day O Week O Month	O 1/2 cup	O more than 1/2 cup	O less than 1/2 cup	
63.	Salad - combination lettuce and tomato	O Y⇔ → O No		O Day O Week O Month	O I cup	O more than I cup	O less than 1 cup	

		Do you have this food or beverage at least once a month?		t how many ti ay or week or h?	7	out how mu	ch do you baw	e each time?
64.	Any other salads such as coleslaw, carrot, bean, spinach	O Y≈ → O No		O Day O Week O Month		O 1 cup	o more than	O less than I cup
65.	Any other vegetables such as cabbage, Brussels sprouts	O Yes → O No		O Day O Week O Month		O 1/2 cup	O more than 1/2 cup	O less than 1/2 cup
66. a) If y	ou eat potatoes or rice do	you add	Alway	s Us	ually	So	netimes	Rarely/Never
• butt	er, margarine, gravy or sou	cream?	0	•	0		0	0
<ul> <li>diet margarine, defatted gravy or diet sour cream?</li> </ul>			0	(	0		0	0
b) If <b>y</b>	ou eat vegetables, do you	ıdd						
• butte	er, margarine, cheese or oth	er sauces?	0	(	0		0	0
<ul> <li>diet margarine, low fat cheese sauces?</li> </ul>			0	(	0		0	0
c) If you eat salads, do you add								
• regu	lar mayonnaise, salad dress	ing or salad oil?	0	(	ာ		0	0
<ul> <li>diet, low fat, low calorie dressing or mayonnaise?</li> </ul>		0	(	<b>O</b>		0	0	
Fruit								
67.	Apples, applesauce	O Yes → O No		O Day O Week O Month		O i appic/ I/2 cup	O 2 apples/ 1 cup	O More than 2 apples/2 cups
68.	Bananas	O Y⇔ → O No		O Day O Week O Month	1	O I banana	O 2 bananas	O 3 or more bananas
69.	Oranges, grapefruit	O Y⇔ → O No		O Day O Week O Month		O orange /2 g'fruit	O 2 oranges 1 g'fruit	O more than 3 oran./l g'fruit
<b>7</b> 0.	Pears, peaches, nectarines, grapes plums	O Y⇔ → O No		O Day O Week O Month		O fruit /2 cup	O 2 fruit 1 cup	O more than 3 fruit/2 cups
71.	Raisins, prunes other dried fruit	O Yes → O No		O Day O Week O Month	1.	O /2 cup	O I cup	O more than 1 cup

		Do you have this food or beverage at least once a month?		t how many t ay or week o	r	uch do you have	each time?
<b>72</b> .	Cantaloupe	O Y⇔ → O No		O Day O Week O Month	less than 1/4 melon	O 1/4 melon	more than 1/4 melon
73.	Any other fruit, including berries, fruit cocktail and salad	O Y⇔ → O No	<del></del>	O Day O Week O Month	O 1 fruit 1/2 cup	O 2 fruit 1 cup	more than 2 fruit/2 cups
Beve	nges						
74.	Orange juice and other citrus juices	O Y⇔ → O No		O Day O Week O Month	O 1/2 cup	O I cup	o more than 1 cup
<b>75</b> .	Apple and other juices	O Yes → O No		O Day O Week O Month	O 1/2 cup	O 1 cup	O more than I cup
<b>7</b> 6.	Tomato, mixed vegetable juices	O Yes → O No	_	O Day O Week O Month	O 1/2 cup	O 1 cup	O more than 1 cup
<b>7</b> 7.	Fruit drinks such as Tang or Kool-Aid	O Yes → O No		O Day O Week O Month	O 1/2 cup	O 1 cup	O more than I cup
78.	Regular soft drinks (NOT diet)	O Yes → O No		O Day O Week O Month	Small or 1 can	O medium	O large
<b>7</b> 9.	Diet soft drinks	O Yes → O No		O Day O Week O Month	O small or 1 can	O medium	O large
<b>8</b> 0.	Beer, wine or liquor	O Yes → O No		O Day O Week O Month	O I glass wine or I can beer or I oz liquor	more than l glass wine or l can beer or l oz liquor	O less than l glass wine or l can beer or l oz liquor
81.	Coffee	O Yes → O No	*******	O Day O Week O Month	O 1 cup	O 2 cups	O 3 or more cups

<b>8</b> 2.	Tea		O Yes → O No		O Day O Week O Month	O 1 cup	O 2 cups	3 or more cups
83.	If you drink a) Do y	coffee ou add si	ıgar?	O Ye	; O	No		
		ch ONE o		ng do you u Cream or whole milk	ise most ofte O 2% mill		nilk O Skir	n milk
84.	If you drink  a) Do y	tea ou add su		O Yes	. 0	No		
		ch ONE o o milk or o		ng do you u Cream or whole milk	se most ofte O 2% mili		ilk OSkin	n milk
			Do you have this food or beverage at least once a month?	About l	how many tir y or week or		uch do you bave	e each time?
Desse	ert and Snacks	s						
85.	Ice cream, ice sherbet, frozen		O Y⇔ → O No		O Day O Week O Month	O 1 scoop	O 2 scoops	3 or more scoops
<b>8</b> 6.	Cake		O Yes → O No	-	O Day O Week O Month	O 1 slice	O 2 slices	O 3 or more slices
87.	Pie		O Yes → O No		O Day O Week O Month	O I slice	O 2 slices	O 3 or more slices
88.	Cookies		O Yes → O No		O Day O Week O Month	O 1-5	O 5-10	O more than 10
89.	Crackers, Ritz, cheese-ty Triscuits	⁄ре,	O Yes → O No		O Day O Week O Month	O 1-5	O 5-10	O more than 10
90.	Donuts, danish croissant	'n	O Yes → O No		O Day O Week	O I	O 2	O 3 or more

		Do you have this food or beverage at least once a month?		how many tin y or week or	nes About how muc	ch do you have	each time?
91.	Potato chips	O Yes → O No		O Day O Week O Month	O small bag	O more than small bag	O less than small bag
92.	Рорсоги	O Yes → O No		O Day O Week O Month	O 2 cups	O more than 2 cups	O less than 2 cups
93.	Peanuts, other nuts seeds	O Yes → O No		O Day O Week O Month	O 1/2 cup	o more than 1/2 cup	O less than 1/2 cup
94.	Chocolate	O Yes → O No		O Day O Week O Month	O regular bar	O large bar	O 2 pieces
		Do you use at least once a month ?	capsule	how many es or tablets eek or monti	-		
95.	Vitamin/mineral supplements	O Y⇔ → O No		O Day O Week O Month			

## APPENDIX F - DUTCH EATING BEHAVIOUR QUESTIONNAIRE

This scale measures a variety of eating attitudes, feelings, and behaviours. There are no right or wrong answers so try very hard to be completely honest in your answers. Read each question and mark your response with an "X".

**NOTE:** When responding to questions 1 and 6 **ONLY**, you may also choose "not relevant" as your response.

Very Often	Often	Sometimes	Seldom	Never	Not Relevant		
						1.	When you have put on weight, do you
	!						eat less than you usually do?
						2.	Do you try to eat less at mealtimes
							than you would like to eat?
						3.	How often do you refuse food or drink
							offered you because you are
					4		concerned about your weight?
						4.	Do you watch exactly what you eat?
						5.	Do you deliberately eat foods that are
							slimming?
		<u> </u>				6.	When you have eaten too much, do
. ;							you eat less than usual the following
							day?
						7.	Do you deliberately eat less in order
							not to become heavier?
						8.	How often do you try not to eat
1							between meals because you are
							watching your weight?
						9.	How often in the evening do you try not
							to eat because you are watching your
							weight?
						10.	Do you take into account your weight
							with what you eat?

### APPENDIX G - MULTIDIMENSIONAL BODY-SELF RELATIONS QUESTIONNAIRE

#### THE MBSRO

#### INSTRUCTIONS -- PLEASE READ CAREFULLY

The following pages contain a series of statements about how people might think, feel, or behave. You are asked to indicate the extent to which each statement pertains to you personally.

Your answers to the items in the questionnaire are anonymous, so please do not write your name on any of the materials. In order to complete the questionnaire, read each statement carefully and decide how much it pertains to you personally. Using a scale like the one below, indicate your answer by entering it to the left of the number of the statement.

1	2	3	4	5
Definitely Disagree	Mostly Disagree	Neither Agree Nor Disagree	Mostly Agree	Definitely Agree

#### **EXAMPLE:**

I am usually in a good mood.

In the blank space, enter a 1 if you <u>definitely disagree</u> with the statement; a 2 if you <u>mostly disagree</u>; a 3 if you <u>neither agree nor disagree</u>; a 4 if you <u>mostly agree</u>; or enter a 5 if you <u>definitely agree</u> with the statement.

There are no right or wrong answers. Just give the answer that is most accurate for you. Remember, your responses are anonymous, so please be <u>completely honest</u> and answer all items.

(The duplication and use of the MBSRQ permitted by Thomas F. Cash, Ph.D., Department of Psychology, Old Dominion University, Norfolk, VA 23529)

1		2	3	4	5
Defini Disag		Mostly Disagree	Neither Agree Nor Disagree	Mostly Agree	Definitely Agree
	1.	Before going how I look.	g out in publ	ic, I always	notice
	2.	I am careful look my best	to buy clot	hes that wil	l make me
	3.	I would pass	s most physica	al-fitness to	ests.
	4.	It is import strength.	ant that I ha	ave superior	physical
<del></del>	5.	My body is s	sexually appea	aling.	
	6.	I am not inv	olved in a re	egular exerci	ise program.
	7.	I am in cont	rol of my hea	alth.	
	8.	I know a lot physical hea	about things	that affect	my
	9.	I have delib life-style.	erately devel	oped a healt	thy
	10.	I constantly	worry about	being or bed	oming fat.
	11.	I like my lo	oks just the	way they are	· •
	12.	I check my a	ppearance in	a mirror whe	enever I can.
	13.	Before going getting read	out, I usual Y•	ly spend a l	ot of time
	14.	My physical	endurance is	good.	
	15.	Participatin	g in sports i	s unimportan	t to me.
	16.	I do not act	ively do thin	gs to keep p	hysically fit.
	17.	My health is	a matter of	unexpected u	ps and downs.
	18.	Good health in my life.	is one of the	most import	ant things
	19.	I don't do amy health.	nything that	I know might	threaten
	20.	I am very comin my weight	nscious of ev •	en small cha	nges

1		2	3	4	5
Definit Disagr		Mostly Disagree	Neither Agree Nor Disagree	Mostly Agree	Definitely Agree
	21.	Most people	would consid	er me good-le	ooking.
	22.	It is impor	tant that I a	lways look go	ood.
	23.	I use very	few grooming	products.	
	24.	I easily le	arn physical	skills.	
	25.	Being physi in my life.	cally fit is	not a strong	priority
	26.	I do things	to increase	my physical :	strength.
	27.	I am seldom	physically i	11.	
	28.	I take my h	ealth for gra	nted.	
<del></del>	29.	I often reacto health.	d books and m	agazines that	t pertain
	30.	I like the	way I look wi	thout my clot	thes on.
<del></del>	31.	I am self-c	onscious if m	y grooming is	sn't right.
	32.	I usually we how it looks	ear whatever . s.	is handy with	nout caring
	33.	I do poorly	in physical	sports or gam	nes.
<del></del>	34.	I seldom th	ink about my	athletic skil	ls.
	35.	I work to in	mprove my phys	sical stamina	ı.
	36.	From day to will feel.	day, I never	know how my	body
	37.	If I am sich symptoms.	k, I don't pay	y much attent	tion to my
	38.	I make no sp nutritious o	pecial effort diet.	to eat a bal	anced and
	39.	I like the w	way my clothes	s fit me.	
	40.	I don't care	e what people	think about	my appearance.

1	2	3	4	5
Definitely Disagree	Mostly Disagree	Neither Agree Nor Disagree	Mostly Agree	Definitely Agree
41.	I take spec	ial care with	my hair gro	oming.
42.	I dislike m	y physique.		
43.	I don't car activities.	e to improve	my abilities	in physical
44.	I try to be	physically a	ctive.	
45.	I often fee	l vulnerable ·	to sickness.	
46.	I pay close of illness.	attention to	my body for	any signs
47.		ng down with a nd go on as us		ı, I just
48.	I am physica	ally unattract	tive.	
49.	I never thin	nk about my ap	ppearance.	
50.	I am always	trying to imp	prove my phys	sical appearance.
51.	I am very we	ell coordinate	ed.	
52.	I know a lot	about physic	cal fitness.	
53.	I play a spo	ort regularly	throughout t	the year.
54.	I am a physi	ically healthy	person.	
55.	I am very aw health.	ware of small	changes in m	y physical
56.	At the first	sign of illr	ness, I seek	medical advice.
57.	I am on a we	eight-loss die	et.	

For the remainder of the items use the response scale given with the item, and enter your answer in the space beside the item.

(continued on the next page)

<del></del>	_ 58.	I have tried going on cra	to lose weigh	t by fasting o	or
		1. Never 2. Rarely 3. Sometimes 4. Often 5. Very Often			
	59.	I think I am	:		
		<ol> <li>Very Unde</li> <li>Somewhat</li> <li>Normal We</li> <li>Somewhat</li> <li>Very Over</li> </ol>	Underweight eight Overweight		
	60.	From looking would think 1	at me, most of	ther people	
		1. Very Unde 2. Somewhat 3. Normal We 4. Somewhat 5. Very Over	erweight Underweight eight Overweight		
61-69.	Use the	is 1 to 5 scal each of the fol	e to indicate lowing areas o	how satisfied or aspects of	you are your body:
	Use the with e	is 1 to 5 scal each of the fol	e to indicate lowing areas o	how satisfied or aspects of	you are your body: 5
	with e	each of the fol	lowing areas o	how satisfied or aspects of y	your body:
Ve:	with e	Mostly Dissatisfied	lowing areas of the second sec	Mostly Satisfied	your body:  5  Very
Ve:	with e  1 ry isfied  61.	Mostly Dissatisfied	3  Neither Satisfied Nor Dissatisfied features, comp	Mostly Satisfied	your body:  5  Very
Ve:	with e	Mostly Dissatisfied Face (facial	Neither Satisfied Nor Dissatisfied features, comp	Mostly Satisfied  Plexion)	your body:  5  Very Satisfied
Ve:	with 6  1  ry isfied  61. 62.	Mostly Dissatisfied  Face (facial Hair (color,	Neither Satisfied Nor Dissatisfied features, comp thickness, tex buttocks, hips	Mostly Satisfied  Plexion)	your body:  5  Very Satisfied
Ve:	with 6  1  ry isfied  61. 62.	Mostly Dissatisfied  Face (facial Hair (color, Lower torso (Mid torso (wa.	Neither Satisfied Nor Dissatisfied features, comp thickness, tex buttocks, hips ist, stomach)	Mostly Satisfied  Plexion)	your body:  5  Very Satisfied
Ve:	with 6  1  ry isfied  61. 62. 63. 64. 65.	Mostly Dissatisfied  Face (facial Hair (color, Lower torso (Mid torso (wa.	Neither Satisfied Nor Dissatisfied features, comp thickness, tex buttocks, hips ist, stomach)	Mostly Satisfied  Plexion)  ture) , thighs, legs	your body:  5  Very Satisfied
Ve:	with 6  1  ry isfied  61. 62. 63. 64. 65.	Mostly Dissatisfied  Face (facial Hair (color, Lower torso () Mid torso (was	Neither Satisfied Nor Dissatisfied features, comp thickness, tex buttocks, hips ist, stomach)	Mostly Satisfied  Plexion)  ture) , thighs, legs	your body:  5  Very Satisfied
Ve:	with 6  1  ry isfied  61. 62. 63. 64. 65. 66.	Mostly Dissatisfied  Face (facial Hair (color, Lower torso (Mid torso (wa. Upper torso (Muscle tone	Neither Satisfied Nor Dissatisfied features, comp thickness, tex buttocks, hips ist, stomach)	Mostly Satisfied  Plexion)  ture) , thighs, legs	your body:  5  Very Satisfied

#### APPENDIX H - MBSRQ SCORING MANUAL

### Scoring the Subscales of the MBSRQ

Item numbers comprising each	n subs	cale (w	ith * fo	or reve	rse-sc	ored it	ems):
Appearance Evaluation	5	11	21	30	42*	48*	
Appearance Orientation	1 32*	2 40*	12 41	13 49*	22 50	23*	31
Fitness Evaluation	24	33*	51				
Fitness Orientation	3 26	4 34*	6* 35	14 43*	15* 44	16* 53	25⁺
Health Evaluation	7	17*	27	36*	45*	54	
Health Orientation	8 52	9	18	19	28*	29	38*
Illness Orientation	37 <b>*</b>	46	47*	55	56		
Body Areas Satisfaction	61 68	62 (69)	63	64	65	66	67
Overweight Preoccupation	10	20	57	58			
Self-Classified Weight	59	60					

#### **Compute Statements for SPSS:**

Calculations assume responses are entered as given on the 1 to 5 scale, designated in order from B1 to B69. Formulae reverse score items by subtracting those items and adding a constant (ie. any reverse-scored item is 6 minus the given response). Each score is the mean of its items. BASS "overall appearance" item 69 is not included in the mean.

Compute Appevf=(B5+B11+B21+B30+B39-B42-B48+12)/7

Compute Apporf=(B1+B2+B12+B13+B22+B31-B23-B32-B40-B49+B41+B50+24)/12

Compute Fitevf=(B24-B33+B51+6)/3

Compute Fitorf=(B3+B4-B6+B14-B15-B16-B25+B26-B34+B35-B43+B44+B53+36)/13

Compute Heaevf=(B7-B17+B27-B36-B45+B54+18)/6

Compute Heaorf=(B8+B9+B18+B19-B28+B29-B38+B52+12)/8

Compute Illorf=(B46-B37-B47+B55+B56+12)/5

Compute BASS=(B61+B62+B63+B64+B65+B66+B67+B68)8

Compute Owpreoc=(B10+B20+B57+B58)/4

Compute Wtclass=(B59+B60)/2

## APPENDIX I - RESULTS PACKAGE FOR PARTICIPANTS

Your participation in this research study was greatly appreciated! If you have any questions, please contact Jacqui Gingras at 492-4267, University of Alberta, Human Nutrition & Metabolism

## Basic Information:

Name

Height cm

Weight kg

Body Mass Index kg/m

Waist to Hip Ratio

Research Group Metabolism



## Test Data:

	Yours	Group Average	Population Average*
Predicted RMR (kcal/day)			not applicable
Measured RMR (kcal/day)			not applicable
Percent RMR			not applicable
% Fat			
% Lean			
Bone Density (g/cm ⁻ )		<del> </del>	
Age-Matched Bone Density (Z-score)			
VO ₂₀₀ , Fitness Test (L/min)			
Insulin - Fasting (mU/L)			
Insulin - Total (mU min/L)			
Glucose - Fasting (mmol/L)			
Glucose - Total (mmol_min/l)			
Leptin - Fasting (ng/mL)			
T ₃ (nmol/L)			
T ₄ (nmol/L)			

^{*} Estimated Values

# Questionnaire Data:

Question in lein e Deite		C - A	
	Yours	Group Average	Recommendations
Calories/day			not applicable
Protein (% of cals)			
Carbohydrates (% of cals)			
Fat (% of cals)		ļ	
Fiber/day (g)			
	Yours	Group Average	Population Average*
Dietary Restraint Score (/5)			
Physical Activity Score (/15)	_		
Appearance Evaluation (/5)			
Appearance Orientation (/5)			
Fitness Evaluation (/5)			
Fitness Orientation (/5)			
Health Evaluation (/5)			
Health Orientation (/5)			
Illness Orientation (/5)			
Body-Area Satisfaction (/5)			
Overweight Preoccupation (/5)			
Self-Classified Weight (/5)			
Comments:			
<del></del>			

^{*} Estimated Values

## APPENDIX J - ETHICAL APPROVAL



University of Alberta

Inter-departmental Correspondence Faculty of Agriculture, Forestry, and Home Economics Office of the Dean

lo:

Dr. L. McCargar

date:

March 3, 1997

from:

R.J. Christopherson

Associate Dean (Research)

our file

c:\christop\ethics\mccarg2

subject:

"Amendment - Factors Affecting Metabolic Rate

your file:

and Body Image of Chronic Dieters"

This amendment was approved by the Faculty Ethics Committee subject to modifications to simplify the information sheet, and to include information on potential risks of infection associated with blood sampling. I also thank you for providing these requested revisions. The proposal is approved.

Yours sincerely,

R.J. Christopherson

Associate Dean (Research)

/may

#### APPENDIX K - CONSENT FORM

## **CONSENT FORM**

I acknowledge that the research procedures described on the Information Sheet (attached) and of which I have a copy have been explained to me, and that any questions that I have asked been answered to my satisfaction. In addition, I know that I may contact the person designated on this form, if I have further questions either now or in the future. I have been informed of the alternatives to participation in this study. I understand the possible benefits of joining the research study as well as the possible risks and discomforts. I have been assured that personal records relating to this study will be kept confidential. I understand that I am free to withdraw from the study at any time without jeopardy to myself. I understand that if any knowledge gained from the study is forthcoming that could influence my decision to continue in this study, I will be promptly informed.

(Name of Participant)	
(Signature of Participant)	
(Name of Witness)	
(Signature of Witness)	
(Date)	
(Signature of Investigator)	

Those individuals who may be contacted about the research are:

<u>Jacqui Gingras RD, BSc</u> (MSc Candidate) 492-4267 OR Linda McCargar PhD, RD 492-9287

## APPENDIX L - CORRELATIONS

Table 14: Correlation coefficients (r) for demographic, anthropometric, body composition, physical fitness, and physical activity related to REE for female chronic dieters (n=30)

CHARACTERISTICS	REE (kcal/day)	REE/kg LBM (kcal/day/kg LBM)	
Age (years)	-0.514**	-0.251	
Height (cm)	0.426*	0.068	
Weight (kg)	0.751**	0.473**	
BMI (kg/m²)	0.692**	0.486**	
LBM (kg)	0.891**	0.458*	
Fat mass (kg)	0.608**	0.420°	
Bone mineral content (g)	0.671**	0.298	
Bone density (g/cm²)	0.434*	0.202	
R1 ^a Fat mass (kg)	0.585**	0.517**	
R1/Total Fat mass	0.236	0.409*	
R2 ^b Fat mass (kg)	0.526**	0.327	
R2/Total Fat mass	-0.373*	-0.401*	
R1:R2	0.347	0.457*	
Absolute VO _{2max} (L O ₂ /min)	0.615**	0.338	
Relative VO _{2max} (mL O ₂ /min/kg body wt)	-0.249	-0.212	
Relative VO _{2max} (mL O₂/min/kg LBM)	0.001	0.008	
Physical activity score ^c	0.301	0.177	

^a R1 = abdominal fat region determined by DXA ^b R2 = gluteal fat region determined by DXA

From Baecke questionnaire, scored on a 15-point Likert scale (score of 15 = high activity)

^{*} p<0.05

^{**&}lt;sup>*</sup>p<0.01

Table 15: Correlation coefficients (r) for metabolic indices (glucose, insulin, leptin, and thyroid), and dietary intake as related to REE indices for female chronic dieters (n=30)

CHARACTERISTICS	REE (kcal/day)	REE/kg LBM (kcal/day/kg LBM)
Fasting Glucose (mmol/L)	0.241	0.378*
30-Min Glucose (mmol/L)	0.081	0.403*
60-Min Glucose (mmol/L)	0.231	0.358
90-Min Glucose (mmol/L)	0.217	0.299
120-Min Glucose (mmol/L)	0.274	0.359
Glucose AUC (mmol·min/L)	0.124	0.263
Fasting Insulin (nmol/L)	0.631**	0.607**
30-Min Insulin (nmol/L)	0.250	0.397*
60-Min Insulin (nmol/L)	0.356	0.509**
90-Min Insulin (nmol/L)	0.426*	0.616**
120-Min Insulin (nmol/L)	0.439*	0.612**
Insulin AUC (nmol·min/L)	0.329	0.559**
Fasting leptin (ng/mL)	0.450*	0.412*
120-Min leptin (ng/mL)	0.349	0.317
T ₄ (nmol/L)	0.472**	0.453*
T ₃ (nmol/L)	0.024	-0.011
r-T ₃ (nmol/L)	0.338	0.399*
Energy (kcal/day)	0.566**	0.287
Fat (g/day)	0.716**	0.379*
% Dietary Fat	0.590**	0.354
Carbohydrate (g/day)	0.238	0.100
% Dietary Carbohydrate	-0.560**	-0.336
Protein (g/day)	0.428*	0.224
% Dietary Protein	-0.076	-0.049

p<0.05 p<0.01

Table 16: Correlation coefficients (r) for other dietary factors, dietary restraint, and body

image as related to REE indices for female chronic dieters (n=30)

CHARACTERISTICS	REE (kcal/day)	REE/kg LBM (kcal/day/kg LBM)
Servings of Fruits/day	0.017	-0.028
Servings of Vegetables/day	0.229	0.102
Servings of Breads & Cereals/day	0.392*	0.284
P:S Ratio	-0.262	-0.374*
Omega-3 Fatty Acids (g/day)	0.493**	0.331
Omega-6 Fatty Acids (g/day)	0.565**	0.195
Fibre (g/day)	0.053	-0.055
Soluble Fibre (g/day)	0.121	0.009
Dietary restraint score	-0.604**	-0.539**
Appearance Evaluation ^a	-0.542**	-0.362*
Appearance Orientation ^a	-0.144	-0.160
Fitness Evaluation ⁸	0.149	0.006
Fitness Orientation ^a	-0.363*	-0.288
Health Evaluation ^a	-0.474**	-0.453*
Health Orientation ^a	-0.605**	-0.564**
Illness Orientation ^a	-0.007	0.044
Body Areas Satisfaction Score ^a	-0.520**	-0.399*
Overweight Preoccupation ^a	0.153	0.020
Self-Classified Weight ^a	0.420*	0.383*

All measures were scored on a 5-point Likert scale whereby a score of 5 denotes high dietary restraint, positive body image evaluations and orientations (appearance, fitness, health, illness), positive body satisfaction (BASS), high overweight preoccupation, and high self-classified weight (both of which are negative body image attributes).

[•] p<0.05

Table 17: Correlation coefficients (r) for demographic, anthropometric, physical fitness, and biochemical indices as related to body composition for female chronic dieters (n=30)

CHARACTERISTICS	LBM (kg)	R1 Fat mass/ R2 Fat mass	WHR*
Age (years)	-0.588**	-0.033	0.066
Height (cm)	0.592**	-0.070	-0.182
Weight (kg)	0.767**	0.147	-0.047
BMI (kg/m²)	0.670**	0.209	-0.024
Absolute VO _{2max} (L O ₂ /min)	0.676**	-0.105	0.038
Relative VO _{2max} (mL O ₂ /min/kg body wt)	-0.212	-0.313	0.103
Relative VO _{2max} (mL O ₂ /min/kg LBM)	-0.011	-0.315	0.018
Physical activity score®	0.309	-0.158	0.197
Fasting Glucose (mmol/L)	0.058	0.106	0.097
30-Min Glucose (mmol/L)	-0.180	0.225	0.339
60-Min Glucose (mmol/L)	0.079	0.171	0.142
90-Min Glucose (mmol/L)	0.082	0.369*	0.009
120-Min Glucose (mmol/L)	0.115	0.187	-0.032
Glucose AUC (mmol·min/L)	-0.011	0.315	0.117
Fasting Insulin (nmol/L)	0.475**	0.478**	0.079
30-Min Insulin (nmol/L)	0.079	0.546**	0.280
60-Min Insulin (nmol/L)	0.169	0.415*	0.101
90-Min Insulin (nmol/L)	0.186	0.548**	0.201
120-Min Insulin (nmol/L)	0.201	0.557**	0.034
Insulin AUC (nmot·min/L)	0.090	0.538**	0.182
Fasting leptin (ng/mL)	0.360	0.118	-0.126
120-Min leptin (ng/mL)	0.287	0.135	-0.038
T ₄ (nmol/L)	0.358	0.226	0.109
T ₃ (nmol/L)	0.020	0.121	-0.061
r-T ₃ (nmol/L)	0.198	0.036	-0.130

^a Three missing values (n=27 for WHR)

From Baecke questionnaire, scored on a 15-point Likert scale (score of 15 indicates high activity)

^{*} p<0.05 ** p<0.01

Table 18: Correlation coefficients (r) for demographic, anthropometric, body composition, and biochemical indices as related to physical fitness for female chronic dieters (n=30)

CHARACTERISTICS	Absolute VO _{2max} (L O ₂ /min)	Relative VO _{2max} (mL O ₂ /min/kg LBM)
Age (years)	-0.585**	-0.234
Height (cm)	0.451*	0.016
Weight (kg)	0.610**	0.099
BMI (kg/m²)	0.545**	0.091
LBM (kg)	0.676**	-0.011
Fat mass (kg)	0.496**	0.116
Bone mineral content (g)	0.713**	0.254
R1 Fat mass (kg)	0.266	-0.071
R2 Fat mass (kg)	0.485**	0.155
Physical activity score	0.287	0.084
Fasting Glucose (mmol/L)	0.088	0.075
30-Min Glucose (mmol/L)	-0.079	0.051
60-Min Glucose (mmol/L)	-0.159	-0.275
90-Min Glucose (mmol/L)	-0.209	-0.349
120-Min Glucose (mmol/L)	-0.108	-0.259
Glucose AUC (mmol·min/L)	-0.298	-0.393*
Fasting Insulin (nmol/L)	0.098	-0.340
30-Min Insulin (nmol/L)	0.035	-0.061
60-Min Insulin (nmol/L)	-0.133	-0.354
90-Min Insulin (nmol/L)	-0.210	-0.464*
120-Min Insulin (nmol/L)	-0.203	-0.456 <b>*</b>
Insulin AUC (nmol·min/L)	-0.220	-0.391*
Fasting leptin (ng/mL)	0.261	0.025
120-Min leptin (ng/mL)	0.212	0.031
T ₄ (nmol/L)	0.366	0.135
T ₃ (nmol/L)	-0.001	-0.008
r-T ₃ (nmol/L)	0.393*	0.332

From Baecke questionnaire, scored on a 15-point Likert scale (score of 15 indicates high activity)

[°] p<0.05 °° p<0.01

Table 19: Correlation coefficients (r) for body composition, fat distribution, and insulin as related to leptin for female chronic dieters (n=30)

CHARACTERISTICS	Fasting Leptin (ng/mL)
LBM (kg)	0.360
Fat mass (kg)	0.846**
% LBM	-0.888**
% body fat	0.893**
R1 ^a Fat mass (kg)	0.783**
R1/total body fat	0.172
R2 ^b Fat mass (kg)	0.859**
R2/total body fat	-0.110
R1Fat mass/R2 Fat mass	0.118
WHR	-0.126
Fasting insulin (nmol/L)	0.395*
30-Min insulin (nmol/L)	0.121
60-Min insulin (nmol/L)	0.171
90-Min insulin (nmol/L)	0.209
120-Min insulin (nmol/L)	0.239
Insulin AUC (nmol-min/L)	0.141

^a R1 = abdominal fat region determined by DXA ^b R2 = gluteal fat region determined by DXA

p<0.05

Table 20: Correlation coefficients (r) for anthropometrics and dietary intake as related to dietary restraint and self-classified weight for female chronic dieters (n=30)

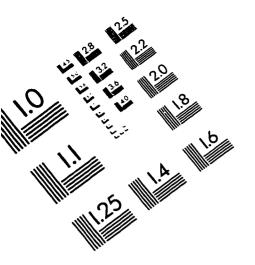
CHARACTERISTICS	Dietary Restraint ^a	Self-Classified Weight ^b
Weight (kg)	-0.436*	0.669**
BMI (kg/m²)	-0.400*	0.727**
Fat mass (kg)	-0.352	0.718**
% body fat	-0.240	0.768**
Energy (kcal/day)	-0.238	0.139
Fat (g/day)	-0.452*	0.280
% Dietary Fat	-0.596**	0.310
Carbohydrate (g/day)	-0.061	-0.006
% Dietary Carbohydrate	0.335	-0.219
Protein (g/day)	0.026	-0.002
% Dietary Protein	0.432*	-0.227
Appearance Evaluation ^b	0.120	-0.587**
Appearance Orientation ^b	0.433*	-0.318
Fitness Evaluation ^⁵	-0.089	-0.377*
Fitness Orientation [®]	0.334	-0.547**
Health Evaluation ^⁵	0.313	-0.390*
Health Orientation [®]	0.550**	-0.685**
Illness Orientation ^D	-0.235	-0.165
Body Areas Satisfaction [□]	0.343	-0.707**
Overweight Preoccupation ^b	0.378*	-0.064
Self-Classified Weight ^b	0.305	1.00

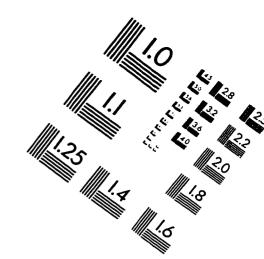
From the Dutch Eating Behaviour Questionnaire, scored on a Likert scale whereby a score of 5 indicates high dietary restraint

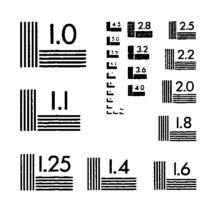
All measures were scored on a 5-point Likert scale whereby a score of 5 denotes positive body image evaluations and orientations (appearance, fitness, health, illness), positive body satisfaction (BASS), high overweight preoccupation, and high self-classified weight (both of which are negative body image attributes).

^{*} p<0.05

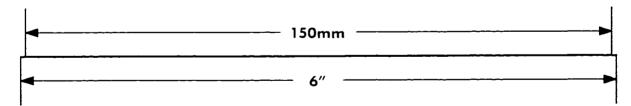
p<0.01

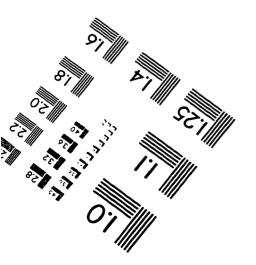






TEST TARGET (QA-3)







© 1993, Applied Image, Inc., All Rights Reserved

