WEIGHT CYCLING IN COMPETITIVE JUDOKAS

by

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ABSTRACT

It has been suggested that repeated cycles of weight loss, followed by weight gain (termed weight cycling) will lead to metabolic and physiological adaptations which increase food efficiency and thus cause resistance to future weight loss. Athletes who compete in Judo (named judokas), were used as an athletic model of weight cycling. Some judokas cut weight (lose weight) repeatedly, in order to meet specific weight categories for competition, and may therefore represent a weight cycling population. Consequently it is important to determine if suggested metabolic and physiological adaptations associated with weight cycling occur in these athletes.

Two groups, weight cyclers (n=9) and matched non-cyclers (n=9), were followed over a period of 10 months (including one competitive season). They followed similar training routines. Weight cyclers dieted (cut weight) in order to compete in a weight class below their usual body weight, because they believed that this would give them an advantage over their opponents. Dieting habits were characterized by food restriction an average of 8.1±6.9 (mean±SD) days before their competition. Following the competition, weight was regained to baseline values.

There was no significant difference in lean body mass, percentage body fat, height, weight, age, or activity level between the two groups at baseline. Measurements were recorded three times within a one year period. The first pre-season (baseline) value was measured when the judokas were training, but had not yet started to diet for competition. The second test session was during their peak season, at which point the weight cyclers had experienced episodes of weight reduction to meet

competitive weight classes. The final test session was conducted during the off-season, at least three months after the Judo season had ended. Measurements included resting energy expenditure (REE), body composition (skinfolds), usual three day food intake (three day food records), and biochemical parameters (fasting insulin, glucose and triiodothyronine).

The weight cyclers lost 4.1±1.5 kg, 4.2±2.7 (mean±SD) times per season, whereas the non-cyclers lost little or no weight throughout the study. Weight loss was achieved primarily through food restriction. There was no significant difference in metabolic or physiological parameters between the weight cyclers and the non-cyclers during any of the three test sessions. This study indicated that there were no effects of weight cycling in this athletic population. It is possible that regular physical activity protected against any suggested metabolic adaptations.

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CHAPTER 1

BACKGROUND

1.1 INTRODUCTION

Dieting has become a common practice among many individuals today. Surveys show that over 50% of Canadians want to change their weight primarily through weight loss and 70% of women at a "good weight-for-height" still want to lose weight.[1] Athletes who must meet weight categories for competition are also frequent dieters. One survey indicated an average wrestler would "cut weight"(lose weight) 15 times per season and 41% of those surveyed would have a weight fluctuation between 5.0 and 9.1 kg per week.[2]

Under normal circumstances, dieting can have beneficial health effects, specifically in obese individuals, who are at risk for coronary heart disease, hypertension, Type II diabetes, and hypercholesterolemia.[3] However, success of maintaining weight loss has been reported as very low. Sjostrom[4] specifically stated that a relapsing patient can be identified in advance with high precision by predicting that everyone will relapse. This necessitates further cycles of dieting which is termed "yo-yo dieting" or "weight cycling".

Such a pattern of weight fluctuation has been correlated with certain health risk factors. High individual variability in body mass index (BMI) accompanied by a high BMI, produced a 2.5 fold increased risk of coronary heart disease, compared to normal and stable BMI values.[5] Ashley et al[6] studied subjects losing and gaining weight, and found that weight loss was associated with a decrease in blood pressure, and weight

gain was associated with an increase in blood pressure. The increase in blood pressure was greater than the decrease, having a net detrimental effect. Additionally, severe protein catabolism associated with dieting may result in myocardial protein loss and impairment of cardiac function. Cardiac atrophy is proportional to muscle atrophy, even when adipose tissue is present.[3]

Athletes are rarely obese, and usually are at low risk for coronary disease, hypertension, and diabetes. However, athletes who must cut weight frequently to meet specific weight categories, may incur deleterious metabolic consequences. The purpose of this study was to determine if weight cycling in competitive judokas altered specific metabolic and physiologic parameters. To accomplish this, resting metabolic rate (RMR), serum triiodothyronine (T_3), insulin and glucose levels, as well body composition and fat distribution were examined. This study was approved by the Clinical Screening Committee at UBC (appendix I).

1.2 METABOLISM

Due to the high prevalence of obesity and a high failure rate of dieting, it has been speculated that food restriction may have detrimental metabolic consequences. It has been well documented that chronic dieting leads to a reduction in resting energy expenditure as an adaptive response for energy conservation[7-13]. This allows the body to minimize loss of weight, fat and lean body mass (LBM)[7] when food becomes

scarce, and it is a useful mechanism when famine may pose a threat to one's survival. It is however, a frustrating consequence when weight loss is desired.

There are three main components to energy expenditure, all of which may be affected by the dieting process. The first and most important component is resting energy expenditure (REE) expressed per day. When expressed as an hourly rate it is termed resting metabolic rate (RMR). It is the largest contributor to daily energy expenditure (70-75%)[14] and has the most profound effect on daily caloric requirement if it is altered. The REE is influenced by lean body mass, hormones, age, genetics and physical training. The second component of energy expenditure is diet induced thermogenesis, which contributes approximately 10%[14]. This is the energy required to digest, absorb, and assimilate nutrients. Of the three main nutrients, (carbohydrate (CHO), fat, and protein) protein has the highest thermogenic effect and fat has the lowest. The final component is exercise induced thermogenesis. This is the energy required to perform activity and it is dependent on the intensity and duration of the activity. It contributes approximately 20% to the daily energy expenditure in the average individual, but is very variable among individuals and can be considerably higher in athletes[14].

If caloric restriction reduces any of these three components, as has been suggested by numerous studies,[7-13] it could make weight loss formidable. If such metabolic consequences were to persist when normal food intake resumed, it would make weight maintenance difficult. This

could initiate the recurring pattern of the cyclic dieter, especially if the effects of each diet were additive. For this reason it is important to take an in depth look at metabolic consequences of dieting. Whether it is in the case of obesity, eating disorders (anorexia nervosa), or competitive athletes cutting weight, if metabolic adaptations lead to a reduced energy expenditure, the use of severe food restriction to achieve weight loss should be avoided.

1.2.1 METABOLIC EFFECTS OF DIETING

Prolonged calorie restriction results in a reduced RMR[9-12]. The composition of weight lost may influence this decrement. Under a weight reducing regime one would expect a loss of both fat and lean body mass (LBM). The percentage of weight lost as LBM is related to the severity of food restriction with losses being the greatest with complete fasting.[8] Studies have shown that RMR is highly correlated with LBM,[15] as it is the most metabolically active component of the body. Although it still remains controversial as to what body measurement ie. fat weight, body weight or LBM is the best predictor of RMR,[16] LBM values can be used to predict RMR from multiple regression equations.[14] It would therefore be expected that weight reduction would result in a decline in RMR in proportion to the amount of LBM lost.[8] Furthermore, if weight loss was rapid (very low calorie dieting [VLCD] or fasting), the percentage of LBM lost would increase, enhancing the depression of metabolic rate. This may be why so many individuals who have had

difficulty losing weight, tend to regain lost weight[17], or else require less than normal energy intake to maintain reduced weight.[18]

Of the numerous studies that conclude RMR is reduced with caloric restriction[9-12] some have shown reduced rates beyond those which compensate for LBM losses.[7,8,11,13] Vansant[8] et al demonstrated that both absolute RMR and RMR/LBM were significantly decreased in women on a long term (6 months) protein sparing modified fast (PSMF) (2840-720kcal/day). However this effect was not observed during a short term fast of two weeks. Elliot[13] et al demonstrated that obese women dieting for 10 to 20 weeks on a PSMF of 300 kcal/day (45g protein) had RMR reduced by 22%. When corrected for LBM, the RMR was still significantly lower than pre-diet values, indicating that loss of lean tissue could not account for the entire reduction in RMR.[13] Barrows[7] et al also concluded that factors other than reduced LBM must be involved in depression of RMR. They found that obese subjects restricted to 420 kcal/day for 4-6 months had a decreased rate of weight loss by 3 months and the RMR per LBM had declined significantly.

Whether reduced energy expenditure is sustained upon refeeding of a weight maintaining diet has been questioned. The obese women in the study of Elliot and co-workers maintained a stable reduced rate for two months and a sustained decrement in RMR persisted, despite an increased caloric consumption.[13] Geissler[19] et al compared the metabolic rate of post obese subjects against matched lean controls and found that the post obese group had a 15% reduction in 24 hour energy expenditure. Only 10%

of the 15% reduction in energy expenditure could be attributed to a depressed basal metabolic rate (BMR). The other 90% reduction in energy expenditure was identified during three different set levels of activity (from sedentary to aerobically active). Therefore it appears that the majority of the reducted 24 hour energy expenditure is due to a decrease in exercise induced thermogenesis and possibly diet induced thermogenesis.[19] Whether such differences are caused by dieting, or necessitate dieting has not yet been determined.

Other studies have found no sustained effect of dieting on RMR beyond that expected with reduction in LBM. Rattan[20] et al showed that subjects on a VLCD for 8 weeks had a reduced RMR of 86% of their initial values, which recovered to 93% upon refeeding with a weight maintaining diet (for another 8 weeks). The decrement could be accounted for by loss of LBM. A recent study demonstrated similar results. Wadden[21] et al studied two groups of obese patients (9 in each group). One group consumed 1200 kcal/day for 48 weeks. The second group consumed 420 kcal/day(VLCD) for 16 weeks and then switched to a conventional reducing diet (1200 kcal/day) for the remaining 31 weeks (total 48 weeks). While on the VLCD the latter group had a decrease of RMR by 19.8%, but when switched to the 1200 kcal/day regime, their RMR was only depressed by 8.3%. This was comparable to a 9.4% reduction in the group continuously consuming 1200 kcal/day and could be accounted for by loss of LBM.[21]

The literature still remains controversial with respect to the metabolic effects of dieting. It is evident that there is a reduction in

daily energy requirements when food restriction is severe enough, but whether it is due to a decreased RMR, or a reduced diet or exercise induced thermogenesis has yet to be confirmed. Whether the RMR remains depressed over a longer time period is not known. Due to the variation in degree of caloric restriction, dietary composition, diet length and populations studied, it is difficult to compare experimental findings and reach a strong consensus.

1.2.2 METABOLIC EFFECTS OF WEIGHT CYCLING

If a single bout of dieting will result in a depressed RMR and increased energy efficiency, then weight loss and regain which is repeated several times could exacerbate this adaptation, resulting in resistance to weight loss. Miller and Parsonage[22] found that women who were resistant to weight loss had a long history of dieting. Jeffery[23] et al suggested that a history of dieting was a poor prognostic sign for treatment. Metabolic effects of weight cycling which could explain these findings have been investigated in both animals and humans. Methods that have been used to assess the metabolic effects of weight cycling include changes in RMR, velocity of weight loss and weight regain from one cycle to the next, and the food efficiency ratio (FER) or weight gained/gram of food consumed.

ANIMAL STUDIES

Weight cycling was first studied in rodents. Brownell[24] et al

studied 21 week old male rats divided into three groups. One group were chow controls, fed ad libitum. The second group were obese controls, fed a high fat diet (63% of energy as fat). The third group were obese "weight cyclers". In the first cycle the obese cycling rats were restricted to 50% of the average intake of chow controls, until they reached the weight of these controls. They were then refed the high fat chow until they reached the weight of the obese controls. The second cycle was a repeat of this protocol. The number of days to lose and regain the weight in each cycle were compared. The desired weight loss in the first cycle required 21 days, but by the second cycle, weight loss occurred at half the rate, requiring 46 days. Weight regain was three times as fast during the second cycle, despite an identical food intake during both cycles. Food efficiency (weight gain/g food) had increased 4 fold when compared to the obese controls. Changes in metabolic efficiency as a result of increased age were controlled for. It was concluded that after two cycles of weight loss and regain, weight loss was slower and weight regain more rapid at the same level of energy intake.

Cleary[25] investigated the effects of weight cycling in 6 week old lean and obese Zucker rats. Four groups were investigated; 1) ad libitum lean (AL), 2) restricted lean rats (RL), 3) ad libitum obese rats (AO), and 4) restricted obese rats (RO). The restricted groups had three weeks feeding at half the amount of the respective ad libitum groups, followed by three weeks ad libitum. They underwent four similar cycles. The RL

rats gained three times the weight of AL rats on similar food intakes and the RO rats gained 50% more weight than the AO rats, despite a lower food intake (ad libitum) during the first refeeding episode. In the subsequent cycles, both weight cycling groups lost and regained weight, were as the non-cycling groups maintained weight (similar intake to refeeding diet of weight cyclers). It is therefore evident that the effect of weight cycling on energy efficiency occurs in both lean and obese rats. Food efficiencies were compared, and during the first and second refeeding period both restricted lean and obese rats had a significantly increased FER as compared to their respective controls. In the last two refeeding periods the FER was greatest for the RO rats, and then the RL rats, both being significantly greater than AL or AO rats.[25]

In contrast, Hill et al[26] found no increase in energy efficiency in weight cycled adult male rats. The rats went through four cycles of 3 days fasting (at either 100%,60% or 40% ad libitum) followed by 7 days refeeding. Similarly, Cleary[27] found four cycles of food restriction followed by refeeding in both lean and obese female Zucker rats, had no effect on food efficiency. A review of 18 weight cycling studies done in animals[27] led to the conclusion that no clear evidence suggested difficulty of future weight loss or ease of weight gain as a result of weight cycling. Nor was there consistent evidence of increased total body fat or central adiposity, increased subsequent caloric intake, increased food efficiency, or decreased energy expenditure[27].

Some of the inconsistencies in animal studies may be explained by

variations in the age of the rodent used. Rodents restricted in a growth stage may undergo compensatory growth or catch up growth[28] upon refeeding. This results in an increased food efficiency ratio which is not necessarily a result of weight cycling. Consequently, similar results may not be demonstrated in adult rats under the same experimental conditions.[25] Further efforts must be made to conduct more controlled animal experiments.

HUMAN STUDIES: OBESE

Human experiments on weight cycling are limited and again, inconsistent. Such inconsistencies result from different diet treatments and/or levels of compliance, different degrees of obesity or leanness, different methods of collecting weight cycling data, and different criteria to define weight cycling. Sex specific differences, and individual differences in response to weight cycling must also be considered.[29]

Blackburn[29] et al investigated the metabolic efficiency of inpatient and outpatient obese individuals by assessing the velocity of weight loss over 2 successive diet cycles. In the inpatient group (n=13) it was concluded that the velocity of weight loss in a second cycle (after regaining 20% of cycle one weight) was slower than in the first (on PSMF). A similar decrease in velocity of weight loss was seen in the outpatient group (n=40) on a second cycle following a 95% regain of lost weight. The authors suggested that chronic dieting leads to permanent

alterations in metabolic function which results in difficult weight loss and easy regain.[29]

There were some limitations to this study. The length of time which had lapsed between diet cycles was as much as 9 years and changes in metabolism with aging must be considered. Secondly, those subjects who weighed less by their second cycle would be expected to have a decreased velocity of weight loss due to reduced energy required for weight bearing activity and reduced lean body tissue.

Beeson et al[30] gave a brief report on the rate of weight loss in obese individuals (n=4) between 2 consecutive VLCD's (330 kcal/day and 405 kcal/d respectively). They found that there was no change in the rate of weight loss by the second cycle. The subjects had regained between 0% and 83% of the lost weight in between the two cycles. No information was given about the individuals' sex, dieting history, LBM, % fat and degree of obesity and there were only four subjects participating in the study.

In contrast Van Dale[31] et al looked at absolute RMR following a 14 week VLCD in obese weight cyclers and non-cyclers. Weight cyclers were defined as those who were "frequently dieting", and had lost and regained greater than or equal to 10 kg in the past 5 years, more than once. Body weights were stable for the past 6 months. Half of each group was randomly assinged to an exercise program. The exercise program consisted of two days of aerobics, 1 day of fitness, and 1 day of jogging,

totalling four hours of exercise at 60% VO_2 max per week. The weight reducing diet included three phases; (1) 5 weeks VLCD (722 kcal/day), (2) 9 weeks VLCD (528 kcal/day + 312 kcal/day conventional foods), and (3) 2 weeks VLCD (722 kcal/day). The RMR had decreased significantly from baseline in all groups following the 14 week study period, but to a significantly lesser extent in both weight cycling and non-cycling exercise groups, when compared to non-exercising groups. There was no significant difference in RMR between the weight cyclers and non-cyclers in either exercising or non-exercising groups.

The dieting history in this study may be questionable, as it is unlikely that an obese person would not have tried to diet at some point in their life time. Only 5 years preceding the study were analyzed and it's possible that those that were not weight cycling had in fact dieted prior to the 5 years and were still suffering the metabolic effects. The definition of a weight cycler is different from other experiments.

A more recent study looked at the BMR of obese female cyclic dieters who underwent 3 cycles of 2 weeks on an 443 kcal/d diet followed by 4 weeks of ad libitum refeeding.[32] Classification as a weight cycler required the subject to have lost 6 kg on 2 or more occasions. It was found that the absolute BMR was significantly different during all restriction times, but when it was corrected for LBM, only the first cycle showed a significant decrease in BMR. Furthermore the BMR returned to normal following each 4 week refeeding period. At the end of the study there was no significant difference in absolute BMR and BMR/kg FFM.

BMR/kg had increased indicating loss of adipose tissue, with no excessive loss of LBM.[32]

HUMAN STUDIES: NON-OBESE

There may be differences in metabolic responses between lean and obese individuals. One group[33] looked at non-obese females who had a history of cyclic dieting. This was defined as having dieted for greater than or equal to 7-10 days, 4 times in the last year. A diet was considered to be an energy intake 1000 kcal/d below energy requirement for weight maintenance. There were 11 weight cyclers who had lost an average of 3.5-8.2 kg on each diet and 12 non-cyclers who had no history of weight loss through dieting. They found that the cyclic dieters weighed more and had more body fat. LBM was the same. The REE of the weight cyclers was compared to the matched controls and they found that REE/kg was significantly reduced (this was because they had a higher percentage fat which is less metabolically active) but that absolute REE was the same (this is because they were matched for lean muscle mass which is more metabolically active).

The investigators also looked at exercise energy expenditure (EEE) to see if there was a difference in exercise induced thermogenesis in the two groups. EEE was estimated by measuring oxygen uptake while exercising on a treadmill. Each subject followed a similar exercise protocol, walking at 4.8 km/h, with the treadmill grade increasing 2.5% every three minutes. Eight workloads were completed. They found that

EEE/kg was significantly reduced, but that the absolute EEE was the same (ie. there was no increase in energy required to move the excess weight of the heavier people as may be expected, even with no differences in LBM). Therefore they concluded that non-obese cyclers did not decrease baseline metabolic rate but that they were more efficient in their exercise, requiring less energy to move around a larger mass.[33] Over a period of a year this could have a significant effect on weight maintenance.

HUMAN STUDIES: ATHLETES

Weight cycling is also common among athletes, especially those who compete in certain weight classes (wrestling and judo) or those who are required to be lean for aesthetic purposes (gymnastics). Cutting weight often requires athletes to go below their natural or usual weight. Following the set point theory, the body will try to defend its natural weight, through changes in metabolism.[34] The effect of food restriction would be expected to be greatest among those furthest below their natural weight and in those who have lost and regained numerous times in the past.[2]

Wrestlers are a notable example of athletes who must "cut weight", and rapid, frequent weight loss is often necessary. One survey[2] showed that of a group of collegiate wrestlers, an average wrestler would cut weight 15 times in a season, and 41% of those studied had a weight fluctuation between 5.0 and 9.1 kg every week. Because weight loss is

short term and may be severe, it can result in loss of both fat and large amounts of lean tissue. Furthermore weight regain is rapid, causing added weight to potentially be deposited as fat rather than muscle.[2]

Steen[35] et al compared the RMR of weight cyclers and non-cyclers in high-school wrestlers. A weight cycler was defined as one who cut weight greater than or equal to 10 times per season, had a weekly loss greater than or equal to 4.5 kg and who was often or always cutting weight. A non cycler would cut weight less than 5 times in a season, with a weight loss not more than 1.4 kg and considered frequency of weight cutting to be never, rarely or sometimes. Cyclers and non cyclers had no significant difference in age, height, weight, body surface area, percentage body fat, or LBM. It was found that RMR was significantly lower (14%) in cyclers, even when corrected for differences in body composition. The cyclers required 255 kcal/day less than the non-cyclers under resting conditions.

A more recent study on weight-cycling in wrestlers was conducted by Melby et al.[36] They designed a longitudinal study which looked at collegiate wrestlers over a period of a year, to see if there was a metabolic effect after a season of cutting weight. The wrestlers were matched to physically active men who did not weight cycle and did not have a history of dieting. The criteria for weight cycling classification included a loss of greater than or equal to 4.5 kg at least 10 times in a season for at least the past 3 years. The three test periods included pre-season, during the competitive season 24-48 hours

prior to a competition when food restriction, but not dehydration had taken place, and off season. This took place 5-6 weeks following the competitive season when weight had normalized and was stable.

The results of the experiment indicated that at baseline the wrestlers in fact had a significantly greater absolute and relative RMR per kg and per fat free weight (FFW) than their matched controls. This indicates that there were no previous metabolic effects of weight cycling in the wrestlers, which might be expected after three years of practising such dieting habits. When the wrestlers were cutting weight, their RMR was significantly decreased from pre-season values but was not significantly different from the controls. This depressed RMR is as expected, due to food restriction. RMR/FFW and RMR/kg were also significantly lower, indicating that RMR was decreased beyond that which compensates for loss of weight and lean tissue. This is also a demonstrated phenomenon of food restriction. The most significant finding was that the absolute and relative RMR returned to baseline values 5-6 weeks after the competitive season. This was accompanied by an increase in LBM back to baseline values. The percentage fat did increase slightly above baseline values but it was not significant.

The subjects were not matched for type, duration, or intensity of exercise, which can significantly affect RMR. Furthermore two different techniques for measuring body fat were used. Underwater weighing was used for baseline values for the wrestlers, whereas skinfolds were used for the remainder of the data collection and for all control measures.

Since there is controversy over the variation between the two methods, the body composition and REE/LBM results may lack precision.[36]

1.2.3 SUMMARY OF METABOLISM

Scientists have yet to establish if altered metabolism promotes weight cycling or if it is the result of weight cycling. Furthermore, it is not known whether only RMR is affected or if other components of energy expenditure are also affected. Applicability of animal studies to humans is questionable due to different metabolic regulatory mechanisms and responses. The few human studies that have been done use different populations with different weight cycling habits and classification criteria. Obese individuals, lean individuals and athletes may differ with respect to body composition and food intake, and may therefore respond differently to repeated bouts of food restriction. Different degrees of restriction may be required for consequences to be realized.

1.3 BODY COMPOSITION

Another potential problem with weight cycling is a change in body composition and fat distribution. It has been suggested that repeated bouts of dieting may result in a higher percentage of body fat, whether it is due to a large loss of lean body mass caused by severe caloric restriction, or a tendency to regain lost weight as adipose tissue (or both). The end result in any case may be a progressive loss of metabolically active tissue, which could be replaced by energy rich

adipose tissue.[8]

1.3.1 BODY FAT

ANIMAL STUDIES

Review of the literature shows conflicting results regarding the effects of weight cycling on body composition. Stock et al[37] found that obese rats on a low calorie diet had an increased percentage fat and a decreased percent lean body mass following weight reduction, indicating an excessive loss of LBM. Ozelci et al[38] found that rats restricted to 50% and 25% of ad libitum food intake for one week and then pair fed with an ad libitum control group for three weeks, had a body fat content greater than their ad libitum controls. The restricted rats demonstrated an increased propensity to convert food energy to body fat. This was not influenced by meal pattern and resulted with either a high carbohydrate or a high fat diet. The increased body fat was not demonstrated in rats restricted to 75% ad libitum food intake at the end of four weeks. It was concluded that weight gain and fat gain were more pronounced with increased severity of caloric restriction.[38] Reed et al[39] found that rats that went through 3 cycles of dieting and refeeding were fatter and had a greater number of adipocytes than chow controls despite a lower body weight.

Other experiments show conflicting results indicating no change in body composition with one cycle[40] or repeated cycles[41,42-44] of food restriction followed by refeeding. Lean female Zucker rats, age 14

weeks, were restricted for 22 days to 40% of the control food intake and were refed ad libitum for 20 days. Lost fat was replaced on the sixth day and protein and body weight were recovered, but did not exceed control rats fed ad libitum, by the 13th day of refeeding. When compared to the control rats, the restricted rats were found to have similar body fat content.[40]

Gray et al[42] found similar results in weight cycled adult female Sprague-Dawley rats made obese by consuming a high fat diet. Those that underwent two cycles of restriction and refeeding ad libitum showed no difference in weight or percentage body fat. Male Sprague-Dawley rats starved for three days had body weights, adipose tissue content and fat cell weights that had returned to control values upon refeeding.[43] Three cycles of weight loss and regain in White-Footed Mice resulted in no significant differences in final weight or fat content.[44]

HUMAN STUDIES

Human studies are limited and equally controversial. An increase in adiposity as a result of food restriction was first demonstrated in normal weight young males who had lost 24% of their body weight during 24 weeks of semi-starvation.[45] Upon refeeding the men recovered 93% of their body fat, yet only 60% of their body weight was regained. Although the study has its limitations in that the physical activity and diet of the refed men were not observed, and weight was not 100% regained, a higher proportion of lost body fat was recovered, as compared to lean body mass. This may indicate that food restriction with subsequent

refeeding results in an increase in percentage of body fat and a reduction in lean body mass.[45] A cross sectional study conducted by Manore et al[33] found that non-obese females who had a history of cyclic dieting had a higher body weight and a higher percentage body fat than women who had never dieted. Their LBM, however was the same as controls.

Melby et al[36] followed wrestlers through a season of cutting weight for competitions and found that during the season they lost a significant amount of weight as both lean tissue and body fat. Upon refeeding LBM and percentage of fat returned to baseline values (pre-season). However, it is possible that the athletic population is resistant to an increase in adiposity as a result of regular exercise which protects against loss of LBM and accumulation of body fat.

1.3.2 FAT SELECTION

Change in body fat may be related to diet composition. Food restriction has been suggested to result in an increased selection of fat upon refeeding[39,46-48] and such a diet has been suggested to promote deposition of adipose tissue, rather than lean body mass.[49-51] With weight cycling there will be repeated episodes of weight regain and the potential for repeated episodes of high fat selection, resulting in increased fat stores. It should be noted that fat selection studies to date, have only been conducted in animals.

Reed et al[39] found that female Sprague-Dawley rats restricted to 25% of their baseline weight, and who were then allowed to self-select their own diet upon refeeding, had a tendency to choose a diet high in

fat. These rats, compared to controls, who could self select throughout the experiment, had a fatter retroperitoneal and parametrial deposit, and heavier fat pad weights, despite a lower overall body weight. They also had a greater number of adipocytes in both fat pads. Fat cell size was significantly smaller, but a longer refeeding period may have resulted in increased size as well (they were sacrificed at the end of the experimental protocol).

Weight cycled rats have shown a progressive increase in the percentage of fat selected in the diet over two cycles.[52] Female rats after one bout of food restriction were found to select 56% of their calories from fat as compared to 44% in the ad libitum control rats. After the second cycle, fat selection had increased further to 73%, whereas the control rats had remained the same. Furthermore the weight cycled rats had an increased percentage body fat despite a lower body weight.[52] These results are difficult to interpret as the rats were in a growth stage. It might be expected that an even further increase in percentage body fat may result in adult rats not experiencing catch-up growth.

Conversely, Graham et al[53] found that lean female cycled rats (three cycles) given a high fat (60%), high caloric diet, only selected the number of calories required to match the reduced RMR caused by the weight cycling. In fact the control rats weighed more and were fattier than the weight cycled rats. These rats showed no excessive intake of calories or fat resulting in an increased adiposity.

1.3.3 DISTRIBUTION

Another area of importance concerning body adiposity is fat distribution. It has been suggested that repeated cycles of weight loss and regain may result in the redistribution of fat which can lead to increased health risks.

Studies have shown that android (upper body) fat is associated with a greater risk of coronary heart disease and that a high waist to hip ratio (WHR) is a better predictor of diabetes and mortality from coronary heart disease than body weight, body mass index and percentage body fat.[2] Gynoid (lower body) obesity is associated with less risk of hypertension and impaired glucose tolerance.[54]

These differences may be attributed to the different types of receptors located in these two areas. Beta-receptors located in the upper body enhance fat transfer. As a result, triglycerides in this area are more readily mobilized and are available to form atherosclerotic plaques, which can subsequently increase blood pressure and increase the risk of coronary heart disease. Alpha-receptors in the gynoid region inhibit fat transfer to and from the cell, making fat mobilization difficult. This fat is trapped and less available to contribute to such health risks.[2]

Currently only one weight cycling study has investigated the question of fat distribution.[55] This was evaluated in non-pregnant, pre-menopausal women. A random sample of 87 normal weight women between the ages of 21 and 40 were measured for WHR determination and identification and rating of weight cyclers was achieved by questionnaire. It was found

that a higher WHR was significantly associated with a higher degree of weight cycling, controlling for age and parity. Normally this population would tend to have a higher deposition of fat in the gluteofemoral region, but if excessive abdominal fat was present, a history of dieting may be one explanation.

Until further studies are done, it can only be speculated that fat redistribution to the upper body would occur with repeated bouts of weight loss and weight gain in other populations.

1.3.4 SUMMARY OF BODY COMPOSITION

Current studies have led to a number of inconsistent findings. Animal studies must be viewed with caution, as some use growing rats and others use adult rats. These two groups may respond differently to weight cycling, as it is known that growing rats who are restricted undergo compensatory growth in an attempt to catch up to their natural growth pattern. As a result they require high caloric intakes for growth and fat deposition may not occur, even on a high fat diet. Furthermore it is questionable as to whether rat studies are representative of humans, particularly with respect to eating behaviors such as fat selection. Rats tend to choose appropriately to increase their chance of survival. Humans however, are influenced by psychological factors and not simply physiological need. As a result they may be more or less susceptible to selecting higher fat diets, as well to consume more calories than are required for weight maintenance.

1.4 BLOOD PARAMETERS

1.4.1 INSULIN AND GLUCOSE

Insulin has an important function in the regulation of CHO, lipid and protein metabolism, and changes in hormone function can be seen with diet manipulation and changes in body weight. In obesity, insulin resistance is quite prevalent. Under such conditions, mild hyperinsulinemia can occur as a result of reduced insulin receptors on target tissues.[56] As a result the ability of the target tissue to respond to insulin is reduced and glucose utilization is impaired.[57] Post-receptor abnormalities can also occur in more severe cases, resulting in limited glucose transport and the possibility of developing diabetes. Weight reduction usually reverses insulin resistance by increasing the number of receptors on various tissues. A simultaneous fall in basal plasma insulin levels will occur.[56]

Fasting and caloric restriction also have a profound impact on plasma insulin concentrations and hormone function. Numerous studies have shown that glucose and insulin levels are reduced with fasting in both lean and obese individuals and a transitory loss of glucose tolerance has been observed.[58] Strubbe et al[59] found that basal insulin levels dropped in 24 hour fasted male rats, as a result of decreased insulin secretory responses of B-cells to glucose. Reduced insulin levels would aid in the change in energy utilization from CHO to lipid metabolism, an important adaptation to conserve glucose to fuel the central nervous system.

Reduced levels of insulin result in enhanced lipolysis and reduced lipogenesis causing endogenous fat to be readily mobilized for fuel, and gluconeogenic precursors to be made available.[60] The lipogenic effect of insulin is inhibited with calorie restriction due to post receptor changes, which alters transport and metabolism of glucose, and makes it unavailable as a glycerol-3-phosphate precursor for reesterification. Lipogenesis becomes unresponsive to insulin even at physiological levels of glucose.[61]

Despite an obvious shift towards enhanced lipid mobilization, increased sensitivity to insulin's antilipolytic action has been demonstrated during caloric restriction.[59,61] Arner et al[61] studied isolated human fat cells from obese females who had fasted for seven days and found an enhanced antilipolytic action, by stimulating insulin receptor binding. This may be a protective mechanism to prevent excess lipolysis and subsequent ketoacidosis.[59] Extreme protein losses are also prevented by a preservation of insulin's role in proteolysis inhibition[62], ensuring that lean body mass is conserved.

A decrease in plasma glucose and insulin is observed with fasting, however cyclic dieting appears to have a completely different effect. Cleary[25] examined lean and obese Zucker rats that went through four cycles of restriction and refeeding. They found that there was a trend towards increased fasting serum insulin and glucose levels in comparison to the control group (fed refeeding diet continuously), after the last restriction period and last refeeding period. The restricted obese rats had even higher values than the restricted lean rats. Reed et al[39] also

demonstrated that female rats that went through 3 weight cycling periods, had significantly higher basal plasma insulin values (after final refeeding period), although plasma glucose concentrations were similar to the control group. It should be noted that retroperitoneal fat pad weights and cell number accounted for 30% of the variance found in the insulin values. The long term effects on insulin levels were not looked at in either study, and therefore it remains unknown if this change was permanent or temporary.

In contrast Harris et al[40] found that female adult rats restricted for 22 days and refed for 20 days ad libitum, showed no increase in insulin values with refeeding.

McCargar et al[63] found that food restriction for 4 days (1000kcal), followed by over eating for 2 days (3000 kcal), produced a greater than normal postprandial rise in serum insulin in normal weight females. The fasting insulin values however were close to control values. The overfeeding of a diet high in CHO may have stimulated such an increase in insulin concentration. Once again, it was not determined if the increased in postprandial rise in serum insulin was a temporary or permanent change.

These experiments may suggest that bouts of food restriction followed by refeeding may lead to elevated insulin levels, especially if overeating occurs. This could have important implications on lipid (adipocyte) metabolism, with enhanced lipogenesis, decreased lipolysis, and thus increased fat deposition at the time of refeeding. Whether insulin levels remain elevated has yet to be determined. Owens et al[64]

found that fasting for 2 days and refeeding for 20 days produced a lasting effect on basal and insulin stimulated metabolism of glucose in rats. Insulin stimulated glucose metabolism increased with refeeding. Although all three products of glucose metabolism were increased, the effect on fatty acid synthesis was the greatest, followed by glucose oxidation and glyceride-glycerol synthesis. This started on day three of refeeding and continued to increase to day 10. After this point it gradually returned to normal by day 20. They concluded that fasting followed by refeeding enhanced lipogenesis and insulin stimulation of glucose metabolism. These changes in adipocyte metabolism are prolonged beyond the restoration of size and adipose mass to prefasting conditions. These changes in insulin and glucose metabolism may help to explain an increase in adiposity found in weight cyclers.

1.4.2 THYROID HORMONES

Changes in thyroid hormone levels may influence a reduction in resting metabolic rate as a result of weight cycling. Thyroid hormones play a role in metabolic regulation and have a calorigenic effect in a variety of tissues through the expression of membrane sodium-potassium (Na+-K+), adenosine triphosphatase (ATPase) and several mitochondrial enzymes.[65] The thyroid gland secretes thyroxine (T_4) and a very small amount of triiodothyronine (T_3), under the influence of the pituitary gland and thyroid stimulating hormone (TSH). The majority of T_3, which is the most active form of the hormone, is produced in peripheral tissues by the deiodination of T_4. A small amount of T_4 is also converted to

reverse triiodothyronine (rT_3), which is metabolically inactive. Within tissue cells, T_3 mediates its action by interacting with specific nuclear receptors, there by inducing specific m-RNAs for gene expression of such proteins as mitochondrial respiratory enzymes. It is through this process that T_3 has its calorigenic effect,[66] stimulating oxidative metabolism and boosting BMR.

Several studies have suggested that fasting and severe caloric restriction will result in a decrease of both free and total serum T_3, which is sometimes accompanied by an increase in rT_3. T_4 levels are virtually unchanged with any degree of calorie restriction.[7] This may have important implications with respect to depressed RMR which often accompanies food deprivation. Barrows et al[7] conducted a study where subjects consumed a very low calorie diet (VLCD) (420 kcal/day) for 4-6 months followed by a 5 week refeeding period. T_3 levels decreased significantly within the first 5 weeks of calorie restriction and remained depressed through the remainder of the diet. This depression paralleled the decrease in BMR and decreased rate of weight loss. Upon refeeding T_3 levels increased but did not reach baseline values. Reverse triiodothyronine increased in the first 5 weeks, but dropped again by 10 weeks and returned to baseline values with refeeding. Total and free T_4 levels remained unchanged. Barrows et al[7] concluded that caloric restriction resulted in decreased T_3 levels. It is possible that such a decline is an adaptation which hinders weight loss, acting as a protective mechanism against famine and may be linked to the diet-induced decrease in BMR that is often observed.

Jung et al[67] also found a significant decline in T_3 and a significant increase in rT_3 when obese women were placed on a low energy diet (9.2kcal/kg desirable weight) for 21 days. Upon refeeding with a high energy weight maintaining diet (40 kcal/kg), T_3 had significantly increased within 72 hours, but did not reach predetermined baseline values (measured when subjects consumed the high energy diet for one week prior to restriction diet). The rT_3 had just begun to decline by 72 hours. Carbohydrate (CHO) content of the diet may have been an important factor in the depressed T_3 values, as energy restriction for the low energy diet was achieved by decreasing the carbohydrate content.[67] Numerous studies have suggested such an effect. This however does not explain why T_3 does not return to baseline on a refeeding diet containing sufficient CHO.

Spaulding et al[65] postulated that carbohydrate was an important factor which regulated the production of T_3. It was found that with complete fasting, serum T_3 was reduced by 53%, and by 47% on a hypocaloric diet of 800 kcal containing no carbohydrate. There was however, no change in T_3 levels with an 800 kcal isocaloric diet containing 50g CHO. Other studies have shown that overfeeding with CHO in normal subjects produced increased serum T_3 levels.[68] Azizi[69] also found that following caloric restriction, depressed T_3 levels would return to normal if refeeding consisted of a CHO or mixed diet, but not if the diet contained only protein and lipid. T_3 production therefore appears to be altered by CHO content of a diet, although the mechanism of its influence is unclear.

Reverse triiodothyronine did not follow the same trend. Reverse triiodothyronine levels did increase by 58% with total fasting, but with both 800 kcal diets (100% CHO and no CHO) there was no significant increase in rT_3.[65] It would appear that rT_3 production is more likely dependent on the severity of caloric deprivation, rather than CHO content.

MECHANISMS

Two hypotheses have been suggested to explain how food restriction may alter normal T_3 activity. Firstly, there may be a decline in T_3 production. This is a result of reduced transmembrane transport and therefore availability of T_4 within the cell, for T_3 conversion. Secondly the number of nuclear receptors which can bind T_3 may be diminished.

The depression in T_3 with caloric deprivation may result from a decreased production of the hormone. Vagenakis et al[70] determined the T_4 and T_3 production and metabolic clearance rates(MCR) in obese patients undergoing prolonged starvation. They found that T_3 MCR was unchanged, thus indicating that T_3 production rates must be substantially reduced, to account for depressed serum values. These findings were consistent with in-vitro studies of the livers of starved rats, where T_4 to T_3 conversion was in fact decreased.

Reduction in T_3 production was first thought to be a result of an altered pathway for conversion of T_4 to T_3, diverting conversion to rT_3 production instead. This may be the case, as rT_3 levels have been found

to increase in parallel with a T_3 decline. However more recent studies did not show a similar relationship between the two hormones, and rT_3 showed variable responses to food deprivation.

Possible mechanisms for decreased T_3 and altered rT_3 production have been postulated. It has been shown from liver homogenates of fasted rats that 5'-deiodinase enzyme activity is decreased, resulting in decreased T_3 formation and diminished rT_3 breakdown.[71] Further study indicated that the transmembrane transport of T_4, T_3 and rT_3, and mass transfer rates of T_4 and T_3 into tissue pools in humans were altered.[72] Triiodothyronine had a 50% decrease in mass transport of which only partially could be accounted for by the decreased T_3 production. There was an even more pronounced inhibition of T_4 transport into tissues resulting in less T_4 within tissues. As a result of this decreased transmembrane transport of T_4, there was a diminished 5'-deiodinase enzyme occupation and thus decreased activity level.

The cause of the transport inhibition is speculative, with the possible suggestion that intracellular energy charge may be low with caloric restriction, as is seen in profused fasted rat livers. T_4, T_3, and rT_3 transport, although by two different pathways, both require energy (active transport mechanism). Another possibility is a decrease in Na+-K+ ATPase activity, which is caused by diet manipulation and can lead to attenuation of membrane fluidity.[72]

Another factor affecting thyroid hormone function, is a decrease in the number of nuclear T_3 receptors, which has been shown to result from prolonged energy restriction.[73,74] Moore et al[74] found that obese

patients on 1.34 MJ/day, for 12 weeks, showed a significant drop in rate of weight loss, RMR, serum T_3 and the number of T_3 receptors on peripheral lymphocytes. Since T_3-nuclear receptor complex is required for the hormonal effect of T_3, this has strong implications on T_3 function. Schussler et al[75] determined that fasted Spraque-Dawley rats had a serum T_3 drop of 50% and that receptors had decreased more so than serum T_3. Nuclear receptor affinity towards T_3 was not found to be altered. It was concluded that T_3 serum concentration was not the cause of diminished hepatic nuclear receptor content, but rather had an independent synergistic effect of fasting.

Both the decrease in T_3 serum concentrations and a decrease in the number of nuclear receptors may be important adaptive mechanisms during food deprivation, and could be strongly associated with the depressed RMR demonstrated in such a situation. Repeated cycles of weight loss and regain may result in a prolonged or permanent alteration in T_3 serum values and nuclear receptor numbers, which could have serious long term consequences on metabolic rate. If such adaptations were to occur, it may help explain the depression in resting energy expenditure which is experienced by some weight cyclers.

1.4.3 SUMMARY OF BIOCHEMICAL PARAMETERS

Weight cycling may result in altered biochemical parameters such as fasting serum insulin, Glucose, and T_3. It is well documented that insulin, glucose and T_3 levels decrease with food restriction. What remains unknown however, is how these biochemical parameters respond to

repeated cycles of weight loss and weight regain. It has been speculated that there will be an increase in serum insulin and a decrease in serum T_3. Changes in these biochemical parameters may provide an explanation, even if only partially, to some of the changes that occur as a result of weight cycling. Accordingly, they were examined in this study.

1.5 HYPOTHESES

As described above, much remains to be learned about the metabolic and physiological effects of weight cycling. Competitive judokas, some of whom lose weight prior to each competition, may be a good model for studying these effects. Accordingly, in this study competitive judokas, half of whom were weight cyclers, were followed for a period of 10 months. The study period included three test sessions. The first test session was conducted pre-season, when the judokas were training, but not yet dieting for competitions. The second test session was during the peak season. At this point the judokas had been competing and therefore cutting weight for competitions. The third and final test session was during the off-season, when the judokas were no longer training for judo or dieting for competitions. The null hypotheses were as follows:

1) There will be no difference in resting metabolic rate when the weight cycling judokas are compared to the non-weight cycling judokas at any of the three time periods.

2) There will be no difference in body composition, specifically percentage body fat, lean tissue, and waist to hip ratio when the weight cycling judokas are compared to the non-weight cycling judokas at any of the three time periods.

3) There will be no difference in fasted serum insulin, glucose or triiodothyronine when the weight cycling judokas are compared to the non-weight cycling judokas at any of the three time periods.

4) There will be no difference in diet composition, specifically proportions and amounts of macronutrients consumed and total calories consumed at any of the three time periods when the weight cycling judokas are compared to the non-weight cycling judokas.

1.6 SPECIFIC AIMS

The purpose of this research project is to determine what effect weight cycling in male competitive judokas has on the following parameters, during pre-season, peak season and off-season, when compared to non-weight cycling judokas:

1) Energy metabolism: RMR
2) Body composition: body fat content and fat distribution
3) Biochemical indices: serum glucose, insulin, and triiodothyronine.
4) Dietary factors: macronutrient distribution and kilocalories consumed.

CHAPTER 2

METHODOLOGY

2.1 SUBJECT SELECTION

Subjects were recruited from the Men's Provincial Senior Judo Team and the UBC Judo Team. Approximately 40 individuals were approached, of whom only twenty subjects were chosen on the basis of previous dieting history, as determined by a questionnaire (appendix II). Those that were excluded did not cut sufficient weight to fit necessary criteria. Subjects who were defined as weight cyclers were to answer specific key questions in the following manner:

(a) frequency of dieting during competitive season?

often or always

(b) number of times weight is reduced in a given season?

≥ 3 times

(c) amount of weight lost during each dieting session?

≥ 4.0 kg

(d) number of years cutting weight for Judo?

≥ 2 years

Ten judokas matched these criteria.

Control subjects who did not weight cycle were to answer rarely or never to frequency of dieting during the competitive season, were to have cut weight no more than once for a competition the previous season, were

to cut less that 1.4 kg the previous season, and were to have had no history of cutting weight for judo. Ten judokas met these guidelines. They were matched as closely as possible to the weight cycling group with respect to age, height, weight, percentage body fat, lean body mass and activity level.

One subject from each group could not complete the study due to relocation from the Vancouver area. Subject #105 was unable to have blood taken, excluding himself and his matched weight cycler, #06, from the blood analysis. Subject #110 failed to complete any diet records, excluding himself and his matched weight cycler, #10, from the diet analysis. Therefore the total number of subjects in each group became nine.

2.2 EXPERIMENTAL DESIGN

All subjects reported to the UBC Sports Medicine Clinic or the Buchanan Sports Laboratory three times within a 10 month study period. Height, weight, girth and body composition measurements were taken, RMR was determined, and a fasting blood sample was taken at each of the three test periods.

The first test period (test session 1, pre-season) was at least three weeks into the training season, when the subjects were trained and in good physical condition, but had not yet started to compete or diet. This time period occurred from November 1990 to February of 1991. The second test period (test session 2, peak season) occurred during the judokas' peak competitive season, at least one week prior to their last

major competition. At this point they had been dieting intermittently throughout the season. The test was done prior to commencement of the last dieting episode required for the final competition. The span of this test period was from March 1991 to June 1991. The third test period (test session 3, off-season) was during the off season when the subjects were no longer competing and were therefore not dieting. This took place a minimum of three months following their last competition and last bout of caloric restriction. Testing during this period took place between July 1991 and October 1991. The judokas had variable competition schedules and season lengths, resulting in the large time span of the test sessions.

2.3 RESTING METABOLIC RATE

Resting metabolic rate (RMR) was measured by indirect calorimetry using the metabolic cart (Medical Graphics Corporation System 2001, Jostens Graphic Products, Chicago, IL) located in the UBC Sports Medicine Centre. Three subjects were tested at the Buchanan Sports Lab for all three test sessions due to shortage of time on the metabolic cart at the Sports Medicine Centre. All subjects reported to the centre early in the morning, after a 12 hour fast and having done no exercise the previous day. They were familiarized with the testing procedures and allowed to rest for half an hour in the supine position. When ready for testing, the subjects were instructed to breathe through a mouth piece and a nose clip was put in place. A relaxation tape was played to put subjects at ease. After an adjustment time of 5 minutes, RMR was measured for 20

minutes. Oxygen uptake and carbon dioxide production were determined, from which RMR and resting energy expenditure were derived using the Nutritional Analysis Program (appendix III).

Initially, an accurate test required the subject to be in a fasted state, to have not exercised for 24 hours, to have rested for half an hour lying down, and to have an RQ below 0.88. It was later found that the metabolic cart at the Sports Medicine Centre gave elevated RQ values due to elevated VCO_2 levels. VO_2 levels were found to be accurate (personal communication). As a result an RQ value of 0.88 was excluded as a criteria and a test was considered valid when the remaining three criterion were met. If the test was not valid it was repeated a week later. The same procedures were followed at the Buchanan Sports Lab, but utilizing a Beckman Metabolic Measurement Cart.

2.4 BODY COMPOSITION

Skinfold measurements were used for indirect determination of body composition. Studies have shown that skinfolds at four sites are of similar accuracy as bioelectrical impedance, and hydrodensitometry.[76,77] Brodie[76] et al found comparable results of body fat determination with all three procedures. It has been found that bioelectrical impedance is very sensitive to degree of hydration, and following a weight loss period, or a bout of strenuous exercise (when glycogen stores are depleted), change in fat free mass may be underestimated.[78] Although controversy remains as to the precision of skinfold measurements for prediction of percentage body fat, repeated measurements have been found

to be consistent when a single trained observer conducts them. This technique would therefore satisfy the requirements of this study, as it is the relative change in percentage body fat, and not absolute value, which was important. For this reason skinfolds at four different sites were used to calculate the percentage of body fat using the Durnin and Womersley regression equations.[79] Equations were specific for men and age categories. The four sites measured include the bicep, tricep, subscapular and supra-iliac areas. Measurements were taken as described in the Canadian Standardized Test of Fitness (appendix IV), using Lange Calipers (Cambridge Scientific Industries, Cambridge MD.). All measurements were taken on the non-dominant side of the body and were repeated twice. If the two measures were not within 0.4mm then a third measurement was taken. The two closest measurements were averaged and if they were equidistance apart, all three values were averaged. Subsequent skinfold tests were performed by the same individual in order to reduce the error caused by variability in technique. Percent body fat was determined using linear regression equations[79] which estimate body density:

$$\text{Density} = c - (m \times \log \text{skinfold})$$

c and m values are specific for gender and age group, and were obtained from tables in Durnin and Womersley[79] (appendix V). Log skinfold is the log of a sum of the four skinfolds. From body density, Siri's equation[80] was used to calculate percentage body fat:

% Fat = ((4.95/density - 4.5) x 100)

Fat free body weight was calculated by:

fat free body wt. = (present wt. - (present wt. x %fat/100))

Girth measurements were taken using a measuring tape. The waist and hip measurements (Appendix IV) were used to calculate waist to hip ratio.

2.5 BIOCHEMICAL MEASUREMENTS

A 5-10 ml blood sample was taken from a vein in the arm by a qualified technician. Samples were centrifuged at 3000 RPM for 15 minutes to separate the serum. The serum was then aliquoted and frozen (at-70 degrees C) for future analysis. T_3 was analyzed at the Department of Laboratory Medicine, in UBC Hospital. Samples were delivered for analysis in two batches; the first contained samples from test one and test two, and the second contained samples from test three. Insulin was analyzed at the Endocrinology Department, Children's Hospital, Vancouver,B.C.. Samples for insulin were also delivered in two batches as described above. Glucose was analyzed in the Division of Human Nutrition, at UBC, following each test session.

2.5.1 SERUM TRIIODOTHYRONINE

Total serum Triiodothyronine (T_3) from blood samples was determined by Microparticle Enzyme Immunoassay (MEIA), using an IMX System (IMx System, Abbott Diagnostics, Illinois).[81] For determination, a blood sample and Anti-T_3 Coated Microparticles are

placed in a reaction cell were T_3 binds the microparticles and an antibody-antigen complex is formed. The microparticles with bound antibody-antigen complexes are then transferred to a glass fiber matrix, where they bind irreversibly. Added T_3 Alkaline Phosphatase Conjugate then binds to available Anti-T_3 microparticle sites (not occupied by T_3) and unbound materials are subsequently washed away. When substrate, 4-Methylumbelliferyl Phosphate, is added to the matrix, a fluorescent product results and can be measured by the MEIA optical assembly of the IMx System. From this, the concentration of T_3 is calculated (nmols/L).

2.5.2 SERUM INSULIN DETERMINATION

Insulin determinations in collected blood samples were carried out using double antibody radioimmunoassay (RIA).[82] The insulin in the blood competes with a known amount of ^{125}I-labelled insulin for specific antibody sites. Bound and free insulin are then separated with the addition of a second antibody immunoabsorbent. Centrifugation produces a radioactive pellet which gives a reading inversely proportional to the insulin quantity in the blood sample.

2.5.3 SERUM GLUCOSE ANALYSIS

A Beckman Glucose Analyzer II (Beckman Instruments, Fullerton, CA) was used for blood glucose determinations.[83] Ten microliters of serum is placed in glucose oxidase enzyme, in the presence of oxygen. A reaction forming gluconic acid and peroxide proceeds

with a resultant depletion of oxygen that can be measured by an electrode. Oxygen consumption is directly proportional to blood glucose concentration and mg of glucose per 100ml of blood was determined.

2.6 DIET ANALYSIS

All subjects were asked to keep a dietary record for three days during each test period. They were thoroughly instructed on recording types and weights or volumes of all foods (in form eaten) and liquids consumed. One of the three days was a weekend day. NutriCom (NutriCom,Smart Engineering LTD., Vancouver,B.C.) was used for diet record analysis.

Three day food records were chosen, as they are believed to be as reliable in determining nutrient intake as seven day food records. Stuff[84] et al found good agreement between three day and seven day food records for estimating individual's nutrient intakes, and concluded that three day records were adequate for obtaining qualitative nutrient intake data. Tremblay[85] et al studied the reproducibility of three day food records and found moderate to high agreement between two consecutive records. They concluded that three day records provided a reliable estimate of intakes for almost all nutrients. Other studies have also found a length of three days to be a sufficient time period to obtain acceptable estimates of nutrient intakes, particularly energy and macronutrients (which are the main concerns of this study).[86] Basiotis et al[87] found that in order to estimate true average energy intake for a

group of males, 3 dyas were required. Other nutrients however, required greater than three days, and up to 39 days for vitamin A. It is conceivable that the 7 day food record accuracy may be compromised due to the length of recording time required. People may find it tedious, resulting in reduced recording precision or inhibition of food consumption, underestimating true intake.[84] For these reasons 3 day records were used.

The overall accuracy of both three day and seven day diet records must be considered. Other studies have indicated that numerous errors can occur in the process of diet analysis. There may be incomplete dietary recording, particularly with forgotten foods if recording is retrospective, and unrepresentative food selection.[88]

There may also be errors in coding and translation of foods into nutrients as well.[88] For this reason great care must be given in instructing subjects on food recording in order to reduce possible error and limitations must be realized to avoid inappropriate conclusions.

2.7 DATA ANALYSIS

Baseline subject characteristics were analyzed by independent t-tests to determine any significant differences. Changes in mean individual values for RMR, REE, fasted serum T_3, insulin, blood glucose, percentage body fat, waist to hip ratio, and energy intake for all three test periods were analyzed using an analysis of variance (ANOVA) for a 2 (experimental and control group) x 3 (3 test sessions) factorial experiment with repeated measures on the 2nd factor. Percentage body

fat, waist to hip ratio, % fat, % CHO, and % protein were arc sine transformed for statistical analysis. When applicable, TUKEY Post-Hoc analysis was performed to determine statistical differences between test periods. Results were presented as the mean ± S.D., with a level of significance of $p \leq 0.05$.

CHAPTER 3

RESULTS

3.1 SUBJECTS

3.1.1 IDENTIFICATION OF A WEIGHT CYCLER

The 9 judokas who met all criteria required for classification as a weight cycler were placed in the experimental group, and 9 judokas who met the specifications of a non-cycler were placed in the control group. Table 1 indicates how well both groups matched their respective inclusion criteria. The weight cyclers reported having lost an average of 4.0±0.9 kgs, 5.0±2.9 (mean±SD) times within the season. The control group reported having lost <0.15 kg (mean), 0.2±0.7 times (mean±SD) the previous season. Five men in the experimental group reported that they always cut weight for competitions, and the remaining four said they often cut weight. None of them indicated having never, rarely or sometimes cut weight. The control group reported having never (6/9) or rarely (3/9) cut weight. None of them indicated having cut weight sometimes, often, or always.

In regards to the history of weight loss (table 1), the experimental group had lost a significantly greater amount of weight ($p=0.00$), losing 5 kg, 11.0 ± 9.2 times in the last 5 years, whereas the control group had only lost 5 kg, 1± 1.5 times. The experimental group had been cutting weight for judo an average of 6.0 ± 5.0 years, whereas the control group had never cut weight for judo (significant at $p=0.00$).

TABLE 1: The criteria required for male judokas to be classified as a weight cycler or a non-cycler and the ratings achieved by each group.

CYCLER (n=9)

FACTOR	CRITERIA REQUIRED	CYCLER RATING
years in judo	≥ 2	8.0±6.1
years cutting weight for judo	≥ 2	5.9±5.3*
times cut in last 5 years**	-t	11.0±9.2*
# competitions last season	≥ 2	5.0±2.0*
# times weight cut last season	≥ 3	5.0±2.9*
ave. kgs lost per competition	≥ 4	4.0±1.0*

NON-CYCLER (n=9)

FACTOR	CRITERIA REQUIRED	NON-CYCLER RATING
years in judo	≥ 2	6.5±5.1
years cutting weight for judo	0	0.0±0.0*
times cut in last 5 years**	-t	1.0±1.5*
# competitions last season	≥ 2	2.5±1.3*
# times weight cut last season	≤ 1	0.0±0.0*
ave. kgs lost per competition	≤ 1.4	0.0±0.0*

mean±SD

* significant difference between weight cyclers and non weight cyclers for given parameter at $p \leq 0.05$

** the number of times greater than 5 kg were cut in the last five years

t no criteria set

3.1.2 SUBJECT MATCHING

The experimental group was not significantly different from the control group with respect to weight (kg), percentage fat, lean body mass (kg), height (cm), and age (years)(table 2) at baseline. There was also no significant difference in hours of moderate to intense levels of exercise at baseline or any other test session, as indicated by self reported weekly records (8.6±5.9 and 7.5±5.3 for weight cyclers and non-cyclers respectively). Table 3 shows the mean differences between the experimental group and their matched controls. It should be noted that the weight cycling group had competed more frequently than the control group during the past season.

3.1.3 DIETING HABITS AND WEIGHT LOSS DURING THE SEASON

Throughout the season the experimental group lost significantly more weight (4.1 ± 1.5 kg)(p=0.00), a significantly greater number of times (4.2 ±2.7 times) (p=0.00) than did the control group (no weight was cut for0 any competitions), as indicated by self reported weight cutting episodes. Examination of the difference between the competitive weights and weight recorded during the second test session (peak season) verifies that approximately 4 kgs were lost for competitions. The maximum weight allowable in the competitive weight category for weight cyclers was an average of 77.1±11.9 (SD), with an average weight during the peak season of 81.3±11.8. The non-cyclers could not exceed an average weight of 87.7±16.9 to meet their weight class. They had an average weight of 86.1±13.1 (mean±SD) during the peak season. Each group participated in

TABLE 2: Physical characteristics of weight cycling and non-weight cycling male competitive judokas for the pre-season, baseline test.

WEIGHT CYCLING JUDOKAS (n=9)					
SUBJECT NUMBER	AGE (yrs)	HEIGHT (cm)	WEIGHT (kg)	LBM* (kg)	%FAT (%)
06	28	188	108.3	86.9	19.8
05	20	172	70.1	63.0	10.1
14	27	169	66.2	59.7	9.0
10	25	178	83.0	68.1	18.0
13	23	181	81.0	72.0	11.3
07	31	165	73.3	61.1	16.7
11	35	172	85.6	68.1	20.4
12	19	178	87.2	70.6	19.0
15	20	176	81.0	64.2	20.8
MEAN	25	175	81.8	68.2	16.2
SD	7.8	6.9	12.3	8.2	4.5
$P_{0.05}$	NS	NS	NS	NS	NS
NON-WEIGHT CYCLING JUDOKAS (n=9)					
CODE	AGE (yrs)	HEIGHT (cm)	WEIGHT (kg)	LBM* (kg)	%FAT (%)
105	26	203	103.0	89.9	12.7
102	25	168	76.8	67.3	12.5
101	22	168	64.6	58.7	9.1
110	25	171	83.3	68.1	18.0
103	34	189	86.6	75.0	13.4
109	24	179	73.2	64.0	12.0
106	30	187	92.4	74.2	19.7
108	22	195	99.8	82.3	17.5
111	19	175	86.5	73.6	14.9
MEAN	25	182	85.1	72.6	14.4
SD	4.5	12.6	12.4	9.5	3.4
$P_{0.05}$	NS	NS	NS	NS	NS

* LBM = lean body mass in kgs = [body weight(kg) - (body weight(kg) x % fat/100)]

TABLE 3: Mean differences between matched weight cycling and non-weight cycling judokass for the pre-season values (test session 1)

CHARACTERISTIC	MEAN DIFFERENCES* (±SD)
Age (yrs)	4.3±3.4
Weight (kg)	4.9±3.9
Height (cm)	9.2±6.4
exercise (hrs/wk)	4.9±6.8
% fat (%)	3.1±3.1
LBM(kg)**	2.5±2.4

- n = 9 in each weight cycling and non-weight cycling group

* mean differences between matched weight-cyclers and non-weight cyclers

** LBM = lean body mass (kg) = [body weight(kg) - (body weight(kg) x % fat/100)]

an average of 5.2±2.4 and 3.0±1.3 competitions respectively. The average body weight did however, show no significant change over time (ie. during the competitive season) as a result of weight cycling. This is due to the fact that the second test was conducted one week before the last major competition requiring weight reduction, and it was prior to the dieting episode required for the competition.

3.2 METABOLIC PARAMETERS

During REE determinations, the average RQ for the weight cyclers was 0.86±0.07 and 0.88±0.07 for the non-cyclers, over the three test periods. Table 4 shows the absolute and relative REE values for both experimental and control groups. There was no significant main effect of group (weight cyclers versus non-weight cyclers) ($F=0.16, p=0.68$) and there was no significant main effect of time (time 1 vs time 2 vs time 3)($F=0.49, p=0.62$) for absolute REE. There was also no group by time interaction ($F=0.26, p=0.77$). When REE was expressed relative to lean body mass, body weight, or surface area there was still no main effect of group ($F=0.12, p=0.73$; $F=0.06, p=0.82$; $F=0.10, p=0.76$ respectively), of time ($F=0.64, p=0.54$; $F=0.22, p=0.80$; $F=0.47, p=0.63$ respectively) or a group by time interaction ($F=0.20, p=0.82$; $F=0.08, p=0.92$; $F=0.35, p=0.70$ respectively). Therefore the experimental group did not demonstrate respectively). Therefore the experimental group did not demonstrate a difference in absolute or relative resting metabolism at any of the three times when compared to the control group. There did not appear to be any metabolic consequences due to previous seasons of weight cycling, during

TABLE 4: The absolute and relative resting energy expenditures (REE) of weight cycling and non-weight cycling judokas at pre-, peak, and off-season.

WEIGHT CYCLING JUDOKAS (n=9)			
METABOLIC PARAMETERS	TEST SESSIONS 1 (pre-season)	2 (peak season)	3 (off-season)
REE*(kcal/day)	1610.5 ± 306.0	1602.7 ± 408.1	1622.9 ± 0365.2
REE*/Kg(kcal/kg)	19.7 ± 2.6	19.9 ± 5.3	20.0 ± 4.1
REE*/LBM**(kcal/LBM)	23.5 ± 2.8	23.3 ± 5.2	23.9 ± 4.3
RMR^{t}(Kcal/m^2/hr)	34.0 ± 4.6	33.9 ± 8.2	34.1 ± 6.7
NON-WEIGHT CYCLING JUDOKAS (n=9)			
METABOLIC PARAMETER	TEST SESSION 1 (pre-season)	2 (peak season)	3 (off-season)
REE*(kcal/day)	1625.9 ± 204.0	1644.6 ± 276.1	1728.1 ± 278.2
REE*/Kg(kcal/kg)	19.3 ± 2.7	19.3 ± 3.0	20.0 ± 1.5
REE*/LBM**(kcal/LBM)	22.6 ± 3.0	22.8 ± 3.1	24.0 ± 2.0
RMR^{t}(Kcal/m^2/hr)	32.7 ± 4.9	32.6 ± 4.7	34.7 ± 3.6

\- non-significant at p value ≥ 0.05
* REE = resting energy expenditure in kilocalories
** LBM = lean body mass = [body weight(kg) - (body weight(kg) x % fat/100)]
t RMR = resting metabolic rate in kilocalories

a season of cutting weight or following a season of cutting weight, in comparison to the non-weight cycling group.

3.3 ANTHROPOMETRICS

There was no significant main effect of group ($F=0.57, p=0.46$), main effect of time ($F=0.67, p=0.52$), or group by time interaction ($F=1.40, p=0.26$) for body weight (table 5), indicating that body weight of the weight cycling group remained the same as that of the non-weight cycling group during the second and third test sessions. Measurements during the second test session were taken one week before the last major competition requiring food restriction and weight loss. At this point the weight cyclers had not yet started to restrict food for the upcoming competition and were therefore not at their typical competing weight. For this reason the weight cyclers did not show a significant reduction in body weight during the competitive season, as may be expected. This demonstrated that following a weight cutting episode the weight cycling group regained weight back to pre-season, baseline values and reduced weight was only maintained for the duration of the competition. Furthermore when they were not cutting weight during the off season, they did not tend to increase their body weight, remaining the same asbaseline values and similar to their control group.

Analysis of body composition also demonstrated that there was no main effect of group, main effect of time or group by time interaction for percentage fat ($F=0.00, p=0.99$; $F=0.02, p=0.98$; $F=1.56, p=0.23$ respectively), or amount of LBM ($F=1.04, p=0.32$; $F=1.43, p=0.25$; $F=0.13$,

TABLE 5: Anthropometric measurements of weight cycling and non-weight cycling judokas during pre-, peak, and off-season.

WEIGHT CYCLING JUDOKAS (n=9)			
ANTROPOMETRIC PARAMETERS	TEST SESSIONS 1 (pre-season)	2 (peak season)	3 (off-season)
weight(kg)	81.8±12.3	81.3±11.8	81.6±10.8
% fat(%)	16.2±4.5	15.5±4.6	14.6±6.5
LBM(kg)*	68.2±8.2	68.5±8.2	67.5±7.9
WHR**	0.87±0.05	0.88±0.04	0.87±0.05
NON-WEIGHT CYCLING JUDOKAS (n=9)			
ANTHROPOMETRIC PARAMETER	TEST SESSION 1 (pre-season)	2 (peak season)	3 (off-season)
weight(kg)	85.1+12.4	86.1+13.1	86.5+13.9
% FAT(%)	14.4±3.4	15.5±3.0	16.5±3.4
LBM(kg)*	72.6±9.4	72.6±10.2	72.1±10.9
WHR**	0.85±0.06	0.85±0.04	0.86±0.05

* LBM = lean body mass(kg)=[body weight(kg)-(body weight (kg) - % fat/100)]

** WHR = waist to hip ratio = waist(cm)/hip(cm)

- non-significant at $P_{0.05}$ for all parameters over time

p=0.88 respectively) (table 5). The weight cycling group experienced no increase in body fat or loss of LBM as a result of repeated dieting and they were not significantly different than the non-cyclers. Because they were matched in the beginning of the experiment it was not possible to determine if they had experienced previous changes in body composition as a result of a history of weight cycling.

There was no redistribution of body fat, as no significant main effect of group (F=0.88,p=0.36), main effect of time (F=0.01,p=0.99), or group by time interaction (F=0.55,p=0.58) was found for the waist to hip ratio. The weight-cycling judokas experienced no redistribution of body fat as a result of cutting weight and their WHR remained the same as the non-cycling judokas through a season of cutting weight.

3.4 BIOCHEMICAL PARAMETERS

Fasting blood samples showed no significant main effect of group (F=0.38,p=0.38), or group by time interaction (F=1.22,p=0.31) for T_3 values and they were all within the normal range (table 6). The T_3 value of the weight cycling group had been the same as that of the non-weight cycling group at baseline, and throughout the study, experiencing no change as a result of weight cycling at any time. There was however a main effect of time (F=43.16,p=0.00). A Tukey Post Hoc revealed a significant decrease at time two (p=0.00) and time three (p=0.00) when compared to time one. Both groups experienced this drop, and it was sustained during the off season test session.

Insulin and glucose values demonstrated no significant main effect

TABLE 6: Fasted serum values for insulin, glucose, and triiodothyronine in weight cycling and non-weight cycling judokas at pre-, peak, and off-season.

WEIGHT CYCLING JUDOKAS (n=8)*			
BLOOD PARAMETERS	TEST SESSIONS 1 (pre-season)	2 (peak season)	3 (off-season)
Triiodothyronine (umol/L)	2.7±0.4[a]	2.0±0.3[b]	2.0±0.3[b]
Insulin (pmol/L)	39.1±23.6	51.6±23.4	52.1±15.7
Glucose (nmol/dL)	4.4±0.2	4.3±0.3	4.0±0.6

NON-WEIGHT CYCLING JUDOKAS (n=8)*			
BLOOD PARAMETER	TEST SESSION 1	2	3
Thyroid Hormone (nmol/L)	2.7±0.2[a]	2.1±0.2[b]	2.2±0.3[b]
Insulin (pmol/L)	41.0±16.0	46.1±15.4	38.3±13.2
Glucose (nmol/L)	4.3±0.5	4.3±0.3	4.2±0.3

* n is reduced from 9 to 8 do to the inability of subject 105 to give blood, thus disqualifying his matched control, 06
- non-significant at $p \leq 0.05$ unless marked a or b
- a is significantly different than b at p=0.00
- same letter superscript means non-significant

of group (F=0.39,p=0.54; F=0.34,p=0.57 respectively), main effect of time (F=2.03,p=0.15; F=3.01,0.07 respectively) or group by time interaction (F=0.96,p=0.40; F=0.59,p=0.56). Values were similar and within normal range at baseline and did not change over time for both weight cycling and non-weight cycling groups.

3.5 DIET

Table 7 shows the average energy intake and proportions of macronutrients consumed. There was no significant main effect of group (F=1.06,p=0.32), main effect of time (F=0.53,p=0.60) or group by time interaction (F=0.05,p=0.95) for energy intake. The weight cycling group was not consuming fewer kilocalories due to a history of weight cycling when compared to the non-cycling group. Nor were they consuming fewer kilocalories during the competitive season (in between the periods of food restriction for making weight) or following a season of cutting weight.

Table 8 shows the predicted kilocalories required per day (estimated by the Harris-Benedict equation and an activity level of 1.5), the estimated number of kilocalories required (as determined by REE and an activity level of 1.5) and the estimated number of kilocalories that were being consumed, at all three test periods. The activity level of 1.5 was selected according to the British Columbia Diet Manual[89] which states BEE multiplied by 1.5 for weight maintenance and heavy activity. Mahalko et al[90] also indicated an activity level of 1.5 for moderate activity. Considering that the judokas were only required to attend

TABLE 7: Macronutrient and energy consumption, as determined by three day food records for weight cycling and non-weight cycling judokas at pre-, peak, and off-season.

WEIGHT CYCLING JUDOKAS (n=8)			
DIET PARAMETERS	TEST SESSIONS 1 (pre-season)	2 (peak season)	3 (off-season)
energy (kcal)	2137±608	2444±1154	2270±1219
energy/kg*	27.1±6.6	30.9±13.8	27.8±13.3
%**CHO^{t}	47.6±12.0	48.6±10.7	42.9±11.0
% protein	17.4±4.4	18.1±6.0	21.0±10.0
% fat	32.5±10.5	32.1±6.4	35.6±10.5
grams$^{\$}$of CHO^{t}	245.8±98.4	298.5±168	238.4±121.8
grams of protein	91.0±33.6	99.3±47.7	98.1±46.7
grams of fat	70.0±29.1	88.0±44.4	85.7±44.6
NON-WEIGHT CYCLING JUDOKAS (n=8)			
DIET PARAMETERS	TEST SESSION 1	2	3
energy (kcal)	2644±590	2773±798	2648±352
energy/kg	31.6±8.8	32.7±10.7	30.9±4.6
%**CHO^{t}	52.9±7.2	49.8±7.0	47.6±5.0
% protein	15.3±2.1	17.5±4.0	18.4±2.7
% fat	31.7±5.5	33.0±5.5	31.4±2.8
grams$^{\$}$of CHO^{t}	342.8±84.5	321.5±97.0	307.9±37.2
grams of protein	105.0±30.9	116.9±40.0	116.5±28.9
grams of fat	93.3±28.8	96.9±35.2	91.3±20.3

* kilocalories consumed per kg body weight
** percentage of daily energy intake as the specified macronutrient
^{t} CHO = carbohydrate
$^{\$}$ grams of specified macronutrient consumed per day
\- non-significant at $p \leq 0.05$

TABLE 8: Predicted energy requirements, estimated energy requirements and estimated energy consumption of weight cycling and non-weight cycling judokas at pre-,peak, and off-season.

WEIGHT CYCLING JUDOKAS (n=8)*			
PARAMETER	TEST SESSIONS 1 (pre-season)	2 (peak season)	3 (off-season)
A predicted** requirement	2837±297	2827±300	2833±179
B actual[t] requirement	2415±459	2403±612	2434±548
C actual[§] consumption	2137±608	2444±1154	2270±1219
A vs B (p value)	0.04	0.04	0.02
A vs C (p value)	0.02	0.40	0.20
B vs C (p value)	0.12	0.70	0.76
NON-WEIGHT CYCLING JUDOKAS (n=8)*			
PARAMETER	TEST SESSION 1 (pre-season)	2 (peak season)	3 (off-season)
D predicted** requirement	2939±339	2958±354	2967±372
E actual[t] requirement	2439±306	2466±414	2592±417
F actual[§] consumption	2644±590	2773±798	2648±352
D vs E (p value)	0.002	0.006	0.001
D vs F (p value)	0.24	0.50	0.03
E vs F (p value)	0.37	0.32	0.80

* n is reduced from 9 to 8 matched subjects due to 1 missing diet record

** the predicted caloric (kilocalorie) requirement as determined by the Harris-Benedict equation and an activity level of 1.5.

t the actual caloric (kilocalorie) requirement as determined by REE determination and an activity level of 1.5.

§ the actual caloric consumption (kilocalorie) as determined by three day food records.

practice three days a week, the remainder of their activity would likely fall within the moderate to sedentary range, particularily for the students. All judokas required significantly fewer kilocalories than were predicted by the equation at all three test times ($p=0.04$, $p=0.04$, and $p=0.02$ for weight cyclers at test 1, test 2, and test 3 respectively; $p=0.002$, $p=0.006$, and $p=0.001$ for the non-cyclers at test 1, 2, and 3). All groups showed no significant difference (at $p\leq0.05$) between estimated daily requirement (as determined by experimental REE and a 1.5 activity level) and estimated daily caloric intake (determined by diet analysis)(table 8). When comparing the actual consumption to the actual requirement, only test session 1 for the weight cyclers and test session 3 for the non-cyclers indicated significant differences (table 8).

There was no main effect of group, main effect of time or group by time interaction for the percentages of CHO ($F=1.09,p=0.32$; $F=2.78,p=0.08$; $F=0.10,p=0.91$ respectively), fat ($F=0.22,p=0.65$; $F=0.23,p=0.80$; $F=0.79,p=0.47$) and protein ($F=1.04,p=0.33$; $F=2.96,p=0.07$; $F=0.12,p=0.89$) consumed. There was no main effect of group, main effect of time or group by time interaction for the grams of CHO ($F=1.83,p=0.20$; $F=0.77,p=0.47$; $F=0.53,p=0.59$), fat ($F=0.66,p=0.43$; $F=0.76,p=0.48$; $F=0.56,p=0.58$) and protein ($F=0.86,p=0.37$; $F=1.35,p=0.28$; $F=0.09,p=0.92$) consumed. There was no change in the composition of the diet consumed as a result of a history of weight cycling or during or following a season of weight cycling.

CHAPTER 4

DISCUSSION

It has been suggested that repeated cycles of weight loss and regain will lead to physiological changes that can make subsequent weight loss difficult and weight regain rapid. Male judokas who had a history of cutting weight to meet specific weight categories were followed for 10 months (including one competitive season) to examine such physiological effects.

4.1 METABOLIC PARAMETERS

4.1.1 HISTORY OF WEIGHT CYCLING: BASELINE VALUES

At baseline the judokas with a history of weight cycling showed no significant reduction in absolute or relative REE (REE/kg, REE/LBM, or REE/m^2) when compared to judokas with no history of dieting.

This is in contrast to a study on adolescent wrestlers which demonstrated that a history of cutting weight was associated with a lower absolute and relative REE (REE/LBM)[35] when compared to non dieting wrestlers. Both the study by Steen et al[35] and the present study had well matched control groups who participated in a similar exercise schedule. Discrepancies in RMR findings may be due to differences in weight cutting habits, such as the number of times that weight was lost and the amount of weight that was lost. The judokas did not compete as often as the wrestlers, only having cut weight an average of 5.1±2.1 times in the previous season. The wrestlers in Steen and coworkers'

study had previously cut weight an average of 18.7 times. Furthermore the wrestlers had cut greater than or equal to 4.5 kg, with 64.3% cutting 5.9-6.8 kg and 14% cutting even greater than 6.8 kg. The judokas only cut an estimated 4 kg for each competition the season prior to the study. The number of years of cutting weight may also be significant, however this was not reported in Steen's[35] study and thus can not be compared.

A more recent study found that collegiate wrestlers with a history of weight cycling actually had a significantly higher RMR, when compared to a physically active, non-cycling matched control group.[36] The unexpected results of their study may be due to the fact that the two groups were not matched for type of exercise. It has been found that high intensity exercise carried out for a long duration, such as that experienced by the wrestlers (approx 2 hours per session) may increase RMR. On the other hand a shorter duration of moderately intense exercise (such as the 30-60 minutes in the physically active controls) may not affect RMR.[91] This could explain the different findings when compared to Steen's group and the current judoka study.

All judokas had an estimated REE that was significantly below predicted values (Harris-Benedict Equation) at baseline ($p=0.00$)(85% and 83% for weight cyclers and non-cyclers respectively). Although it has been suggested elsewhere[92] that this equation overestimates REE, these values are much lower than those demonstrated by other athletes, such as Melby's physically active controls (4% below predicted), their weight cyclers (112% max and 96% min) and Steen's non-cycling wrestlers (also 4% below predicted), using the same equation. In fact the values

obtained for all judokas were in line with the reduced REE experienced by Steen's weight cycling wrestlers (85% of predicted).

4.1.2 A SEASON OF WEIGHT CYCLING

During a season of cutting weight there was no decrease in absolute or relative RMR. Off season values also remained constant, despite a reduction or cessation of training for judo. Some studies have suggested that weight cycling leads to increased metabolic efficiency and decreased REE.[4,25,29,32,33,35] It is difficult however, to compare male judokas to animal studies and different human populations, such as obese individuals, lean individuals, females and even lean-non active males, due to the possibility of wide variations in metabolic function.

The only other longitudinal study recorded in the literature, which looked at male collegiate wrestlers, found no detrimental decrease in RMR during or following a season of cutting weight.[36] These findings concur with the findings of the judoka study, however there were some significant differences. Firstly, the wrestlers in Melby's study in fact had a significantly greater absolute and relative RMR (11% above the value predicted by Harris-Bennedict Equation) at baseline, when compared to a non-cycling physically active control group (4% below predicted). In the present study, both weight cycling and non-weight cycling judokas had a similar RMR, 85% and 83% of the predicted value respectively.

As previously mentioned, this may be explained by differences in the exercise regime. The wrestlers trained for 2 hours per session, but the number of sessions per week is not mentioned. Because the judokas do

not compete as often (average 5 times a season) they may not have had as intense a training session. Practice sessions were conducted for 1-1 1/2 hours 3 times per week. They did engage in other forms of activity, although intensity is very subjective and may not have been accurately identified, making exact quantification difficult.

Secondly, during the competitive season the wrestlers' absolute and relative RMR which was measured in the food restricted state, but not yet dehydrated state, demonstrated a significant drop from their baseline values. Such a drop was not experienced by the judokas. This may be explained by differences in methodology between the two studies. In the judoka study, RMR was measured the week prior to the last major competition which required food restriction. The measurement was taken however, before the food restriction episode had begun. On the other hand, the wrestlers in Melby's study were measured during the actual process of cutting weight. It has already been established that RMR decreases when food restriction occurs.[7-13] The methodology used for the judoka study would indicate whether a reduced RMR due to a dieting episode was sustained when normal food intake resumed.

The most important finding of Melby's study[36] was that the RMR of the weight cycling wrestlers returned to baseline values, suggesting no sustained effect of cutting weight. The judoka study and Melby's study thus suggest the same outcome; there was no evidence of a sustained decrement in either absolute or relative REE as a result of repeated cycles of weight loss and weight regain. A follow-up study conducted by Melby's group further showed no effect of weight cycling on REE after two

years.[93]

4.1.3 REASONS FOR NO DIFFERENCES

NO WEIGHT CYCLING EFFECTS

There are several possibilities which may explain the lack of significant differences between the two groups. Firstly it is possible that there are, in fact, no effects of weight cycling. There have been studies conducted which show that weight cycling does not result in any change in metabolic rate.[30-33,36] There are however studies that do suggest physiological effects.[24,25,29,35]

NO DEFINITION FOR A WEIGHT CYCLER

An important element of all weight cycling studies which must be considered when comparing and interpreting data, is that there is a wide variation in the description of a weight cycler. As indicated by table 9, no two studies share the same definition. This inconsistency in defining weight cyclers along with the use of metabolically different populations could result in inappropriate application to other populations. Studies that show no metabolic consequences may not in fact represent a true weight cycling population.

This must be considered in the judoka study. It is possible that the duration of food restriction, degree of food restriction and number of times weight was cut were not sufficient in this study and in other studies (ie.Melby et al[36]) to show an effect. The Melby study[36] and the

TABLE 9: Definitions of weight cyclers from various weight cycling studies.

GROUP	DEFINITION	FINDING
Steen et al,[a] 1988 (wrestlers)	- lost wt > 10 times during a season - > 4.5 kg loss/wk - often of always cutting wt	- decreased RMR
Van Dale et al,[b] 1989 (obese women)	- frequently dieting - lost & gained ≥ to 10kg in past 5 yrs more than once - often or always cutting wt.	- no change in RMR
Blackburn et al,[b] 1989 (obese patients)	- wt. regain of a min. of 20% of cycle 1 loss during inter-diet period	- velocity of wt loss slower
Beason et al,[b] 1989 (obese patients)	- no definition given - dieted - refed - dieted	- no change in veloc. of wt loss
Melby et al,[b] 1990 (wrestlers)	- lost ≥ 4.5 kg at least 10 times a season - ≥ 3 seasons of cutting wt	- no effect on RMR
Jebb et al,[b] 1991 (obese females)	- lost ≥ 6 kg ≥ 2 times - self reported history of yoyo dieting	- no sustained drop in RMR
Manore et al,[a] 1991 (lean females)	- dieted ≥ 10 days, 4 times in last year - dieting was considered ≤ 1000 kcal/d below energy requirement for wt maint.	- no effect on RMR

a - indicates cross sectional studies
b - indicates longitudinal studies

judoka study had similar definitions of a weight cycler (except number of times weight was to be cut) and no significant results were found. The definition for weight cycling for wrestlers in Steen's study[35] was also similar but actual weight loss during the competitive season was more severe (≥ 5.9-6.8 kg and some greater that 6.8 kg). These weight cyclers did not represent the specified definition given, but rather more severe food restricters than the latter wrestler study and the judoka study (hence possible reason for significant findings). Furthermore the judokas cut weight half as many times as both wrestling populations, further increasing the possibility of insignificant findings.

A clear definition of a weight cycler is required before any solid conclusions will be reached. It is likely that this will not be one specific definition, but rather a wide range of definitions for each specific population. For example small amounts of weight lost and regained, several times in a year (Steen et al[35]) may be just as detrimental as one large weight loss and regain, once a year (Blackburn et al[29]). Degree of food restriction, composition of the diet, and the specific population must also be considered. Thus, it becomes evident why defining a weight cycler is such a difficult task and has not yet been accomplished.

METHOD OF WEIGHT LOSS

The method of weight loss, whether through food restriction, dehydration, or exercise, is also an important consideration in studies of weight cycling. Weight loss practices of wrestlers have been found to

be a combination of both dehydration and food restriction.[94] Steen et al[35] did not mention the wrestlers' method of weight loss. This could lead to erroneous identification of the actual weight lost as LBM and fat, if a large proportion was actually water. Melby et al[36] identified weight loss before dehydration. All of the judokas achieved their weight loss primarily by food restriction for an average of 8.1±6.9 days, as indicated by self reported records. Since both judoka groups had similar exercise regimes, and the non-cyclers remained weight stable, it is not likely that weight loss was achieved through exercise.

EXERCISE PREVENTS RMR DEPRESSION

It is possible that exercise played a protective role against a reduced RMR expected with weight cycling. It has been suggested that exercise may prevent the drop in RMR experienced with caloric restriction, although studies are controversial and inconclusive. When obese individuals engage in regular exercise while restricting food, exercise has been found to counteract the depression in RMR that often accompanies dieting.[95-97] Similar results were found in obese individuals who were weight cyclers.[31] Mole et al[97] however found that this protective effect was removed as soon as exercise ceased. In contrast Phinney[98] found that dieting with exercise had the exact opposite effect, decreasing RMR three fold below baseline values when compared to dieting alone (10% decreased RMR). He postulated that this was a protective mechanism to compensate for the additional negative energy balance.

Few studies on the effect of exercise on metabolic rate have been done in lean athletes. Steen et al[35] found no protective effect of exercise on depressed RMR in the adolescent wrestlers. Melby et al[36] also demonstrated that wrestlers cutting weight experienced a significant drop in RMR from their baseline (pre-dieting) values, despite an intense exercise schedule. This suggests no protective effect, although RMR values were only 4% below predicted values and did return to baseline when weight cycling ceased. Exercise may have resulted in, or helped REE bounce back to normal. It is possible that exercise protected the judokas from a drop in RMR due to caloric restriction.

Exercise may prevent a drop in RMR in two ways. It may lead to a short-term increase in RMR following activity and it may also increase RMR over the longer-term by maintaining or increasing LBM, which is a main determinant of RMR. Exercise may have protected against a drop in RMR in the judokas through both of these mechanisms. Effects of weight cycling on LBM will be discussed under anthropometrics.

EXERCISE INCREASES RMR

Exercise may prevent a drop in REE caused by energy restriction, but it can also increase REE above normal predicted values, in both dieting and non-dieting individuals. This effect must be considered as a possible confounding factor in metabolic determinations. Numerous studies have shown that highly trained athletes have a significantly increased REE (absolute and per LBM) when compared to sedentary individuals.[91,99-101] This is controversial however, as some have in

fact found REE to decrease with training.[102]

The type, duration, and intensity of exercise has important implications on the effect exercise has on RMR. Intense exercise (11-16 hours per week) has been shown to increase RMR whereas moderate exercise (6-10 hours per week) has no effect.[91] This phenomenon was suggested in pre-season (baseline) values of collegiate wrestlers (Melby et al) who exhibited a RMR significantly higher (11% higher) than predicted values and 15% higher than their physically active, matched controls. The discrepancy in RMR's may be explained by differences in training schedules. The wrestlers trained for a longer duration of time (2 hrs per session), whereas the control group only exercised between 30 and 60 minutes per session three times per week.

The hours of moderate to intense exercise between the weight cycling and non-cycling judokas was not significantly different, averaging 8.6±5.9 and 7.5±5.3 hours per week respectively. Exercise was recorded on a weekly basis and duration of each exercise episode was not identified. Furthermore, judokas only indicated whether exercise was moderate or intense, which may be too subjective to obtain quantitative data. As a result it is difficult to interpret the impact exercise may have had on RMR.

It should be noted also, that 10 hours a week, which is considered moderate exercise, has been found to have no prolonged effect on RMR.[91] The weight cycling and non-cycling judokas averaged 8.6±5.9 and 7.5±5.3 hours per week respectively. It has also been found that 90 minutes of exercise (stationary bike at 175 watts) the evening before, has no effect

on RMR the next morning.[103] Furthermore, an increased RMR due to exercise, was not sustained when the exercise was discontinued for three days.[104] The point at which RMR returned to normal was not known but it could be as soon as a day following the exercise. In the present study, all subjects were asked to refrain from any form of exercise for 24 hours before the test day. Furthermore, an RMR 15-16% below predicted values does not likely indicate an elevated RMR due to exercise. It is therefore unlikely that such an effect on RMR would significantly alter the results of this study.

Although there is evidence to support that there was no impact of exercise on RMR with the judokas, there is controversy in the literature regarding this subject. Discrepancies arise in the classification of athletes, classification of "trained state" and the lack of acknowledgement that there is a wide spectrum of trained athletes. The duration, type and intensity of training are all factors which must be considered in these studies, yet very few have controlled for all of them. This makes it very difficult to classify the judokas in the current study.

SMALL NUMBERS AND WIDE VARIABILITY

The number of subjects in the study was very small due to unavailability of weight cycling judokas. A small subject population with large individual variability may contribute to lack of significant differences in RMR. Large variabilities in RMR can be found due to different genetic and environmental influences. This is an issue in

numerous weight cycling studies. In the studies of wrestlers, Steen et al[35] had 13 matched cyclers and Melby[36] had 12 matched cyclers. Manore et al[33] had 11 non-obese weight cycling women, Jebb et al[32] had 11 obese cycling women and Beeson[30] had only 4 obese weight cycling women. Much larger numbers may be required in order to obtain consistent results.

Furthermore, if there is a small subgroup of individuals who are particularly susceptible to weight cycling, as has been suggested by Brownell et all[2] they will be missed in studies with a small sample size. Attempts must be made to identify those people who are specifically susceptible.

NON-RESTING ENERGY EXPENDITURE IS ALTERED

It is possible that adaptation to food restriction is not manifested in a reduced RMR but rather results in an alteration of non-resting energy expenditure. There may be a decrease in the thermogenic effect of food, the thermogenic effect of exercise, or both. Geissler et al[19] found that post obese subjects had a 15% decrease in 24 hour energy expenditure, when compared to lean counterparts, with no history of dieting. They found that energy cost of sleep (ie. no effect of food and exercise thermogenesis) in the post obese subjects could only account for one tenth of the 15% drop below controls. The remaining 90% of the 15% reduction in 24 hour energy expenditure occurred during the day, under varying exercise regimes. This indicated that differences were due to a reduction in thermogenesis, as well as BMR. Furthermore, an elevation in metabolic rate seen in the controls following an episode of exercise, did

not occur in the post-obese subjects. Manore et al[33] found that lean females had no change in RMR as a result of weight cycling, but the amount of energy expended for a given body weight (thus work load) was less than lean matched controls.

Evaluation of exercise and food thermogenesis in the judokas may have identified adaptations due to their weight cutting habits. It must also be considered that the judokas who weight cycle may have an inherently reduced thermogenesis which necessitated weight cycling.

4.1.4 THYROID HORMONE

In studies where the REE and REE/LBM have decreased due to repeated cycles of weight loss and regain, possible mechanisms for this decrease must be indicated. This study investigated the possibility of a reduction in T_3 to explain the depressed REE. No drop in REE was found and no change in T_3 as a result of weight cycling occurred. As with REE, more dramatic food restriction, for a longer period of time may be required to show a sustained effect on T_3. Studies which have demonstrated a sustained decrease in T_3 values have used very low calorie diet (VLCD) (420-600 kcal/day) for 21 days and up to 3-4 months.[7,67] A decreased RMR accompanied these decreases in T_3.[67]

The significant drop in T_3 values which occurred for both groups from pre-season to peak season and off-season must be explained by something other than weight cycling. Seasonal changes may explain the drop in T_3, since pre-season values were obtained in the winter time and the remaining test sessions occurred in the spring through late summer,

into the first part of fall. Increases in T_3 have been seen during winter in Japanese individuals living in Kijimadaira, however they were living in houses with temperatures between minus three degrees and six degrees Celsius (15-20 degrees in summer). No changes were seen in individuals living in heated rooms with outside temperatures between minus three and plus six degrees Celsius.[105] It is therefore unlikely that the judokas would experience a drop in T_3 as a result of climate change.

It is also possible that vigorous exercise may result in an increase in T_3. Chronic voluntary exercise in mice demonstrated a rise in serum T_3.[106] Athletes may have been training vigorously during their first test session, when their serum T_3 levels were high, and less vigorously during their competitive and off-season, when T_3 levels dropped. It should be noted however, that there was no significant difference in exercise during any of the test sessions. The reason for a drop in serum T_3 remains unknown.

4.2 ANTHROPOMETRICS

One negative consequence of weight cycling that has been postulated is an increase in percentage body fat and a decrease in LBM. Because LBM is a good predictor of REE, loss of LBM could potentially explain a decrease in absolute REE. Furthermore increased adiposity has been related to increased health risks for coronary heart disease and high blood pressure. There was no evidence in this study of an increase in body weight, percentage body fat or a decrease in LBM as a result of

weight cycling.

4.2.1 REASONS FOR NO DIFFERENCES

NO WEIGHT CYCLING EFFECTS

This may indicate that there is no effect of weight cycling on body composition. Both animal studies and human studies have indicated that there is no increase in adiposity with repeated cycles of weight loss and regain.[41,35,42-44] In contrast however, other studies have indicated increased adiposity with one cycle[45] or repeated cycles of weight loss and regain.[33,38] There are several possible explanations as to why no effects were demonstrated in this study, and possibly other studies.

SEVERITY OF WEIGHT CYCLING

Weight cycling may not have been severe enough to invoke such changes. Changes in body composition may be related to the degree of caloric restriction, the duration of caloric restriction, and the number of times caloric restriction occurs. Ozelci et al[38] demonstrated that rats restricted to 50% or 25% of their ad libitum food intake for one week had an increase in body fat following refeeding. This however was not demonstrated in rats restricted to 75% of ad libitum intake. It is possible that restriction was not severe enough to incur large changes in body fat and LBM. In humans, an increase in adiposity and decrease in LBM was seen in normal weight young males after weight regain. However, they had undergone semi-starvation, losing 24% of their body weight in 24

weeks.[45] The athletes in this study lost an average of only 4.1±1.5 kg per competition and the duration of food restriction was only an average of 8.1±6.9 days.

It is possible that the judokas did not cut weight enough times to influence the body fat to lean ratio (only cutting an average of 5.2±2.4 times). However the wrestlers in Melby et al's study, who underwent similar weight cycling habits, but cut weight more frequently (11 times,range9-13), also showed no increase in body fat following a season of cutting weight.

ACCURACY OF SKINFOLD MEASUREMENTS

Another limitation of small weight losses, is that skinfold changes may have been too small to detect, considering the method of body fat determination. Although numerous studies report that skinfolds are as accurate as bioelectrical impedance and hydrodensitometry, and have good reproducibility[76-78] this still remains controversial. Skinfolds have been indicated to be accurate within 3%.[79] The same trained observer conducted all skinfold measurements and repeated them 3 times, which increases the precision of the recorded measurement.[79] Furthermore, skinfolds on lean individuals are known to produce more accurate results, as compared to obese individuals.[107] Even if the actual fat percentage was not valid, the change in percentage fat relative to baseline values was determined with validity. Since the relative change in percentage fat was important, and not the absolute values, the method was appropriate.

EXERCISE MAINTAINS LEAN BODY MASS

Exercise may have maintained LBM and prevented the accumulation of fat. Studies have shown that exercise can prevent the loss of LBM and can even increase it during weight loss.[5] Weight regained while exercising is more likely to be regained as lean tissue rather than fat.[5] Different types of exercise will have different effects on body composition. Weight lifting will increase LBM whereas sustained aerobic exercise will decrease body fat. Exercise may have prevented any decline in LBM and any accumulation of body fat in the judokas, which may have occur in the sedentary individual. Melby et al[36] found that there was no proportional loss of LBM or increase in percentage fat in their wrestlers when weight was regained.

FAT SELECTION

An increase in fat selection following a period of food restriction has been suggested to occur,[39,46-48] resulting in an increase in adiposity.[49-51] There was no suggestion of an increase in fat selection in this study possibly preventing an increase in percentage body fat. It should be noted however that if there was an increase in fat selection, it would most likely occur immediately following food restriction. Diet records were not kept during a refeeding period and if an increase in fat selection had occurred it would have to be maintained throughout the season to be detected. Off-season diet records did not indicate that athletes increased the percentage of fat in their diet when they were no longer required to watch their caloric intake.

4.2.2 INSULIN

Fasting serum insulin and glucose levels of weight cycling judokas were no different from non-cycling judokas at baseline and did not change following a season of weight cycling. There did not appear to be a prolonged effect of weight cycling on insulin or glucose values. Some studies have suggested that refeeding following a period of calorie restriction can result in a rise in serum insulin levels,[25,39,63,64] especially if overfeeding occurs.[63] Such changes may have explained in part, any increases in body fat, through promotion of lipogenesis and fat storage.

It should be noted however, that measurement of fasting insulin values may not have been appropriate if there were only changes in postprandial insulin levels, as indicated by McCargar et al.[63] Fasting serum insulin levels were normal in normal weight 4 day food restricted, 2 day overfed females, and only the insulin response (2 hours postprandial) to glucose in a meal was exaggerated, rising above normal.

Owens et al[64] found that an effect on insulin and glucose metabolism following 2 days of food restriction, remained up to 10 days, after which time, values would return to normal. The judokas were not measured immediately after a bout of dieting.

Once again it should be noted that the duration, frequency and level of calorie restriction may not have been sufficient to cause any alteration in serum insulin and glucose levels.

4.2.3 FAT DISTRIBUTION

Distribution of body fat is also an important factor in relation to health. Android fat has been associated with increased risk of coronary heart disease whereas gynoid fat has not[2]. It has been suggested that weight cycling may lead to a redistribution of body fat to the abdominal region.[55] Results of this study however do not indicate any such change. The WHR of the experimental group remained the same during all test sessions, and was never significantly different than the control group. The WHR measurements of this population were well below levels that would be considered a "risk factor" at all times points during the study.

Once again, it is possible that the severity and duration of weight loss was not enough to mobilize significant amounts of fat to be redistributed. There may not have been enough cycles of weight loss and regain to show such effects. It is possible that repeated seasons of weight cycling may be required to detect an increase in WHR, however the judokas did have a history of weight cycling (average 6.0±5.3 years) and showed no increase in WHR as compared to non-cycling judokas.

4.3 DIET ANALYSIS

The weight cycling judokas did not consume significantly fewer calories at any test time as a result of cutting weight. This would suggest that they did not require fewer calories at baseline (pre-season), or following a season of cutting weight, to maintain weight. Diet analysis at peak season did not occur during an episode of food restriction, and thus did not reflect the caloric intake during weight

reduction.

Diet analysis at baseline also demonstrated that weight cycling and non-cycling judokas were consuming 25% and 11% fewer calories than predicted by the Harris-Bennedict Equation, respectively (table 8). This trend continued throughout remaining test sessions. Once again it has been suggested that the Harris-Benedict Equation overestimates REE,[92] although this appears to be extreme.

It is possible that energy consumption was underestimated due to the limitations of three day food records. A recent study by Livingston et al[108] indicated that energy intakes determined by seven day food records were only about 80% of outputs, as indicated by use of doubly labeled water, for a period of two weeks. Underreporting was suggested to occur in all subjects (not just obese individuals as previously suggested[109]) and was a result of failure to record snack items and alcohol, as well as an alteration in habitual intake to avoid weighing and recording tasks.[108]

There was no significant change in the percentage or absolute amounts of macronutrients consumed in both groups. This indicates, as mentioned earlier, that there was no sustained increase in fat selection as a result of food restriction. The consumption of protein was never compromised, being in excess of the calculated requirements for athletes based on body weight for both non-cycling and cycling groups.

CHAPTER 5

CONCLUSIONS AND RECOMMENDATIONS

It is very difficult to study the metabolic effects of weight cycling. Populations which are metabolically different, such as the severely obese, the moderately obese, the mildly obese, the sedentary lean, the active lean and the very athletic lean, may all respond to weight cycling differently. How they respond will depend on the severity, duration and frequency of food restriction and their metabolic differences. For these reasons it has been difficult to devise a classification system for weight cycling. A different classification scheme may even be required for each individual population. Furthermore, there is wide individual variability in metabolism and there may be wide variability in response to weight cycling, even within the same population. Any solid conclusions with respect to the effects of weight cycling will require a clear definition. Factors which identify individuals who are particularly susceptible to weight cycling, will also have to be defined.

Recently there have been numerous additional studies looking at the effects of weight cycling on RMR and body composition in humans. To date, few are showing detrimental effects of weight cycling. In particular, there appears to be no reduction in RMR beyond that expected for loss of LBM. This may be due to the fact that only a select few are susceptible to the effects of weight cycling. Since the current literature consists of studies of quite small numbers, the susceptible few would not be identified. Furthermore it is possible that the main

detrimental effect of weight cycling is a disproportionately large loss of LBM due to rapid weight loss and rapid weight regain, and not an actual reduction in RMR/LBM. Further study with larger sample sizes and defined weight cyclers will have to confirm this.

Another important issue to consider is that weight cycling may cause a reduction in non-resting components of RMR, such as diet or exercise induced thermogenesis. This may be with or without a reduction in RMR. Most studies fail to measure these parameters, therefore missing any such effect.

The judoka weight cyclers showed no decrease in absolute or relative RMR. There was no increase in percentage body fat or decrease in lean tissue. The distribution of body fat also remained unchanged. Serum triiodothyronine, insulin and glucose all remained within the normal range and did not change significantly as a result of weight cycling. There was no change in caloric consumption or macronutrient distribution. Additionally no increase in fat selection was observed. No detrimental effects of weight cycling were identified in this study.

Some changes in experimental protocol may have helped to enhance the conclusions. More precise exercise records (hours per day, type of exercise, intensity) may account for any possible variation in RMR as a result of change in type, intensity or duration of exercise. This would be difficult, due to unavailable classification schemes for exercise. Furthermore it would have been difficult to get the athletes to keep such detailed and complete records, requiring daily phone calls to ensure accurate recording or any recording at all.

It would have been useful to record diets at different periods within the peak season. These may include their diet while not cutting weight for a competition, exact diet while cutting weight for competition, and the diet immediately following the competition. These would give a good indication of the changes in eating behaviors. This would also identify imbalances or deficiencies in the restrictive diet and the composition of the refeeding diet (ie did they over eat, how much, for how long, did they consume excess fat, or excess CHO?). Duplicate or triplicate diet records would also verify the accuracy of recording or the accuracy of the diet analysis itself.

Lipid profiles may have been a useful addition to biochemical parameters studied. Although it would not have helped explain changes in body composition and insulin metabolism, it may have indicated additional effects of weight cycling, such as elevated lipid and cholesterol levels.

The results of this study indicate that judokas who cut an average of 4 kg, an average of 4 times a season, will experience no reduction in RMR, and no alteration in body composition, fat distribution, and fasting serum insulin, glucose and T_3 levels. It may be possible that judokas do not provide a good model for the study of metabolic and physiological effects of weight cycling, on the premise that they may not cut enough weight, for a sufficient length of time, to induce such consequences.

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APPENDIX I

CERTIFICATE OF APPROVAL

The University of British Columbia
Office of Research Services

C89-009

CLINICAL SCREENING COMMITTEE FOR RESEARCH
AND OTHER STUDIES INVOLVING HUMAN SUBJECTS

C E R T I F I C A T E of A P P R O V A L

INVESTIGATOR:	McCargar, L.
UBC DEPT:	Family & Nutr Sci
INSTITUTION:	FMSC
TITLE:	Metabolic consequences of recurrent dieting (Amended 16 September 1990)
NUMBER:	C89-009
APPROVED:	SEP 25 1990

The protocol describing the above-named project has been reviewed by the Committee and the experimental procedures were found to be acceptable on ethical grounds for research involving human subjects.

Dr
Cl

es

THIS CERTIFICATE OF APPROVAL IS VALID FOR THREE YEARS
FROM THE ABOVE APPROVAL DATE PROVIDED THERE IS NO
CHANGE IN THE EXPERIMENTAL PROCEDURES

APPENDIX II

WEIGHT CYCLING QUESTIONNAIRE

WEIGHT CYCLING QUESTIONNAIRE

Date _____

OFFICE USE ONLY

No. _____
BMI _____
WHR _____

Personal Data

Age _____ Sex _____
Height _____ Weight _____
Waist _____ Hip _____

Eating and Weight Patterns

1. Have you ever tried to lose weight by dieting, i.e., restricting food intake to a level less than usual? (please circle the number of your answer)

   1 NO, NEVER IF NO, GO TO QUESTION 2
   2 YES, ONCE
   3 YES, MORE THAN ONCE

A. How often are you dieting to lose weight? (circle number)

   1 NEVER
   2 RARELY
   3 SOMETIMES
   4 OFTEN
   5 ALWAYS

B. Have you tried the following weight loss diet programs or methods to lose weight in the past five years? (please check either yes or no)

	Yes	No	
Weight Watchers	___	___	
Diet Center	___	___	
TOPS	___	___	
Nutrisystem	___	___	
Herbalife	___	___	
formula diets (specify)	___	___	_____
diet books (specify)	___	___	_____
hospital program (specify)	___	___	_____
Overeaters Anonymous	___	___	
diet pills	___	___	
diuretics/emetics	___	___	

other (please specify) _____

C. How many times have you tried to lose weight in the past year? (circle number of your answer)

1 0
2 1-2
3 3-5
4 6-9
5 10 OR MORE

D. For what reason(s) have you dieted to lose weight? (circle all that apply)

1 A SPORT OR ATHLETIC EVENT, ON THE ADVICE OF A COACH
2 A SPORT OR ATHLETIC EVENT, WITHOUT A COACH'S ADVICE
3 TO LOOK MORE ATTRACTIVE
4 HEALTH REASONS, ON THE ADVICE OF A DOCTOR
5 HEALTH REASONS, WITHOUT A DOCTOR'S ADVICE
6 OTHER (please specify) _____

E. When was the last time you started a diet to lose weight? (circle number of your answer)

1 WITHIN THE LAST TWO WEEKS
2 WITHIN THE LAST MONTH
3 WITHIN THE LAST SIX MONTHS
4 WITHIN THE LAST YEAR
5 MORE THAN ONE YEAR AGO

F. How much weight did you <u>want</u> to lose on your most recent diet? _____ lbs.

G. How much weight <u>did</u> you lose on your most recent diet? _____ lbs.

H. How long did you stay on your most recent diet? _____

I. How old were you when you first dieted to lose weight? ____

J. Are you a yo-yo dieter (that is, do you experience frequent weight losses and regains)? (circle number of your answer)

1 YES
2 NO

2. What is the maximum amount of weight you have ever lost within one week, not due to illness or pregnancy? _____ lbs

3. What is your maximum weight gain within a week? _____ lbs

4. When you gain weight, where do you gain it? (check all that apply)

_____ AROUND THE MIDDLE (WAIST, ABDOMEN)
_____ HIPS, THIGHS AND BUTTOCKS
_____ ALL OVER
_____ ELSEWHERE (please specify) _____
_____ NOT APPLICABLE--MY WEIGHT GAIN IS MINIMAL

5. When you lose weight, where do you lose it? (check all that apply)

_____ AROUND THE MIDDLE (WAIST, ABDOMEN)
_____ HIPS, THIGHS AND BUTTOCKS
_____ ALL OVER
_____ ELSEWHERE (please specify) _____
_____ NOT APPLICABLE--MY WEIGHT LOSS IS MINIMAL

6. How many times <u>in the past 5 years</u> would you estimate you have lost the number of pounds shown below, excluding pregnancy/post-partum? (write in number of times)

<u>Weight loss</u>	<u>Number of times</u>
1-5 lb	_____
6-10 lb	_____
11-20 lb	_____
21-30 lb	_____
31-50 lb	_____
51+ lb	_____

7. Describe your weight between the ages: (check where appropriate)

	6-10 years	11-13 years	14-17 years	18-25 years	26+ years
Extremely thin	( )	( )	( )	( )	( )
Very thin	( )	( )	( )	( )	( )
Somewhat thin	( )	( )	( )	( )	( )
Average	( )	( )	( )	( )	( )
Somewhat overweight	( )	( )	( )	( )	( )
Very overweight	( )	( )	( )	( )	( )
Extremely overweight	( )	( )	( )	( )	( )

8. What has been your minimum weight as an adult (>25 years old)? _____ lbs

9. What has been your maximum non-pregnant weight ever? _____lbs

10. Have you ever purged (used self-induced vomiting, laxatives, diuretics) to control your weight? (circle number of your answer)

1 YES
2 NO IF NO, GO TO QUESTION 17

11. Have you ever used emetics or vomitted to control weight?

1 YES
2 NO IF NO, GO TO QUESTION 13

12. How often do you vomit to control weight?

1 LESS THAN ONCE PER MONTH
2 1-3 TIMES PER MONTH
3 ONCE PER WEEK
4 2-6 TIMES PER WEEK
5 DAILY
6 MORE THAN ONCE PER DAY

13. Have you ever used laxatives to control weight?

1 YES
2 NO IF NO, GO TO QUESTION 15

14. How often do you use laxatives to control weight

1 LESS THAN ONCE PER MONTH
2 1-3 TIMES PER MONTH
3 ONCE PER WEEK
4 2-6 TIMES PER WEEK
5 DAILY
6 MORE THAN ONCE PER DAY

15. Have you ever used diuretics to control weight?

1 YES
2 NO IF NO, GO TO QUESTION 17

16. How often do you use diuretics to control weight?

1 LESS THAN ONCE PER MONTH
2 1-3 TIMES PER MONTH
3 ONCE PER WEEK
4 2-6 TIMES PER WEEK
5 DAILY
6 MORE THAN ONCE PER DAY

17. How satisfied are you with your current body size and shape? (circle number)

1	2	3	4	5	6	7
NOT AT ALL SATISFIED			MODERATELY SATISFIED			EXTREMELY SATISFIED

18. What do you consider to be your ideal weight? _____ lbs

Exercise Patterns

19. How many hours per week do you engage in vigorous physical activity (e.g. running, swimming, cycling, brisk walking)? (circle number)

1 0-1 hour
2 1.1-3 hours
3 3.1-5 hours
4 5.1-8 hours
5 8.1-11 hours
6 More than 11 hours

Please specify the activities in which you participate:

_____

20. How many years have you been exercising vigorously (working out at least three days per week)? _____ years
Not applicable _____

21. Please list any medications that you are currently using:

_____

Thank you for completing this questionnaire.

JUDO SUBJECT QUESTIONNAIRE

(1) How many years have you been in competitive judo?_____

(2) How many years have you been cutting weight for judo competitions?_____

(3) What is your normal weight range off-season?_____

(4) What is your normal weight during the competitive season?_____

(5) What is your competing weight?_____

(6) How frequently would you say you reduce weight for competitions?(circle one):
never rarely sometimes often always

(7) Estimate how many times you reduce weight in a given season._____

(8) How many competitions, where meeting your competitive weight is necessary, can you estimate this season?_____

(9) How much weight would you lose each dieting session?(circle one):
<1.4 kg 1.5-2.9 kg 3.0-3.9 kg 4.0-4.9 kg =>5 kg

(10) What is involved in your judo training?:
(a)strength training: high moderate low
(b)cardiovascular training: high moderate low

(11) What forms of vigorous exercise do you do:
(a)pre-season_____total hrs/wk_____
(b)during season_____total hrs/wk_____
(c)off-season_____total hrs/wk_____

(12) Have you found losing weight is getting harder to do?:
(a)from season to season? yes___ no___
(b)with in a season? yes___ no___

(13) Have you found that you regain weight more rapidly or with greater ease:
(a)from season to season? yes___ no___
(b)within a season? yes___ no___

(14) Off season, do you:
(a)regain to previous off season weight? yes___ no___
(b)gain more than your off season weight? yes___ no___
(c)remain the same weight? yes___ no___

APPENDIX III

MEDICAL GRAPHICS CORPORATION SYSTEM 2001

Medical Graphics Corporation System 2001

RMR will be determined using the System 2001 metabolic cart located in the Sports Medicine Center. It utilizes open circuit, indirect calorimetry, which allows energy expenditure determination by measuring oxygen(O_2) consumption and carbon dioxide(CO_2) production.

Air expired by the subject is directed through a hose into the System 2001, and flow rate and volume is measured by heated linear pneumatach. these measurements are then amplified and sent to a wave form analyzer. This analyzer will compute the following:

- relative time since start of collection (min.sec.)
- inspiratory time of breath(sec)
- total time of breath(sec)
- tidal volume(ml)
- frequency of breathing(br/min)
- minute ventilation(L/min)
- volume of CO_2 in breath(ml)
- end tidal CO_2(%)
- inspired CO_2(%)
- volume of O_2 in breath(ml)
- end tidal O_2(%)
- inspired O_2(%)
- heart rate

The computer will recieve this data to calculate energy expenditure, respiratory quotient, and percentage of each fuel source utilized(CHO, protein, and fat) during the time of testing.

The testing conditions stated by the Systems 2001 manual are as follows:

- rest 20-30 minutes prior to testing
- fast 2 to 4 hours prior (12 hour fast will be used)
- early morning measurements(before breakfast)
- thermoneutral and quite environment
- 15 to 20 minute data collection

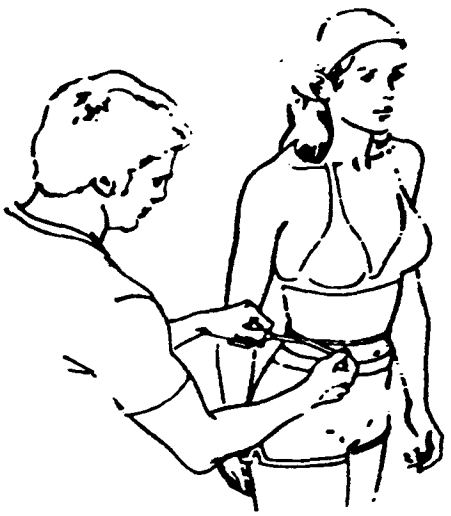
The conditions under which RMR will be tested in this study have been indicated in the methods section.

APPENDIX IV

PROCEDURE FOR ANTHROPOMETRIC MEASUREMENTS

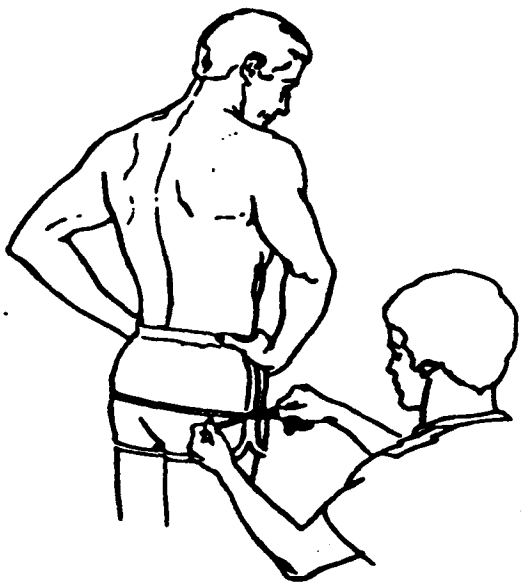
b) Waist (Abdomen) Girth

The participant stands erect. The appraiser uses a cross-handed technique to position the tape horizontally at the level of noticeable waist narrowing. The tape is then placed in the recording position and the measurement is made at the end of a normal expiration. In some participants an indeterminate waist can be approximated by taking the girth at the estimated lateral level of the twelfth or lower floating rib.



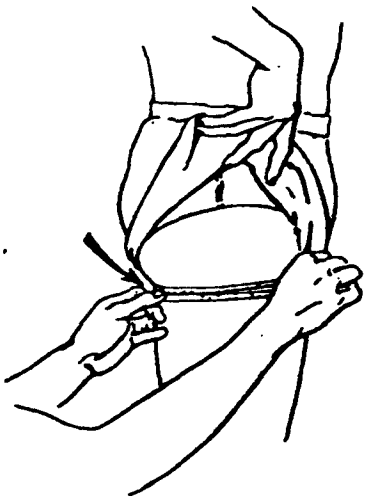
c) Hip (Gluteal) Girth

The participant stands erect with feet together. The tape is positioned around the hips at the level of the symphysis pubis and the greatest gluteal protuberance.



d) Right Thigh Girth

The participant stands erect, feet slightly apart. The tape is positioned around the right thigh to a level one centimeter below the gluteal line.



4.3.4 Skinfold Measurements

See section 5.2 – BODY WEIGHT, ADIPOSITY AND FAT DISTRIBUTI (regarding use of the skinfold measurements)

Equipment: Harpenden or Lange (ipers.

Do one complete round of all the sku measurements before repeating the cedure to obtain a second skinfold surement for each site. All measuren are taken only on the right side of th body

General Procedure

During skinfold measurements, it is essential that the participant relax t underlying musculature as much as sible. When the site of the skinfold been determined, a fold of skin plus underlying fat is grasped between t thumb and forefinger with the back the hand facing the appraiser. Keep the jaws of the calipers always at ri angles to the body surface, the con faces of the calipers are placed one centimeter below the point where th skinfold is raised. While maintaining pressure of the fingers on the skinfol the trigger of the calipers is fully released and the measurement is tak The measurement is noted when the indicator stabilizes which is approximately two seconds after the full pres sure of the caliper jaws is applied to skinfold. The reading is recorded to nearest 0.2 millimetres; e.g. 16.8

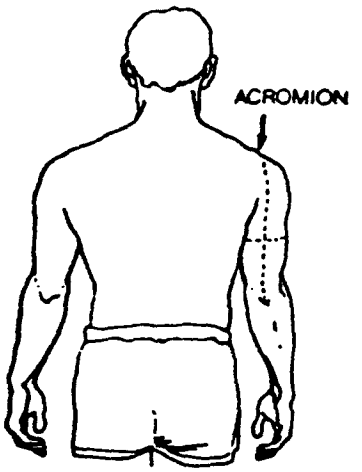
Complete the first set of skinfold mea surements for all sites. Then, repeat t procedure to obtain a second set of measurements for each skinfold site. Record the mean of the two measure unless the difference between the firs and second measure of that particula skinfold site is found to be greater tha 0.4 mm. If so, take a third measure of that skinfold site and choose from among the three values, the two measures which most closely match each other in value. Determine the mean of those two measures. Should the three measures be equidistant, e.g., 18.6 19 19.8 determine the mean of all three values.

It should be noted that the accuracy of skinfold measurements depends on:

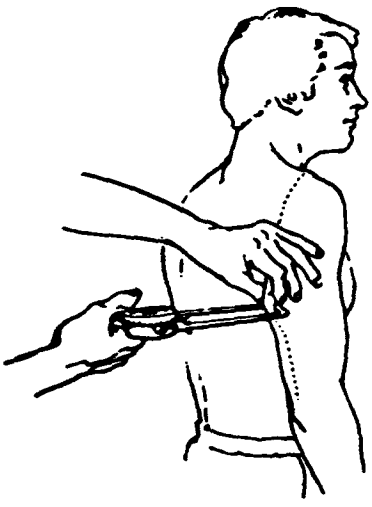
- precise identification of the site of the skinfold;
- forming the skinfold prior to the applica tion of the caliper jaws;
- the standardization of the alignment of the skinfold crest;
- maintenance of the pressure by the fingers on the skinfold when the measurement is taken;
- complete release of the caliper jaws.

a) Triceps Skinfold

The participant stands with the arms relaxed by the sides. The triceps skinfold is taken on the back of the right arm at the point midway between the tip of the acromion (right shoulder) and the tip of the olecranon (right elbow). The midpoint is determined by placing the fifth finger of the left hand on the tip of the acromion (right shoulder), the fifth finger of the right hand on the tip of the olecranon (right elbow) and then the thumbs are placed together to determine the midpoint.



The skinfold is then raised at the mid-arm point, so the fold runs vertically along the midline of the back of the arm.

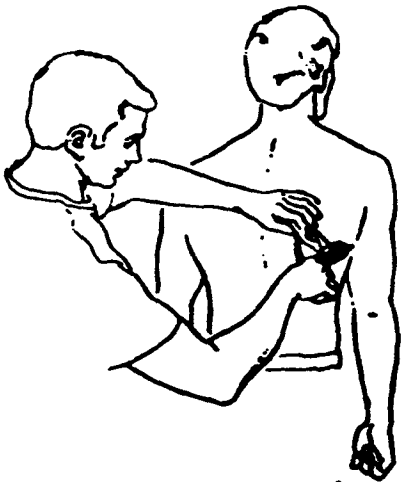


b) Biceps Skinfold

The biceps skinfold is measured on the right extended upper arm over the biceps at the same level as the mid-arm point for the triceps. The skinfold is then raised at the mid-arm point so the fold runs vertically along the midline of the front of the arm.

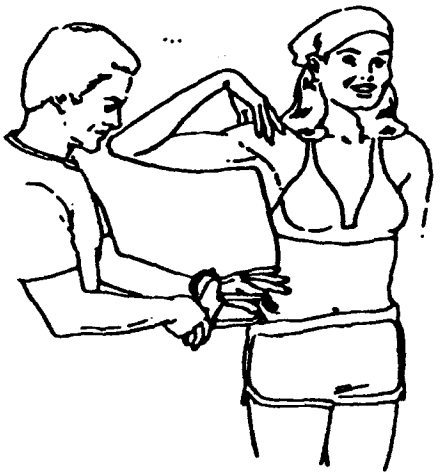
c) Subscapular Skinfold

The participant stands with the shoulders relaxed and the arms by the sides. The skinfold is raised so it can be measured on a diagonal line coming from the vertebral border of the scapula to a point 1 cm. beneath the inferior angle. The skinfold runs downward and outward at an angle of approximately 45 degrees to the spine.



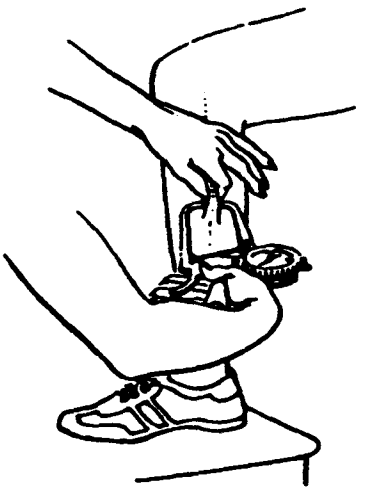
d) Iliac Crest Skinfold

The participant stands in a normal erec position. Have the participant raise the right arm to the side so that it is horizo tal and place the right hand on the rig shoulder. If the participant is unable to place hand on shoulder, keep the horizontal arm extended. The skinfold is the measured three centimetres above the crest of the ilium at the midline of the body so that the fold runs forward and slightly downward.



e) Medial Calf Skinfold

Have the participant place the unweighted (relaxed) right foot flat on a step so that the knee is at 90°. The skinfold is raised on the inside of the right calf just above the level of the maximum calf girth so that the fold runs vertically along the midline.



FROM: CANADIAN STANDARDIZED TEST OF FITNESS, OPERATIONS MANUAL , 1986

APPENDIX V

DURNIN AND WOMERSLEY LINEAR REGRESSION EQUATIONS

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Table 5. *Linear regression equations for the estimation of body density* $\times 10^3$ *(kg/m³) from the logarithm of the skinfold thickness: density* $= c - m \times$ *log skinfold*

(*a*) Males

Skinfold		Age (years) 17–19	20–29	30–39	40–49	50+	17–72
Biceps	*c*	1·1066	1·1015	1·0781	1·0829	1·0833	1·0997
	m	0·0686	0·0616	0·0396	0·0508	0·0617	0·0659
Triceps	*c*	1·1252	1·1131	1·0834	1·1041	1·1027	1·1143
	m	0·0625	0·0530	0·0361	0·0609	0·0662	0·0618
Subscapular	*c*	1·1312	1·1360	1·0978	1·1246	1·1334	1·1369
	m	0·0670	0·0700	0·0416	0·0686	0·0760	0·0741
Supra-iliac	*c*	1·1092	1·1117	1·1047	1·1029	1·1193	1·1171
	m	0·0420	0·0431	0·0432	0·0483	0·0652	0·0530
Biceps + triceps	*c*	1·1423	1·1307	1·0995	1·1174	1·1185	1·1356
	m	0·0687	0·0603	0·0431	0·0614	0·0683	0·0700
Biceps + subscapular	*c*	1·1457	1·1469	1·0753	1·1341	1·1427	1·1498
	m	0·0707	0·0709	0·0445	0·0680	0·0762	0·0759
Biceps + supra-iliac	*c*	1·1247	1·1259	1·1174	1·1171	1·1307	1·1331
	m	0·0501	0·0502	0·0486	0·0539	0·0678	0·0601
Triceps + subscapular	*c*	1·1561	1·1525	1·1165	1·1519	1·1527	1·1625
	m	0·0711	0·0687	0·0484	0·0771	0·0793	0·0797
Triceps + supra-iliac	*c*	1·1370	1·1362	1·1273	1·1383	1·1415	1·1463
	m	0·0545	0·0538	0·0531	0·0660	0·0718	0·0656
Subscapular + supra-iliac	*c*	1·1374	1·1429	1·1260	1·1392	1·1582	1·1522
	m	0·0544	0·0573	0·0497	0·0633	0·0771	0·0671
Biceps + triceps + subscapular	*c*	1·1643	1·1593	1·1213	1·1530	1·1569	1·1689
	m	0·0727	0·0694	0·0487	0·0730	0·0780	0·0793
Biceps + triceps + supra-iliac	*c*	1·1466	1·1451	1·1332	1·1422	1·1473	1·1556
	m	0·0584	0·0572	0·0542	0·0647	0·0718	0·0683
Biceps + subscapular + supra-iliac	*c*	1·1469	1·1508	1·1315	1·1452	1·1626	1·1605
	m	0·0583	0·0599	0·0510	0·0640	0·0768	0·0694
Triceps + subscapular + supra-iliac	*c*	1·1555	1·1575	1·1393	1·1604	1·1689	1·1704
	m	0·0607	0·0617	0·0544	0·0716	0·0787	0·0731
All four skinfolds	*c*	1·1620	1·1631	1·1422	1·1620	1·1715	1·1765
	m	0·0630	0·0632	0·0544	0·0700	0·0779	0·0744

Body fat and skinfolds

Table 5 (*cont.*)

(*b*) Females

Skinfold		Age (years) 16–19	20–29	30–39	40–49	50+	16–68
Biceps	*c*	1·0889	1·0903	1·0794	1·0736	1·0682	1·0871
	m	0·0553	0·0601	0·0511	0·0492	0·0510	0·0593
Triceps	*c*	1·1159	1·1319	1·1176	1·1121	1·1160	1·1278
	m	0·0648	0·0776	0·0686	0·0691	0·0762	0·0775
Subscapular	*c*	1·1081	1·1184	1·0979	1·0860	1·0899	1·1100
	m	0·0621	0·0716	0·0567	0·0505	0·0590	0·0669
Supra-iliac	*c*	1·0931	1·0923	1·0860	1·0691	1·0656	1·0884
	m	0·0470	0·0509	0·0497	0·0407	0·0419	0·0514
Biceps + triceps	*c*	1·1290	1·1398	1·1243	1·1230	1·1226	1·1362
	m	0·0657	0·0738	0·0646	0·0672	0·0710	0·0740
Biceps + subscapular	*c*	1·1241	1·1314	1·1120	1·1031	1·1029	1·1245
	m	0·0643	0·0706	0·0581	0·0549	0·0592	0·0674
Biceps + supra-iliac	*c*	1·1113	1·1112	1·1020	1·0921	1·0857	1·1090
	m	0·0537	0·0568	0·0528	0·0494	0·0490	0·0577
Triceps + subscapular	*c*	1·1468	1·1582	1·1356	1·1230	1·1347	1·1507
	m	0·0740	0·0813	0·0680	0·0635	0·0742	0·0785
Triceps + supra-iliac	*c*	1·1311	1·1377	1·1281	1·1198	1·1158	1·1367
	m	0·0624	0·0684	0·0644	0·0630	0·0635	0·0704
Subscapular + supra-iliac	*c*	1·1278	1·1280	1·1132	1·0997	1·0963	1·1234
	m	0·0616	0·0640	0·0564	0·0509	0·0523	0·0632
Biceps + triceps + subscapular	*c*	1·1509	1·1605	1·1385	1·1303	1·1372	1·1543
	m	0·0715	0·0777	0·0654	0·0635	0·0710	0·0756
Biceps + triceps + supra-iliac	*c*	1·1382	1·1441	1·1319	1·1267	1·1227	1·1432
	m	0·0628	0·0680	0·0624	0·0626	0·0633	0·0696
Biceps + subscapular + supra-iliac	*c*	1·1355	1·1366	1·1212	1·1108	1·1063	1·1530
	m	0·0622	0·0648	0·0570	0·0536	0·0544	0·0727
Triceps + subscapular + supra-iliac	*c*	1·1517	1·1566	1·1397	1·1278	1·1298	1·1327
	m	0·0689	0·0728	0·0646	0·0609	0·0650	0·0643
All four skinfolds	*c*	1·1549	1·1599	1·1423	1·1333	1·1339	1·1567
	m	0·0678	0·0717	0·0632	0·0612	0·0645	0·0717