

The Impact of Acute and Chronic Weight Restriction and Weight Regulation Practices on Physiological, Osteogenic, Metabolic and Cognitive Function in Elite Jockeys

Thesis submitted for the degree of Doctor of Philosophy

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Abstract:

Horse racing is a weight category sport. One of the key challenges facing jockeys is the pressure of “making weight” throughout the protracted racing season. **Aim:** The aim of this study was to examine the effect of a chronically weight restrictive lifestyle and acute weight loss practices on aspects of physiological, osteogenic, metabolic and cognitive function in jockeys. **Methods:** The primary aim was achieved through the completion of four related studies. *Study One:* The effect of a four % reduction in body mass in 48 hours on physiological and cognitive function was assessed through performance on an incremental cycle ergometer test to volitional exhaustion and a computer based cognitive test battery. *Study Two:* Bone mass was compared between jockeys (flat and national hunt), elite amateur boxers and a group of age, gender and BMI matched controls. *Study Three:* Bone mass, bone turnover and endocrine factors related to growth and metabolism were analysed in a group of jockeys and age, gender and BMI matched controls. *Study Four:* The impact of 6 months whole body vibration therapy (0.3 g and 30 Hz) on bone mass and turnover was analysed in a group of professional jockeys. **Results:** In study one maximal aerobic exercise performance was negatively affected following a four % loss in body mass in 48 hours as evidenced by a reduction in peak power output achieved and an increase in submaximal cardiovascular strain. No changes to cognitive performance were identified in this study. In study two both groups of jockeys had lower bone mass at a number of sites than either the boxer or control groups. Adjustment of bone data revealed that differences in height and lean mass accounted for some of the variation between the groups, but that additional factors were present which may have impacted on bone mass in. Study three showed that bone mass was reduced and bone resorption increased in a jockey group. Elevated SHBG and reduced IGF-1 levels in comparison to an age, gender and BMI matched control group appeared to have a role to play in this finding. No aspect of body composition, bone mass or turnover was affected by the vibration therapy protocol used within study four. **Conclusion:** Results from this research appeared to indicate that aspects of physiological, osteogenic and metabolic function are affected in jockeys. This is likely to have occurred in response to a chronically weight restricted lifestyle. These findings may convey both long and short term health risks to jockeys and as such represent a major health and safety concern to the racing industry.

Declaration

I hereby certify that this material, which I now submit for assessment on the programme of study leading to the award of PhD is entirely my own work, that I have exercised reasonable care to ensure that the work is original, and does not to the best of my knowledge breach any law of copyright, and has not been taken from the work of others save and to the extent that such work has been cited and acknowledged within the text of my work.

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Glossary of Terms

Claiming Allowance:

Incentive whereby trainers and owners are encouraged to allocate rides to apprentice jockeys, whereby apprentice jockeys may “claim” up to 10 lbs/4.5 kg reduction to their mount’s allocated racing handicap.

Energy Availability:

The proportion of available energy required to maintain usual metabolic processes after the additional requirements of physical activity are accounted for.

Estimated Average Requirement:

The mean requirement of the population.

Flat Racing:

Races of 5 – 20 furlongs which consist of a run with no obstacles. Weight allocations for Flat jockeys range from 52.7 – 64kg.

Furlong:

1/8 of a mile/201.2 metres.

Hormone Half Life:

The time it takes to reduce the concentration of circulating hormone by one half when no new hormone is being produced.

Lower Threshold Intake:

That intake which is two standard deviations below the estimated average requirement and is the estimated intake below which nearly all individuals (97.5%) will be unable to maintain metabolic integrity.

Making Weight:

The practice used by weight category athletes to attain the required weight.

National Hunt Racing:

Races at least 3.2km long throughout which the horse must jump a number of fences or hurdles. Weight allocations for national hunt jockeys range from 62 – 76kg.

Quantitative Trait Loci:

Stretches of DNA that are closely linked to the genes that underlie the trait in question.

T Score:

The bone mass standard score describing the individual's deviation from the mean as described for a young (mid twenties) healthy population.

Thoroughbred Horses:

The horses used in thoroughbred horse-racing. A purebred horse which originates from the breeding of native English mares with one of three imported Arabian stallions.

Vibration Amplitude:

The extent of oscillatory motion, which manifests as peak to peak displacement. Can be measured in mm or in accordance with g – the gravitational constant of the earth ($9.81 \text{ m}\cdot\text{s}^{-1}$)

Vibration Frequency:

The repetition rate of oscillatory cycles – measured in hertz (Hz)

Z Score:

A standard score describing bone mass deviation from an age matched mean.

List of Publications

Peer Reviewed Journal Articles

Warrington, G. D., Dolan, E., McGoldrick, A., McEvoy, J., MacManus, C., Griffin, M., & Lyons, D. 2009b, "Chronic Weight Control Impacts on Physiological Function and Bone Health in Elite Jockeys", *Journal of Sports Sciences*, vol. 27, no. 6, pp. 543-550.

Dolan, E., O'Connor, H., O'Loughlin, G., McGoldrick, A., & Warrington, G. 2010b, "Nutritional and Lifestyle Practices of the Horse Racing Jockey", *Journal of Sports Sciences*. In Press.

Abstracts

Dolan, E., McGoldrick, A., McCaffrey, N., O'Connor, P., May, G., Fitzpatrick, P., & Warrington, G. 2009, "Comparison Between Skinfolts and DEXA For Determination of Body Composition in Weight Category Athletes", *Medicine and Science in Sport and Exercise*, vol. 41, no. 5, p. S541.

Dolan, E., McGoldrick, P. A., MacManus, C., O'Gorman, D. J., Moyna, N. M., & Warrington, G. D. 2007, "Comparison of the Hydration Status of Top Level Jockeys on a Racing and Non-Racing Day", *Medicine and Science in Sport and Exercise*, vol. 39, no. 5.

Dolan, E., Warrington, G., McGoldrick, A., O'Loughlin, G., & Moyna, N. 2008, "An Analysis of the Energy Balance of Professional Flat Jockeys on a Competitive Race Day", *Medicine and Science in Sport and Exercise*, vol. 40, no. 5, p. S14.

Warrington, G., Dolan, E., McGoldrick, A., May, G., & Fitzpatrick, P. 2009a, "Acute Weight Loss Patterns By Professional Horse Racing Jockeys in Preparation for Racing", *Medicine and Science in Sport and Exercise*, vol. 41, no. 5, p. S563.

Warrington, G. D., McManus, C., Griffin, M. G., McGoldrick, P. A., & Lyons, D. 2006, "Bone Mineral Density and Body Composition Characteristics of Top-Level Jockeys", *Medicine and Science in Sport and Exercise*, vol. 38, no. 5.

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List of Abbreviations

ACTH: Adrenocorticotrophic Hormone
ADH: Anti Diuretic hormone
AGRP: Agouti Related Protein
AMP: Adenosine Monophosphate
ANG II: Angiotensin II
ANP: Atrial Natriuretic Peptide
ASIS: Anterior Superior Iliac Spine
AVP: Arginine Vasopressin
BA: Bone Area
BAT: Bioavailable Testosterone
BD: Body Density
BF: Body Fat
BMAD: Bone Mineral Apparent Density
BMC: Bone Mineral Concentration
BMD: Bone Mineral Density
CART: Cocaine and Amphetamine Regulated Transcript
CRT: Choice Reaction Time
DHEA: Dehydroepiandrosterone
DXA: Dual Energy X-Ray Absorptiometry
EAR: Estimated Average Requirement
FM: Fat Mass
FMI: Fat Mass Index
FN: Femoral Neck
FN BMD: Femoral Neck Bone Mineral Density
FN BMC: Femoral Neck Bone Mineral Content
FN BA: Femoral Neck Bone Area
FSH: Follicle Stimulating Hormone
FT: Free Testosterone
FT4: Free T4
GH: Growth Hormone
GHIH: Growth Hormone Inhibiting Hormone

GHRH: Growth Hormone Releasing Hormone
 GnRH: Gonadotropin Releasing Hormone
 HRI: Horse Racing Ireland
 Hz: Hertz
 IGF-1: Insulin Like Growth Factor-1
 IGFBP: Insulin Like Growth Factor Binding Protein
 LBM: Lean Body Mass
 LH: Lutenizing Hormone
 LMI: Lean Mass Index
 LTI: Lower Threshold Intake
 MES: Minimum Effective Strain
 NPY: Neuropeptide Y
 NTx: Cross Linked N-Telopeptide of Type 1 Collagen
 OPG: Osteoprotegerin
 P1NP: Total Procollagen Type 1 Amino Terminal Propeptide
 PAL: Physical Activity Level
 PPO: Peak Power Output
 QCT: Quantitative Computer Tomography
 RACE: Racing Academy and Centre of Education
 RANKL: Receptor Activator for Nuclear Factor κ B Ligand
 RMR: Resting Metabolic Rate
 RPE: Rating of Perceived Exertion
 RVIP: Rapid Visual Information Processing
 SHBG: Sex Hormone Binding Globulin
 SRT: Simple Reaction Time
 TBBMD: Total Body Bone Mineral Density
 TBBMC: Total Body Bone Mineral Content
 TBBA: Total Body Bone Area
 TNF α : Tumor Necrosis Factor-Alpha
 TSH: Thyroid Stimulating Hormone
 Usg: Urine Specific Gravity
 WBV: Whole Body Vibration
 WHO: World Health Organisation

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Chapter One: Introduction



1.1. Study Aim:

To examine the acute and chronic effects of weight restriction and weight cycling practices on physiological, metabolic, osteogenic and cognitive function in jockeys.

Objectives:

1. To examine the effect of an acute reduction in body mass on physiological and cognitive function.
2. To compare bone mass and body composition in flat and national hunt jockeys with that of an age, gender and BMI matched boxer and control group.
3. To assess bone mass, bone turnover and related endocrine function in a group of jockeys and an age, gender and BMI matched control group.
4. To examine the effects of six months whole body vibration therapy on bone mass and turnover in jockeys.

Hypothesis:

That both acute and chronic aspects of physiological, metabolic, osteogenic and cognitive function may be affected by a life of chronic weight restriction and weight cycling, which is apparently prevalent in jockeys.

1.2. Background Information and Justification

Making weight in sport refers to the practice of reducing body mass prior to competition (Fogelholm et al., 1993) and appears to have an impact on athletic performance if body mass loss is greater than that which is physiologically tolerable (Brownell et al., 1987). It has been suggested that any body mass loss necessary for competition should be undertaken in the off season (Sudi et al., 2004), over a period greater than seven days and that mass should be reduced from the adipose tissue compartment (Fogelholm, 1994). This is not always practical or possible however and the use of acute weight loss practices such as energy restriction and active and passive dehydrating techniques appear commonplace among weight category athletes (Alderman et al., 2004; Oppliger et al., 1996). A rapid reduction in body mass in preparation for competition may result in the loss of body water, substrate stores and

lean muscle mass, all of which are necessary for optimum performance (Nattiv et al., 2007; Oliver et al., 2007; Sawka et al., 2007). A rapid reduction of body mass in order to comply with stipulated competition weight standards has previously been shown to have a detrimental impact on athletic performance in a number of sports, including judo (Filaire et al., 2001; Umeda et al., 2004), boxing (Hall and Lane, 2001), lightweight rowing (Burge et al., 1993; Slater et al., 2007; Slater et al., 2005; Slater et al., 2006) and wrestling (Oppliger et al., 1996). In contrast to other weight category sports, there appears to be a dearth of research available which investigates the effect of such weight loss techniques on jockeys.

Horse-racing is a unique example of a weight category sport by the virtue that jockeys must weigh-in at a designated weight immediately before and after each race that they compete in. Other weight classification sports are required to weigh-in prior to competition only. This weigh-in may take place up to 24 hours before competition thereby allowing the athlete time to replenish any energy and fluid stores which may have been depleted while making weight. This time between weigh-in and competition has previously been shown to attenuate the physiological strain associated with a rapid reduction in body mass, provided appropriate recovery strategies are implemented (Slater et al., 2007; Slater et al., 2006). This opportunity is not afforded to jockeys however who are required to weigh-in immediately before and after every race in which they ride. Weight allocations or “handicaps” are based solely on the ability of the horse and are designed to maximise the competitiveness of professional racing. Rather than competing in a specific weight category jockeys are required to align their body mass with that allocated to their designated mount in each race. Furthermore, in contrast to other weight category sports, professional horse-racing takes place seven days a week, and has no definable off-season, providing little respite for jockeys from the rigours of making weight. The need to relentlessly align body mass with strict competition limits can at times necessitate the use of strict and potentially dangerous weight management strategies by these athletes, in order to maximise their riding opportunities (Hill & O’Connor, 1998; King & Mezey, 1987).

Current weight allocations in horse-racing appear to be based more on tradition than on sound scientific principles. In light of the ever increasing size and stature of the Irish (Whelton et al., 2007) and worldwide population, along with advances in sport

scientific and medical knowledge, it has been suggested that these weight restrictions, and the methods used to achieve them may be antiquated and potentially dangerous (Warrington et al., 2009b). Previous research suggests that jockeys appear to follow a chronically energy restricted lifestyle (Dolan et al., 2010; Leydon & Wall, 2002) accompanied by regular use of rapid weight loss strategies, including both active and passive dehydration, such as saunas, exercising while wearing heavy clothing or sweat suits as well as the use of diuretics and laxatives (Dolan et al., 2010; Labadarios, 1988; Moore et al., 2002). It is likely that such a lifestyle may have a negative impact on physiological (Filaire et al., 2001; Hall & Lane, 2001), metabolic (Degoutte et al., 2006; Umeda et al., 2004), osteogenic (Walberg Rankin, 2006; Warrington et al., 2009b) and cognitive (Choma et al., 1998) function. Low bone mass in jockeys has previously been suggested as an adverse effect of a chronically weight restricted lifestyle (Leydon et al., 2002; Warrington et al., 2009b). There appears to be a dearth of population specific research available however, which examines the impact of such practices on jockeys. The literature suggests that both acute and chronic aspects of physiological, metabolic, osteogenic and cognitive function may be affected by a life of chronic weight restriction and weight cycling, as is apparently prevalent in this group. The aim of this study therefore was to examine such parameters in jockeys, so to identify if any acute or chronic adaptations occur in response to the lifestyle which they lead. This primary aim was achieved through the implementation of four independent, though related studies. Specific aims, objectives and hypotheses of each of these studies are outlined in the following section.

1.3. Study One: The Effects of a Four % Reduction in Body Mass in 48 Hours on Physiological and Cognitive Function in Jockeys

Aims and Objectives:

The aim of this study was to determine the extent of body mass reduction habitually undertaken by jockeys and to examine the effect of a four % reduction in body mass in 48 hours on physiological and cognitive function in a group of professional jockeys.

Objective One:

To assess the typical magnitude of weight loss adopted by jockeys in preparation for racing using a self report questionnaire.

Objective Two:

To identify weight loss practices used through the maintenance of a food and weight loss diary throughout the period of weight reduction.

Objective Three:

To examine the impact of a four % reduction in body mass on physiological function in a group of jockeys, as assessed through performance on an incremental cycle ergometer test to volitional fatigue.

Objective Four:

To examine the impact of a four % reduction in body mass on cognitive function as assessed through performance on a laptop based cognitive test battery.

Hypothesis:

That reducing body mass by the magnitude indicated and time allowed would have a negative impact on both physiological and cognitive function in this group of jockeys.

1.4. Study Two: A Comparison of Bone Mass between Professional Jockeys, Elite Amateur Boxers and Age, Sex and BMI Matched Controls

Aims and Objectives:

The aim of this study was to assess differences in bone mass and body composition in two different types of weight category athletes (professional jockeys and elite amateur boxers) and a group of age, gender and BMI matched controls.

Objective One:

To compare bone mineral density (BMD), bone mineral concentration (BMC) and bone area (BA) of the four groups as indicated by DXA scanning.

Objective Two:

To normalise bone mass results to reflect the size and stature of the groups so to identify whether differences exist independently of variations in body composition.

Hypothesis:

That differences in bone mass may exist between the groups, with bone mass being shown to be reduced in the two jockey groups and enhanced in the boxer group and that these findings may exist independently of variations in body stature and composition.

1.5. Study Three: An Analysis of Bone Mass, Turnover and Metabolism in Jockeys

Aims and Objectives:

The aim of this study was to examine a number of reproductive and metabolic endocrine factors in a group of jockeys and to identify whether any of these factors may influence bone regulation in this group.

Objective One:

To evaluate bone mass in a group of jockeys and age, gender and BMI matched controls.

Objective Two:

To evaluate bone turnover in a group of jockeys and age, gender and BMI matched controls.

Objective Three:

To examine the status of a number of essential reproductive and metabolic hormones associated with bone metabolism in a group of jockeys and age, gender and BMI matched controls.

Hypothesis:

That endocrine levels of a number of reproductive and metabolic hormones may be disrupted in the jockey group and that this may impact on bone mass and regulation.

1.6. Study Four: The Effects of Six Months Whole Body Vibration Therapy on Bone Mass and Turnover in a Group of Jockeys

Aims and Objectives:

The aim of this study was to examine the impact of six months whole body vibration therapy on bone mass and turnover in a group of professional jockeys.

Objective One:

To assess the impact of six months of whole body vibration therapy at 30 Hz and 0.3g on bone mass and body composition in a group of professional jockeys

Objective Two:

To assess the impact of six months of whole body vibration therapy at 30 Hz and 0.3g on bone turnover in a group of professional jockeys

Hypothesis:

That WBV therapy will have a positive osteogenic effect on bone mass and/or turnover in this group.

1.7. Delimitations

- ⇒ This study was restricted to professional jockeys involved in thoroughbred horse-racing and currently licensed by the Irish Turf Club and did not include any other types of jockeys, such as amateur riders, point to point jockeys or pony racers.
- ⇒ This study was restricted to male jockeys as female jockeys are in the minority in Ireland

1.8. Limitations

The specific limitations associated with each experiment are included in the individual study discussions (see Chapters 3 – 6).

Chapter Two: Review of Literature



Introduction

Horse-racing is a weight category sport and as such jockeys are subject to many of the challenges typically associated with participation in sports of this nature. The unique characteristics of, and issues facing weight category athletes, and jockeys in particular will be explored in the forthcoming chapter, with specific reference to the potential impact of “making weight” and weight cycling on physiological, osteogenic, metabolic and cognitive function. This review will begin with a description of horse-racing and describe some of the challenges and issues typically associated with jockeys. A more detailed overview of weight category sports will follow, including the negative effects associated with “making weight” for sports performance. It appears that dehydration and energy deficiency are common consequences of making weight for sports performance. The physiological and performance related implications of a hypo-hydrated and hypo-energy state for athletes will be discussed, along with the potential endocrine response to weight cycling. This review will conclude with an examination of bone function and regulation, and the potential impact of weight restriction and weight cycling practices on bone health and metabolism. The aim of this review therefore is to examine the potential physiological, metabolic, osteogenic and cognitive effects of weight restriction and weight cycling and as such is intended to provide background information to the four studies which were conducted as part of this thesis.

1. Horse-Racing – A Weight Category Sport

Competitive horse-racing is one of the first known competitive sports, originating among the prehistoric nomadic tribesmen of Central Asia, who first domesticated the horse around 4500 BC. Horse-racing, often referred to as “The Sport of Kings”, has developed and flourished in the intervening years, and is today one of the worlds most popular sports, attracting followers from every age, nationality and walk of life. Thoroughbred horse-racing is one of Irelands most popular sports and one in which this country can claim significant international success. In Ireland in 2009, 345 race meetings were held, attracting a total attendance of 1, 237, 171 people. A total of €174 million euros was placed in on course betting in 2009, with an additional €3, 099 million placed in off course bets (HRI, 2009). There are currently 6,483 registered thoroughbred racehorses in training in Ireland. Irish racehorses compete in two main

types of races, namely flat and national hunt. Horses that race on the flat generally start running as two - three year olds. Races range from distances of 5 – 20 furlongs (1 furlong = 1/8 of a mile/0.201 km), and consist of a straight run with no obstacles. In contrast, horses competing in national hunt races typically do not begin racing until they are four - five years old. Races are at least two miles long, throughout which the horse must jump a number of obstacles in the form of hurdles or fences. All of today's thoroughbred race-horses descend from one of three Arabian stallions, known as the "foundation sires", demonstrating the depth of history and culture entrenched within this sport. These horses were imported into England from the Mediterranean Middle East around the turn of the 17th century and bred with native stock to produce the famed thoroughbred horse. The foundation sires, named the Godolphin Arabian, The Byerley Turk and the Darley Arabian, form the foundation of the thoroughbred horse as it is known today, combining the speed and endurance of the Arabian horse with the strength of the native stock, so producing the ultimate racing animal.

Irish horse-racing is governed by Horse-Racing Ireland (HRI), which is a commercial, semi-state body designed to oversee the organisation and development of Irish horse-racing and whose mission is to "develop and promote Ireland as a world centre of excellence for horse-racing and breeding". The Turf Club is the chief regulatory body of Irish horse-racing and is a private body, whose main aim is to ensure the reputation and integrity of Irish horse-racing. This is achieved through the implementation, amendment and overseeing of the rules governing horse-racing. The Racing Academy and Centre of Education (RACE) is the designated training body of the Irish Turf Club and has responsibility for the provision of training and development courses for trainee, apprentice and professional jockeys, breeders, trainers, stable staff and farriers.

1.1. "Handicapping" or Weight Allocation in Horse-Racing

Horse-racing is a weight category sport for which its athletes (jockeys) must "weigh-in" at a designated weight immediately before and after each race in which they compete. Weight allocations or "handicaps" are based solely on the ability of the horse and are designed to maximise the competitiveness of the sport. Professional jockeys do not compete in a specific weight category but must align their own body mass with

that allocated to their designated mount in each race. The need to relentlessly align body mass within racing limits may encourage the use of potentially dangerous acute weight loss strategies by jockeys in an attempt to optimise riding opportunities (Hill & O'Connor, 1998; King & Mezey, 1987). Weight allocations in Irish horse-racing currently range from 52.7 - 64 and 62 - 76 kg for flat and national hunt jockeys respectively. Declarations of all entries and stipulated weights must be made before 10 am on the day before a race meeting. Jockeys must weigh-in for racing fully clothed, wearing riding boots, back protector and carrying their saddle. In addition trainers and owners are encouraged to allocate rides to apprentice jockeys through the "claiming allowance" incentive, whereby apprentice jockeys may "claim" up to a 10lb/4.5kg reduction to their mounts' allocated racing handicap. The size of this allowance is progressively reduced in accordance with the number of wins an apprentice accumulates. This practice may represent a significant challenge to the young athlete in that they must strive to attain minimal weights at a critical growth and maturational phase.

Weight allocations appear to be dictated largely by tradition and have remained relatively static in Irish racing over many years. Secular increases in mass and stature of the Irish population (Whelton et al., 2007), along with evidence supporting increasing difficulty and health issues associated with weight making (Warrington et al., 2009b), indicate that current weight limits may be antiquated and in need of revision. Analysis of the records of trainee jockeys entering the Racing Academy and Centre of Education (RACE) in Ireland has revealed that that over the past 30 years the mean body mass of apprentice jockeys has increased by 30lbs (37%) whilst in the same time period the minimum weight for flat jockeys has risen by just 6%.

1.2. Horse-Racing Jockeys and the Typical Challenges Facing Them

The demands placed on professional jockeys appear to be ever increasing, as is evident by the long racing season and intensive competition programme. One of the key challenges facing jockeys is the pressure of making weight and remaining at weight throughout the prolonged racing season. Making weight is the practice used by athletes from weight category sports to allow them to compete at the stipulated weight standard. The major difference between horse-racing and other weight category sports such as

lightweight rowing, boxing and other combat sports however is that while weight loss occurs in all cases, other weight classification sports are required to weigh-in prior to competition only. Additionally, this weigh-in may take place up to 24 hours before competition, thereby allowing the athlete time to replenish energy and fluid stores depleted when making weight. This time between weigh-in and competition has previously been shown to attenuate the physiological strain associated with a rapid reduction in body mass, provided appropriate recovery strategies are employed (Slater et al., 2007; Slater et al., 2006). This opportunity is not afforded to jockeys however who have to weigh-in immediately before and after each race that they ride, which may be as many as five - seven races per day. The situation is further compounded by the virtue that jockeys race at weight throughout the week, and in many cases over a 10-12 month racing season. In contrast, most other weight category sports have a defined competitive season which may only last four – six months and contain a small number of major competitions, so allowing the athletes to regain some weight between competitions and in the off-season.

1.2.1. Weight Regulation Practices

Evidence suggests that the primary method used by jockeys to make weight is dehydration by a number of different mechanisms, accompanied by severely restricted fluid and food intake (Dolan et al., 2010; Hill & O'Connor, 1998; Leydon & Wall, 2002; Moore et al., 2002). Such methods of weight regulation appear to be consistent with research conducted in other weight making sports such as wrestling, boxing and judo (Burge et al., 1993; Filaire et al., 2001; Oppliger et al., 1996) and have been shown to have a negative impact on performance in a number of sports.

Recent research in a group of Irish jockeys (Dolan et al., 2010) revealed that the most commonly used acute weight loss techniques included saunas (86%), exercising to sweat (81%), restricted energy intake (71%), wearing plastic (43%), excessive exercising (38%) and self induced vomiting (14%). No participant in this study reported utilising a weight loss strategy greater than four days prior to race-day, with the majority (86%) aiming to attain the required body mass acutely and within 24–48 hours of or on the race-day itself. Rapid weight loss via food and fluid restriction may

compromise hydration status, muscle and liver glycogen stores and potentially circulating blood glucose concentration all of which are necessary for maintenance of usual physiological (Sawka et al., 2007); cognitive (Choma et al., 1998; Gopinathan et al., 1988), metabolic (Misra et al., 2003) and osteogenic (Ihle & Loucks, 2004) function. Participants in this study reported experiencing many negative side effects from making weight, including thirst (52%), dehydration (43%), hunger (38%), headaches (29%) and fatigue (25%) (Dolan et al., 2010). The reported methods of rapid weight loss reported in this study were consistent with those previously observed in similar studies in South African (Labadarios, 1988), New Zealand (Leydon & Wall, 2002) and Australian (Moore et al., 2002) jockeys.

1.2.2. Nutritional Practices:

Chronically restricted energy intake appears to be prevalent among jockeys (Dolan et al., 2010; Leydon & Wall, 2002). Recent research showed that total estimated calorie and carbohydrate intake, as measured through analysis of seven day food diaries appeared to be quite low in a group of Irish jockeys (Dolan et al., 2010) when considered in accordance with estimated resting metabolic rate (Mifflin et al., 1990) and general athletic recommendations (ADA, 2009). Macronutrient intakes in this sample of Irish jockeys were consistent with those previously reported for jockeys in New Zealand (Leydon & Wall, 2002) and South Africa (Labadarios, 1988). Micronutrient intake in this sample was also very low. More than 50% of the sample failed to meet the Irish estimated average requirement (EAR) for vitamins A and C, riboflavin, folate, calcium and zinc (FSAI, 1999). A concerning proportion of jockeys also consumed less than the lower threshold intake (LTI) for vitamins A (27%) and C (38%), folate (55%), calcium and zinc (11%). Low intake of vitamins A, C and folate was likely due to an inadequate consumption of fruit and vegetables, which only ranged up to two servings a day when the recommended intake is five or more servings per day (WHO & FAO, 2003).

Disordered eating appears to be prevalent in jockeys (Dolan et al., 2010; Labadarios, 1988; Leydon & Wall, 2002), which has been proposed as a risk factor for development of clinical eating disorders such as anorexia and bulimia nervosa

(Boisseau, 2006; Caulfield & Karageorghis, 2008; Rouveix et al., 2006). This theory was not supported by the work of King & Mezey (1987) who examined the psychological effects of erratic and irregular eating habits in a group of English male jockeys. Results of this study suggested that weight making practices and disordered eating in jockeys do not necessarily increase the risk of development of clinical eating disorders such as anorexia nervosa or bulimia, which are characterized by severe psychological disturbances, as well as disordered eating patterns (Beals & Houkooper, 2006). This finding was consistent with the views of Dale & Lander (1999), who suggested that disordered eating among wrestlers may impact on physiological function but did not necessarily represent a threat to psychological and emotive integrity (Dale & Lander, 1999). More recently however, research by Caulfield & Karageoris (2008) suggests that the psychological well being of jockeys may be at risk due to weight making practices as evidenced by more negative mood states and disordered eating attitudes when riding at minimum weight.

1.2.3. Injury Risk and Implications of “Making Weight”

Horse-racing is classified as a high risk sport (Hitchens et al., 2009; Waller et al., 2000). Thoroughbred horses typically weigh up to 500kg, are capable of velocities in excess of 60 km/h and are often ridden by jockeys weighing as little as 50 kg. Soft tissue injuries and fractures have been reported as being the most common form of injury experienced by jockeys during participation in their sport (McCrory et al., 2006; Turner et al., 2002). Falls appear to be most common in national hunt races and it has been estimated that national hunt riders in Ireland may fall off their horses in six % of all rides (Turf Club Chief Medical Officer, personal communication). It appears that the majority of injuries to flat jockeys occur either in the starting gate, or in the home stretch or finish line. It is assumed that an excited horse, in a small confined space may predispose the jockey to a greater risk of being crushed against a hard surface when in the starting stalls. While no obstacles are present in the home stretch or finish line, the greater speed and close proximity of the horses, in addition to the final risk taking tactics of the jockey make injury more likely (Waller et al., 2000).



The velocities attained by the horse coupled with the positioning of the jockey have been suggested as the primary cause of falls and accidents in this sport (Waller et al., 2000). Jockeys are positioned in a forward stance, approximately three metres above the ground, and are in a state of dynamic imbalance while racing. Any sudden change in direction or speed of the horse may result in the rider being pitched headfirst into the ground from a height, and at a relatively high velocity (Jenkinson & Jenkinson 2001). While there is little doubt that horse-racing is an extremely high risk sport it has been suggested that the risks associated with participation in this sport may be exacerbated through the use of acute weight loss strategies (Warrington et al., 2009b). In particular factors such as dehydration and low bone mass, which have been identified in this group (Warrington et al., 2009) may have particular implications for jockeys in light of the high-risk nature of the sport.

1.3. Weight Category Sports

Horse-racing is a unique example of a weight category sport, due mainly to the length and intensity of the racing season. Other examples of weight category sports include lightweight rowing, boxing, wrestling and judo. “Making weight” refers to the practice of reducing body mass prior to competition and is reported to be undertaken by weight category athletes for three reasons: to compete in a lower weight class; to improve aesthetic appearance or to improve physical performance (Fogelhom et al., 1993). The practice of making weight may place a significant physiological and psychological

stress on the athlete, depending on a number of factors including the amount of body mass to be lost; the time available and the mechanisms employed in order to achieve the required weight loss. Moderate restrictions in energy intake may induce a loss in body mass without adversely affecting performance (Zachwieja et al., 2001), however it appears that many weight category athletes attempt to dramatically reduce their body mass in the period immediately preceding competition in quantities far in excess of what the body may safely tolerate (Alderman et al., 2004; Maffuli, 1992; Warrington et al., 2009b). The physiological and performance related consequences of “making weight” for sport will be discussed in the forthcoming section.

1.4. Gradual Vs Rapid Weight Loss

The amount of time available in which to reduce body mass and the practices adopted may have a significant impact on the effect of body mass loss. Gradual weight loss refers to a targeted reduction in body mass over an extended period greater than seven days, while rapid weight loss occurs within seven days (Fogelholm et al., 1993). It is recommended that athletes who are required to reduce body mass for competition do so gradually (Fogelholm, 1994) and that any excess levels should be lost in the off-season, so that body mass maintenance is the primary concern during the competitive season (Sudi et al., 2004). Gradual body mass loss may be achieved through a combination of reduced energy intake and increased energy expenditure, as the resultant negative energy balance should result in the oxidation of adipose tissue. The oxidation of adipose tissue takes time however, and body mass lost rapidly may necessitate the loss of body water and lean muscle mass (McCargar et al., 1993; Slater et al., 2006), both of which are necessary for optimum athletic performance. Van Italie and Yang (1977) suggest that in the first ten days of fasting, 54 – 58% of body mass loss comprises total body water, 30 – 35% adipose tissue stores and 6 – 16% protein.

Restricted fluid and fuel intake, along with active and passive dehydrating practices appear to be the most commonly employed methods used by weight category athletes in an attempt to make weight for competition (Dolan et al., 2010; Fogelholm, 1994; Fogelholm, 1993; Hall & Lane, 2001; Oppliger et al., 1996). Such practices may have a detrimental impact on performance, and many of the more dangerous practices employed by these athletes, such as self-induced vomiting, laxative and diuretic use,

excessive exercise and fasting are consistent with the symptoms of those suffering from eating disorders such as anorexia and bulimia nervosa (Beals & Houkoooper, 2006). While psychologically these athletes appear not to be at an increased risk for development of such eating disorders, physiologically they are placing their health at a major risk by engaging in such practices (Dale & Lander, 1999).

1.5. Rapid Weight Loss and Sports Performance

A rapid reduction in body mass in order to comply with stipulated competition standards has been associated with decreased athletic performance in a number of sports including judo (Filaire et al., 2001; Umeda et al., 2004), boxing (Hall and Lane, 2001), lightweight rowing (Burge et al., 1993) and wrestling (Oppliger et al., 1996). Filaire et al (2001) demonstrated a negative physiological effect of a five % reduction in body mass induced by seven days of food restriction in a group of judoists on a 30, but not seven second maximal jumping test. This finding was in agreement with research demonstrating detriments to bouts of repetitive judo movements lasting 30 seconds or longer, following body mass loss for competition achieved through a combination of gradual and rapid weight loss techniques (Koral and Dosseville, 2009). Burge et al (1993) noted a 22 second decrease in maximal rowing time following a 5% loss of body mass within 24 hours. Performance decrements were attributed mainly to reduced plasma volume as a result of dehydration and a reduction in muscle glycogen (Burge et al., 1993). Further research by Slater et al (2005), partially supported this research, showing performance decrements in rowing time to exhaustion following acute weight loss (4% in 24 hrs). The extent of the performance decrement was much smaller however than that reported by Burge et al (1993) and this was thought to be due to the aggressive nutritional recovery strategies which were used between weigh-in and maximal performance testing in this study (Slater et al., 2005). Further research undertaken to test the validity of this assumption employed the use of different nutritional recovery strategies between body mass losses and testing of maximal rowing performance. Results suggest that intake of fluid, carbohydrates and sodium in accordance with current guidelines may represent the most effective recovery strategy, with fluid replacement appearing to be the most critical component of nutritional recovery following weigh-in (Slater et al., 2006), although enhancement of psychological and motivational factors may also have been involved.

Emotive (Hall & Lane, 2001; Horswill et al., 1990) and cognitive (Choma et al., 1998) function have also been shown to be affected by a rapid reduction in body mass in preparation for competition. Boxing performance was shown to be unaffected by a 5% body mass loss in one week in a study by Hall and Lane (2001), however subjective ratings of anger, fatigue, confusion and tension were demonstrated as being negatively affected in this study in agreement with the findings of others (Filaire et al., 2001; Horswill et al., 1990). Choma et al. (1998), demonstrated impairment to short term memory and mood state following a 5% body mass loss for competition in a group of competitive wrestlers. It was thought that changes in exercise behaviour, reduced blood volume, sleep disturbances or hypoglycaemia may all have had a role to play in this finding (Choma et al., 1998).

Sport imposed body mass restrictions in childhood and adolescence may have serious and potentially irreversible consequences in later life. These include alterations in endocrine and metabolic parameters affecting growth, maturation and development of peak bone mass, emotional disturbances and increased risk of development of psychological disorders such as anorexia and bulimia nervosa, disturbances to reproductive function and an increased risk of chronic injury (Boisseau, 2006).

In addition to the performance implications of “making weight”, acutely cutting body mass for competition may have severe health implications if taken to the extreme. Excessive dehydration in an attempt to make competition weight has been implicated in the deaths of three American collegiate wrestlers (Anonymous, 1998). It has also been suggested that excessive weight control and dehydration were responsible for the deaths of two young jockeys in America, one of whom collapsed after a race due to severe dehydration (Finley, 2005; Scheinman, 2005), the other who was said to suffer a fatal heart arrhythmia caused by potassium deficiency developed through chronic weight control and energy restriction (Finley, 2000).

Summary

It appears that jockeys are subject to many of the challenges typically associated with participation in weight category sports. In addition, the detrimental effects of making weight may be exacerbated in jockeys due to the length and intensity of the racing

season. Restricted fluid and food intake, along with dehydration by a variety of mechanisms appear to be the weight loss practices most commonly employed. The implications of dehydration and energy deficiency on athletic performance will be discussed in the following section.

2. Fluid and Fuel Requirements for Sport and Health

2.1. Fuel Requirements

Appropriate nutritional and caloric intake is essential for maintenance of usual physiological function and everyday health as food provides the energy and substrate required for the completion of biological work. All athletes, regardless of age, gender or type of sport, must consume enough food to cover the energy costs of daily living, the energy cost of their sport and the energy costs associated with building and repairing muscle tissue (Burke and Deakin, 2006). Energy balance is tightly regulated within the body, in order to maintain a stable body mass (Shin et al., 2009) and an upper adiposity level (Speakman, 2004). This balance is regulated through a variety of homeostatic, metabolic and non-homeostatic mechanisms. Such factors may represent a significant challenge to any athlete attempting to achieve a body mass lower than that which is homeostatically and genetically programmed. The forthcoming sections provide a brief overview and description of the principal factors involved in energy and body mass regulation and the potential impact of insufficient energy intake on health and athletic performance.

2.2. Weight and Energy Regulation

Unless actively attempting to achieve losses or gains in body mass, athletic energy intake and expenditure should be matched so to ensure a stable body mass and composition (ADA, 2009). As meal intake is often more strongly influenced by circadian and sensory factors than by actual requirements however, discrepancies often occur in short term energy balance. The body appears capable of overcoming short term imbalances however, so allowing body composition remain relatively static over the long term (Shin et al., 2009). The adipose tissue acts as a homeostatic regulator of this point, through the action of its secreted adipokines, including leptin and adiponectin (Rosen & Spiegelman, 2006). These factors appear to relay signals to the hypothalamus primarily, along with other neural systems and brain areas which act as control centres for the energy regulatory system (Berthoud, 2002). Appropriate neural and hormonal factors are then employed, so to adjust energy intake and expenditure in accordance with homeostatic requirements (Harris et al., 1990). A number of non-

homeostatic factors are present however (Speakman, 2004) which allow development of energy imbalances. For example, activation of the mesolimbic dopamine system or “wanting” food is necessary to elicit the motor behaviour necessary to obtain pleasurable stimuli (including physiologically beneficial foods) and this activation may be triggered by psychological factors such as pleasant associative memories (Berthoud, 2007). It has been suggested that non-homeostatic neural signals may be capable of over-riding metabolic signals in an attempt to protect an upper adiposity level (Speakman, 2004). This may reflect an “opportunistic” relationship with food, developed in response to repeated exposure to famine throughout the evolutionary process (Berthoud, 2007).

A number of hormones have been identified as being involved in the regulation of energy balance. One in particular which has received a considerable amount of scientific attention is leptin. The action and function of this hormone will be described in greater detail in the forthcoming section.

2.2.1. Leptin

Leptin is a protein hormone secreted primarily from the adipose tissue. Leptin is the ob gene product, is secreted in a stable pulsatile fashion and is believed to be involved in long term energy regulation. Leptin levels are reduced during fasting as a result of decreased basal secretion and secretory burst mass (Misra et al., 2005) and this reduction appears to encourage energy conservation and promotion of feeding behaviours (Rosen & Spiegelman, 2006). Although secreted primarily from the adipose tissue research indicates that its effects may also occur independently of the size of the adipose tissue (Baylor & Hackney, 2003). It seems that the adipose tissue may amplify leptin response but that pulsatile secretion is determined by additional factors (Koutkia et al., 2003) of which energy availability is likely to be its main determinant (Borer et al., 2009; Romon et al., 1999; Thong et al., 2000). The primary role of leptin appears to be in the regulation of energy balance, although it is also involved in numerous other elements, including reproduction, angiogenesis, immunity, wound healing and cardiovascular function (Gomez-Ambrosi et al., 2008).

Leptin exerts its energy regulatory action through binding to hypothalamic receptors, so activating effector systems, which exert an influence on both energy intake and expenditure. Leptin receptors are located on two populations of hypothalamic neurons located in the arcuate area; those that control the actions of the oreogenic peptides NPY and AgRP and the anorexigenic neuropeptides such as α MSH and CART. It therefore acts to simultaneously suppress and stimulate the actions of the oreogenic and anorexigenic neuropeptides respectively (Jequier, 2002). Leptin appears to act as a tonic long term endogenous regulator of energy balance and as such does not appear to be affected by episodic shorter term exogenous factors such as meal size and satiety in humans (Jequier, 2002). Meal energy content has however been shown to have an effect on leptin response, in that a high carbohydrate meal was shown to induce a greater postprandial leptin effect than an isocaloric but high fat meal (Romon et al., 1999) which was likely related to the available energy content of the two meals. Leptin seems to exert its most potent effect at low levels (Arch, 2005) and is less capable of protecting against the effects of excess energy intake, likely due to a naturally derived leptin resistance (Klein et al., 2000; Levin et al., 2003; Russell et al., 2001).

As with any hormone, leptin does not act in an isolated fashion and its actions are regulated by and in turn regulate action of a number of different hormones. For example, leptin and thyroid hormone appear to exert similar effects and it was suggested that these hormones may be related to each other (Baylor & Hackney, 2003). Animal models however suggest that their effects are additive, but independent (Wang et al., 2000). A study examining the synchronicity of leptin pulsatility with that of growth hormone, cortisol and insulin showed that leptin pulsatility was synchronous with and primary to that of growth hormone and cortisol but lagged behind that of insulin. This finding suggests that leptin is regulated by nutritional signals, including insulin response and in turn regulates the action of the metabolic hormones such as growth hormone and cortisol; supporting the hypothesis that leptin acts as a secondary messenger relaying critical nutritional information to higher neuroendocrine centres (Koutkia et al., 2003). Data from this study agree with research examining leptin secretory dynamics in a group of adolescent girls with anorexia nervosa and a group of healthy controls, which reported that leptin was strongly predicted by measures of nutritional status such as % body fat and insulin resistance and in turn regulated other

nutritionally regulated hormones such as growth hormone and cortisol (Misra et al., 2005).

2.2.2. Body Composition

The extent of the body fat compartment has been suggested as being indicative of the stored energy in that body and imbalances between intake and expenditure are largely absorbed by this compartment (Flatt, 1995). The adipose tissue is the most efficient energy storage compartment within the body due to its high energy content and hydrophobic orientation (Saladin, 1999a). Optimal competitive body weight and composition varies considerably according to individual requirements and predispositions and should be determined when an athlete is healthy and performing to their personal best. Minimum body fat levels necessary for health and usual metabolic function have been suggested as 5% for men and 12% for women (ADA, 2009), although individual variability may occur.

2.3. Energy Restriction and Physiological Function

Short term energy restriction (48 hours) is known to negatively impact on exercise performance (Oliver et al., 2007), while a more chronic negative energy balance state may manifest in a number of adverse consequences, the extent of which may depend on the length and severity of energy restriction. These include: muscle mass loss (Finn and Dice, 2006; Lowry et al., 1984); reproductive dysfunction (De Souza & Williams, 2004; Wade et al., 1996); bone loss or failure to reach peak bone mass (Bolton et al., 2005; Hamrick et al., 2008); increased susceptibility to injury; fatigue or infection; a prolonged recovery process (Lowry et al., 1984) and decreased immune function (Ohta et al., 2002) and may even result in cardiac impairments or failure if taken to the extreme (Schocken et al., 1989). Usual metabolic function is dependent on the state of energy availability. This refers to the proportion of energy required to sustain usual metabolic processes after the additional requirements of physical activity are accounted for. It has been suggested that a threshold of $30 \text{ kcal/kgLBM}\cdot\text{day}^{-1}$ is required for maintenance of usual metabolic and endocrine function (ADA, 2009; Loucks, 2003; Loucks & Thuma, 2003).

The components of the fuel mix oxidised and the relative contribution of the energy systems used during exercise are chosen so to ensure minimal changes in protein and glucose concentrations. This occurs due to the functional importance of protein in the body, and the need to ensure a sufficient supply of glucose to the brain. A negative energy balance may result in the use of less efficient energy systems, in order to preserve this function (Flatt, 1995). For example, severe energy restriction may induce protein oxidation (Finn & Dice, 2006; Lowry et al., 1984) so reducing fat free mass and muscular strength (Umeda et al., 2004).

Resting metabolic rate (RMR) appears to be disproportionately reduced when the body is in a state of energy deficiency (Deutz et al., 2000; Hall, 2006) and periods of prolonged underfeeding may be followed by a rapid and disproportionately high replenishment of body adipose tissue stores, likely due to an accelerated rate of *de novo* lipogenesis and an increase in *ad libitum* fat intake (Hall et al., 2008). Higher body fat levels have been demonstrated in energy deficient runners and gymnasts in contrast to a group of their energy replete counterparts (Deutz et al., 2000). A reduction in RMR during times of energy deficiency may reflect the tendency of the body to maintain energy balance under whatever conditions it is exposed to (Siervo et al., 2008), and this may occur independently of change in body mass (Weinsier et al., 2000). This study reported a return of body composition adjusted RMR values to baseline levels once energy balance was restored at the reduced body mass (Weinsier et al., 2000). It has been suggested that weight cycling, namely repeated phases of body mass loss and regain may enhance food efficiency in weight category athletes, by such mechanisms as a disproportionate decrease in RMR, so rendering weight loss and maintenance progressively more challenging (Brownell et al., 1987). Despite this however, a study by McCargar et al (1993) revealed that a period of weight cycling in a group of female lightweight rowers had no sustained effect on resting metabolic rate. Further research may be required so to more fully investigate the long term implications of weight cycling.

Low energy availability in female athletes causing a disruption to reproductive function and bone loss has been titled the “female athlete triad” (De Souza & Williams, 2004; Nattiv et al., 2007). The effect of inadequate energy intake on bone regulation will be discussed in greater detail in section 4, while it has been suggested that

disrupted reproductive function during times of inadequate energy intake may occur in an attempt to conserve energy for maintenance of more immediate and essential processes (Wade et al., 1996).

It is clear that inadequate energy intake has negative consequences for both general health and athletic performance, the extent of which may be dependent on an interaction between environmental and genetic influences (Heck et al., 2004). Weight category athletes need to be aware of the consequences of inadequate energy intake and ensure that competition performance is not adversely affected by insufficient nutrient intake in the time preceding competition.

2.4. The Role of Body Fluids

Quantitatively, water has been defined as the most important nutrient in the human body (Manz et al., 2002), comprising approximately 63% of total body mass and 80 – 84% of kidney, lung and skeletal muscle tissue (Armstrong et al., 2005). Body water has a variety of roles to fulfil within the body in order to ensure usual homeostatic function. Water acts as a transport medium via the blood allowing movement of the substrate required for and waste produced by metabolism. It also provides a reactive medium for a number of water soluble particles, offers lubrication and cushioning between the tissues (Versey et al., 2006) and has a key role to play in thermoregulation (Murray, 1996). Hydration status and cell size also appear to be involved in cellular metabolism. The cell swelling theory suggests that cellular volume may provide a signal for cell metabolic orientation. Excessive alterations of cell volume may compromise structural integrity so compromising metabolic function (Lang et al., 1998) and it has been suggested that cellular shrinkage as occurs during dehydration may induce a catabolic state (Lang et al., 1998; Ritz et al., 2005). It has been suggested however, that care must be taken in the interpretation of the relationship between hydration and metabolism as hypo-hydration is often accompanied by a hypo-energetic state making it difficult to separate their individual metabolic effects (Ritz & Berrut, 2005).

Deliberate or incidental dehydration often occurs when body mass is reduced rapidly for competition and has associated implications for physiological, cognitive and

metabolic function. Fluid, electrolyte and pH homeostasis are all tightly regulated through the body's water medium and irregularities of any of these three systems have many detrimental consequences for usual metabolic, physiological and cognitive processes. Balance between these three systems is controlled through the collective action of many of the systems of the body, namely the urinary, respiratory, digestive, integumentary, endocrine, nervous, cardiovascular and lymphatic systems.

2.5. Control of Body Fluid Homeostasis

Body water is distributed between the intra and extra cellular fluid spaces, with approximately 65% of total body water filling the intracellular fluid spaces (Versey et al., 2006). The remaining 35% is distributed throughout the interstitial fluid (25%), blood plasma and lymph (8%) and transcellular fluid (2%). Cell volume is maintained between the intra and extracellular fluid spaces through the osmotic pressure of the body tonomoles, of which sodium and potassium are the main extra and intra cellular tonomoles respectively (Mallie et al., 2002). Equilibrium between these electrolytes is maintained by the action of the membranous sodium-potassium ATPase pump (Lobo, 2004). Fluid volume is maintained within strict homeostatic limits, despite a daily turnover of approximately 2500 ml of fluid as illustrated in figure 2.1.

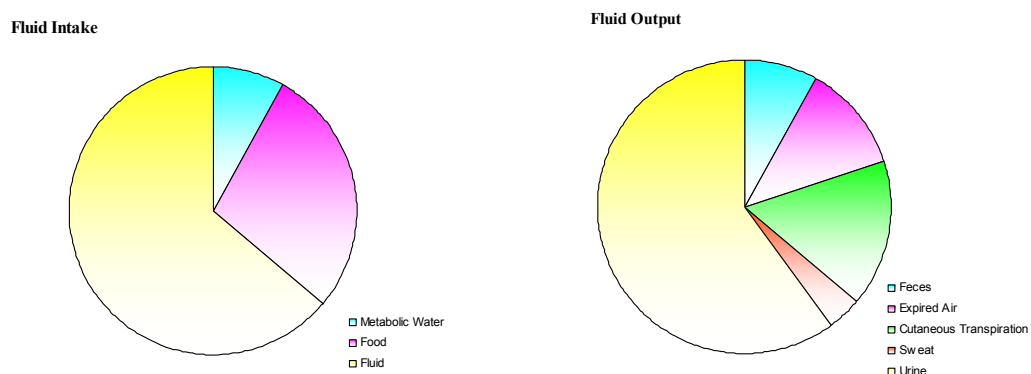


Figure 2.1. Typical Daily Fluid Intake and Output. Adapted from Saladin, 1999c

Fluid intake is primarily influenced through activation of the thirst mechanism which is regulated by the CNS, primarily the AV3V area, consisting of the region anterior and ventral to the third ventricle, containing the *organum vasculosum laminae*

terminalis (OVLT), subfornical organ (SFO) and the median preoptic nucleus of the hypothalamus (Antunes-Rodrigues et al., 2004). Blood osmo and volume receptors represent the primary activators of the thirst sensation (Lobo, 2004). Fluid output is primarily regulated through the urinary system which is composed of two kidneys, two ureters, the bladder and urethra. This system has responsibility for maintenance of fluid, electrolyte and pH homeostasis, waste elimination, blood pressure maintenance, erythrocyte count and blood PO₂ and PCO₂ (Saladin, 1999c).

An extremely complex neuroendocrine system is in place controlling the volume and composition of fluid intake and excretion. A comprehensive review of this system is described by Antunes-Rodrigues et al. (2004). Briefly however vasopressin (AVP)/anti diuretic hormone (ADH) and oxytocin represent the primary water retaining hormones. They are secreted simultaneously in response to hyperosmolarity and hypovolemia, where they both independently and synergistically exert an anti-diuretic and anti-natriuretic effect through an influence on collecting duct permeability (Hans et al., 1993). In addition to their water and sodium retaining actions, vasopressin also appears to exert a central dipsogenic effect (Szczepanska-Sadowska et al., 1982). AVP's dipsogenic effect is aided by that of angiotensin II, which is the primary component of the renin-angiotensin-aldosterone system and acts to increase blood pressure, thirst, sodium appetite and AVP and ACTH release in response to hypovolemia (Antunes-Rodrigues, 2004). The release of aldosterone is the final component of the renin-angiotensin system and it is released in response to increases in ANG-II and K⁺ levels. Aldosterone has a key role to play in the maintenance of electrolyte and fluid homeostasis and this is achieved through exerting an effect on the epithelia of the kidney and colon to regulate sodium absorption and K⁺ secretion (Booth et al., 2002). The atrial-natriuretic peptide (ANP) acts as an antagonist to the action of the renin-angiotensin-aldosterone system whereby it acts to acutely reduce plasma volume through increasing renal excretion of water and sodium so reducing blood pressure (Curry, 2005).

Fluid and electrolyte homeostasis may be achieved only through a coordinated effort and interaction between all of these primary elements. The complexity of this system may further be demonstrated however by evidence that a number of additional factors are present which also have a role to play in the regulation of fluid and electrolyte

balance. These include: bradykinin; dopamine; hypothalamic GABAergic neurons; endothelin; neurotensin; substance P; melanocyte stimulating hormone; neuropeptide Y and the opioid peptides. Further research may be required so to more fully elucidate the precise mechanisms by which fluid and electrolyte homeostasis are regulated (Antunes Rodrigues et al., 2004).

2.6. Hydration Status and Physiological Function

Evidence suggests that hydration state has a vital role to play in aerobic exercise (Casa et al., 2000; Coyle, 2004; Ebert et al., 2007; Sawka et al., 2007) and sports performance (Baker et al., 2007) and initiation of exercise in a dehydrated state is known to impact on performance (Armstrong et al., 2006; Moquin & Mazzeo, 2000). In addition, initiation of moderate to high intensity exercise in a hypo-hydrated state may also have an adverse effect on the cortisol-testosterone ratio, suggesting a catabolic effect of dehydration, in agreement with the cell swelling theory (Lang et al., 1998). Results from this study indicate that testosterone is largely unaffected by hydration state, but that cortisol response is closely correlated to the hydration state of the body (Maresh et al., 2006). It is generally acknowledged that performance decrements occur at a dehydration threshold of 2% body mass loss (Baker et al., 2007; Maughan & Shireffs, 2004; Sawka et al., 2007). Hyperosmotic hypovolemia is the most common form of dehydration caused by exercise as sweat composition is hypotonic to that of plasma (Sawka et al., 2007) and a number of mechanisms have been proposed to explain the effect of hydration state on physiological function, although precise effects remain to be determined (Casa et al., 2000; Coyle, 2004; Ebert et al., 2007; Sawka et al., 2007).

2.6.1. Hydration State and Sports Performance

The effect of hydration status on prolonged exercise performance which is predominantly aerobic in nature is well documented (Casa et al., 2000; Coyle, 2004; Ebert et al., 2007; Sawka et al., 2007). The detrimental effects of dehydration on aerobic exercise performance may be demonstrated by the reduced physical capacity (Ebert et al., 2007; Moquin & Mazzeo, 2000) and enhanced physiological strain demonstrated (Armstrong et al., 2006; Ebert et al., 2007; Kenefick et al., 2002). Mild

dehydration of approximately 1.5% body mass induced through exercising in a sweat suit and restricted fluid intake induced a shift in the lactate threshold toward a lower % of VO_2max in a group of moderately active, healthy females. The lactate threshold has been suggested as being a good predictor of endurance performance (Faude et al., 2009). This finding was accompanied by and correlated to a significant reduction in time to exhaustion (Moquin & Mazzeo, 2000). The results of Moquin and Mazzeo (2000) are supported by Kenefick et al. (2002), who reported a lowering of the lactate threshold to an earlier time and lower absolute VO_2 when exercising in a hypo-hydrated state, while Ebert et al. (2007) reported a noticeable increase in the rate of lactate accumulation when dehydrated by 2.5% body mass.

In contrast to aerobic exercise, anaerobic function, strength and power appears to be somewhat more robust to the detrimental effects of dehydration. Cheuvront et al., (2006) showed no change to 15 second single bout Wingate performance in response to either moderate dehydration or hyperthermia (Cheuvront et al., 2006). Dehydration of 2% body mass has been shown to have no effect on 50, 200 or 400 m sprint performance or in vertical jump performance, despite an increase in physiological strain experienced, as demonstrated by increased heart rate and lactate accumulation (Watson et al., 2005a). It has been suggested that dehydration of greater than the proposed threshold of 2% may be required in order to induce impairments to shorter duration, aerobically independent exercise (Yoshida et al., 2002). Simulated basketball performance, a sport comprising both aerobic and anaerobic elements has however been shown to be progressively affected by dehydration protocols ranging from 1 – 4%, with declines in performance reaching significance at a dehydration threshold of 2%. It was suggested that impaired mental attention and increased subjective feelings of fatigue may have contributed to the performance decrements reported in this study (Baker et al., 2007).

Somewhat contradictory to the well documented detrimental effects of dehydration on sports performance is evidence that highly trained and more successful marathon runners often complete races in dehydrated states of 4 – 8% (Coyle, 2004). It was proposed that the reduced body mass caused by fluid loss may have lowered the energy cost of running, potentially offsetting the detrimental effects of dehydration. This theory was investigated in a study by Armstrong et al. (2006), who examined the

effect of 5% hypo-hydration on running economy in a group of competitive runners exercising in a 23°C environment. Results indicated that hydration status had no measurable effect on running economy at either 70 or 85% VO_2max . Although VO_2 was unchanged in the hypo-hydrated trials, increased heart rate and norepinephrine values along with a reduction in cardiac output and stroke volume suggested increased physiological strain when exercising in a hypo-hydrated state. It was suggested that the highly trained nature of these runners may have enabled this group to overcome this increased stress, so allowing maintenance of running economy and lactate response (Armstrong et al., 2006). In support of this study maximal cycling hill performance, an exercise which theoretically may benefit from reduced body mass was shown to be compromised when the hill climb was initiated in a dehydrated state. This occurred despite a significantly reduced power output required to maintain a given racing speed, which occurred as a result of a loss in body mass (Ebert et al., 2007).

2.6.2. Mechanistic Effects of Dehydration on Exercise Performance

A number of postulated mechanistic explanations have been proposed in an attempt to explain the effect of hydration status on exercise performance. A key postulated mechanism is the reduction of plasma volume which occurs when water is lost from the body. Blood supply to active skeletal muscle is of paramount importance during exercise performance. Exercise represents a physiological strain which increases metabolic rate, so increasing blood transportation requirements. This is achieved through a responsive increase in cardiovascular function, namely increased stroke volume and a redirection of blood flow toward the working muscles. Dehydration reduces stroke volume and muscle blood flow however, so limiting blood supply (Coyle, 2004). Cardiovascular strain is routinely increased following dehydration and is evident in a number of studies (Armstrong et al., 2006; Ebert et al., 2007; Kenefick et al., 2002; Moquin & Mazzeo, 2000).

Metabolic alterations and changes in substrate utilisation may also occur when exercising in a dehydrated state. Dehydration appears to have an inhibitory effect on glycogen and protein synthesis (Shireffs et al., 2004) in accordance with Haussingers cell shrinkage theory (Lang et al., 1998). A reduced respiratory exchange ratio and a shift of substance use from carbohydrate to lipid sources when exercising in a

dehydrated state suggesting an increased reliance on lipid metabolism has also been reported (Armstrong et al., 2006; Cheung & McLellan, 1998), while dehydration may increase glycogen utilisation rate during exercise, so diminishing an already limited substrate supply (Coyle, 2004; Morris et al., 2005).

Reduced plasma volume and an increased glycogen utilisation rate do appear to be involved in dehydration associated performance decrements however they cannot be the primary limiting factor (Armstrong et al., 2006; Moquin and Mazzeo, 2000; Morris et al., 2005). Elevated core body temperature is a known consequence of exercising in a dehydrated state (Coyle, 2004; Ebert et al., 2007; Kay & Marino, 2000). This alone is capable of impairing exercise performance and may convey an additive effect when combined with dehydration (Morris et al., 2005).

An elevation in core body temperature when dehydrated appears to occur as a result of an impaired sweat response, causing disruption to thermoregulatory function (Greenleaf & Castle, 1971). During exercise evaporation through sweating is the primary avenue for heat loss and is achieved through a diversion of blood flow toward the skin by a dilation of the smooth muscles around the arterioles (Hoffman et al., 2002). The high sweat rates required to dissipate the heat created by the working muscles during exercise may induce dehydration. Dehydration and an associated cell shrinkage and reduction in plasma volume (Jimenez et al., 1999, Oppliger & Bartok, 2002) impairs the ability of the body to thermoregulate so increasing the risk of development of heat related illnesses such as heat exhaustion and stroke (Sawka et al., 2007). It appears that the CNS is capable of triggering an anticipatory adaptation in muscle force production, prior to development of a body temperature which may compromise function and safety (Tucker et al., 2004). Power output and integrated electromyographic activity in this study began to fall within the first 30% of a maximal self-paced cycle time trial in the heat in comparison to that conducted in a cooler environment, prior to a rise in core body temperature, heart rate or RPE. This anticipatory response may represent a fail safe mechanism in the human body (Kay & Marino, 2000) in an attempt to curtail dangerous rises in core body temperature. This assumption is supported by the work of Watson et al., (2005) who reported an increase in heart rate and lactate when dehydrated prior to a 50, 200 and 400 m race

performance which was attributed to an anticipatory stress and catecholamine response (Watson et al., 2005).

2.7. Hydration Status and Cognitive Function

Research regarding the effects of dehydration on cognitive function is somewhat less extensive and more ambiguous than that regarding its effect on physiological function. As indicated by Lieberman (2007) severe dehydration invariably leads to delirium and coma, and it is certain that many aspects of cognitive function will be affected long before this critical level of dehydration is reached. Serum sodium concentration is closely controlled by water homeostasis (Adroque & Madias, 2000). Hypernatremia, or elevated sodium levels, causing cell shrinkage and cellular dehydration (Offenstadt & Das, 2006) is known to manifest in central nervous system depression, decreased mental status, confusion, abnormal speech and stupor or coma in the more extreme cases (Achinger et al., 2006). Development of voluntary hypernatremia is unusual in that the body has strong reactive mechanisms, including the thirst response (Achinger et al., 2006). Hypernatremia has however previously been reported in a group of jockeys on a competitive race day (Warrington et al., 2009) and it is likely that such severe levels of dehydration may have been accompanied by certain cognitive impairments as previously described (Achinger et al., 2006).

Hypernatremia and its associated cognitive response is an extreme example of the effects of dehydration on cognitive function. Aspects of cognitive function do however appear to be impaired at much lower levels of dehydration (Ritz & Berrut, 2005). It has been suggested that impairments to cognitive function may occur at similar thresholds to physical impairments ($> 2\%$ body mass) (Gopinathan et al., 1988). The brain areas most vulnerable to the effects of dehydration are the reticular activating system, which controls attention and wakefulness, the autonomic structures which regulate psychomotor and regulatory function and the cortical and mid brain structures which are responsible for thought, memory and perception (Wilson & Morley, 2003). In addition it seems that complex cognitive processes are more consistently affected and that simpler aspects of cognitive function are more robust to the effects of dehydration. The review of Wilson and Morley (2003) described cognition as being of limited capacity and cognitive processes perceived as working in parallel to execute a complex

task are actually performing in competition with each other. Dehydration represents a stressor to the cognitive domain, so increasing competition among these parallel processes and compromising overall cognitive performance (Wilson & Morley, 2003)

Gopinathan et al., (1988) reported detriments to a number of measures of cognitive function (short term memory, arithmetic efficiency and visuomotor tracking) in response to dehydration. Cognitive function was compromised in a manner proportional to the level of dehydration, with detriments becoming significant at a body mass loss of 2% (Gopinathan et al., 1988). Perceptive discrimination, short term memory and an increase in subjective fatigue have been shown to be affected by dehydration of 2.8% in a study by Cian et al., (2001), regardless of whether dehydration was induced through heat exposure or treadmill exercise, with no appreciable difference demonstrated between the two methods of hypo-hydration (Cian et al., 2001). Tomporowski et al., (2007) reported that exercise induced dehydration to 3.7% body mass loss adversely affected executive functioning as evidenced by an increase in the number of errors made, but not short term memory, which was actually improved following exercise performance in both a dehydrated and euhydrated group. Suhr et al., (2004) demonstrated that hydration status was significantly related to performance on tests of psychomotor processing speed and attention/memory skills in a population of community dwelling older adults.

A major limitation of many of the studies involving dehydration and cognitive function is that the methods used to induce dehydration, namely heat and exercise (Cian et al., 2001; Gopinathan et al., 1988; Tomporowski et al., 2007) represent individual physiological stressors, making it difficult to isolate the effects of dehydration (Grandjean & Grandjean, 2007). Thermal stress alone has been indicated as having a detrimental impact on cognitive functioning (Hancock, 1986), while exercise may in fact improve certain aspects of cognitive performance (Tomporowski, 2003). Inclusion of a control group who are exposed to the same physiological stressor, but prevented from developing dehydration may aid in the isolation of dehydration induced impairments to cognitive function (Lieberman, 2007).

Many studies have attempted to control for this factor. Gopinathan et al., (1998) provided recovery time, between heat + exercise induced dehydration and cognitive

testing and this was not shown to attenuate the cognitive detriments demonstrated. In contrast to this however, confounding variables in the form of physiological stressors were absent in the study by Szinnai et al., (2005) who examined the effect of dehydration induced by 24 hours of water deprivation on event related potentials, cognitive motor function measures and subjective ratings of task compliance in a group of young healthy men and women. Dehydration of 2.6% body mass resulted in no effect on cognitive motor performance, although subjective measures of effort and concentration were significantly increased. Participants were able to overcome these subjective feelings however and maintained performance on the cognitive function aspects of this study (Szinnai et al., 2005). Results from this study were somewhat supported by the results of Cian et al., (2001), for whom cognitive function was normalised within 3.5 hours after fluid deficit, regardless of whether or not fluids were consumed.

Fluid restriction and hypo-hydration appear to have a more robust effect on subjective feelings related to alertness and fatigue (Lieberman, 2007; Shireffs et al., 2004; Szinnai et al., 2005), than on cognitive aspects of motor function (Szinnai et al., 2005; Tomporowski et al., 2007).

A number of potential mechanisms have been proposed which may explain the potential effects of dehydration on cognitive function. A review by Wilson and Morley, (2003) discussed many of these. Potential mechanisms included:

- ⇒ Hypercortisolemia: Impairs active learning, short term and verbal memory. Increased cortisol secretion is related to hydration status (Maresh et al., 2006) and has been shown to be predictive of cognitive performance (Lieberman et al., 2005).
- ⇒ Elevated cerebral arginine vasopressin: AVP is known as one of the primary water retaining hormones released during the process of dehydration (Antunes-Rodrigues et al., 2004). The AVP receptors are thought to have a role to play in both psychological and cognitive functions (Egashira et al., 2009) and as such an elevation in AVP concentrations may impact on psychological and cognitive parameters.

- ⇒ Enhanced nitric oxide release: Nitric oxide, whose release is elevated during dehydration, is a free radical which acts as a major signalling molecule in the nervous system and is thought to have a role to play in the homeostatic preservation of cognitive function.
- ⇒ Cell energy deprivation: A resultant loss in ATP dependent cellular activity may trigger inappropriate membrane depolarization and a critical accumulation of intracellular calcium, which may eventually lead to neuronal death.
- ⇒ Glutamate hypertransmission: Cellular dehydration may induce protein catabolism (Lang, 1998) causing an increase in the tissue liberation of the free amino acid glutamate. This may impact on cellular energetics, causing a reduction in adenyl cyclase activity and intracellular calcium mobilization.

In addition, it has been suggested that reduced plasma volume causing decreased blood flow through the brain may be involved in observed detriments to cognitive performance (Zuri et al., 2000). Brain cell volume has been shown to be unchanged in dehydrated rats however, and it appears that the majority of water lost may come from the skeletal muscle (Nose et al., 1991). Further research may be required so to more fully elucidate the effect of dehydration on cognitive function and associated mechanisms.

2.8. Hydration Recommendations for Athletes

A recent ACSM position stand regarding exercise and fluid replacement (Sawka et al., 2007) outlined guidelines for appropriate pre, during and post hydration strategies aimed at the prevention of development of performance impairing dehydration levels. Fluid provision during exercise may attenuate the physiological strain associated with dehydration (Armstrong et al., 1997; Maughan et al., 1996; Melin et al., 1997). During exercise, the goal of drinking should be to prevent excessive fluid loss of greater than 2% body mass. Examination of sweat rates may allow individualisation of hydration practices (Coyle, 2004) and intake of water during exercise has been shown to enhance endurance exercise performance (Maughan et al., 1996). Care should be taken however as fluid over-consumption during exercise may cause gastrointestinal discomfort (Daries et al., 2000), while replacement of total sweat losses with pure water can result in the development of dilutional hyponatremia (Speedy et al., 2000). It has been

suggested that if time is limited between performance bouts, 1.5 litres of water may be required to replace each kg of body mass loss. The excess fluid is required to compensate for increased urine production which accompanies consumption of large fluid volumes (Sawka et al., 2007).

The human thirst mechanism is incapable of maintaining water homeostasis as it is not triggered until a body water loss of 2% body mass (Kay and Marino, 2000) a level at which performance impairments are typically noted. This has been termed as voluntary dehydration and occurs even when unlimited fluids are available (Passe et al., 2007). It appears however that a certain amount of dehydration is tolerable to the human body, and that if allowed drink *ad libitum* human beings are capable of maintaining plasma osmolality within physiological limits, if not body mass (Armstrong et al., 1999; Maresh et al., 2004; Noakes, 2007). It appears therefore that in terms of rehydrating from a previously dehydrated state, the thirst mechanism, and other related hormonal responses may be sufficient to ensure regulation of plasma osmolality (Maresh et al., 2004). Melin et al., (1997) reported however that exercising in a dehydrated state may decrease renal concentrating ability and compromise kidney function (Mallie et al., 2001). Dehydration may blunt the action of the sympatho-adrenal system, so impairing the renal concentrating ability of the kidneys, although this situation is ameliorated through ingestion of water (Melin et al., 1997).

It is clear that dehydration may have a negative effect on both physiological and cognitive function and may impair the ability of the body to protect against serious illnesses such as heat exhaustion and stroke (Coris et al., 2004). Dehydration, particularly on competitive race days, appears to be prevalent within jockeys (Warrington et al., 2009) however and the use of deliberate dehydrating mechanisms, such as saunas, appears commonplace (Dolan et al., 2010). Maughan et al., (2004) have suggested that although athletes may learn to cope with the physiological discomforts associated with dehydration, there is no evidence supporting a physiological adaptation to this stressor (Maughan & Shireffs, 2004). It is likely therefore that dehydration used to aid body mass loss for competition may affect performance in this group, as well as representing a significant health and safety concern to the horse-racing industry.

Summary

Adequate nutritional and fluid intake is essential for maintenance of usual homeostatic function and optimum athletic performance. It is clear that dehydration and energy deficiency may impact on health and performance in weight category athletes. Many of the detriments to health and performance discussed here are likely to occur as a result of disrupted endocrine function, the importance of which will be discussed in the following section.

3. Endocrine Response to Weight Cycling:

The primary function of the endocrine system is to co-ordinate function between all body systems, so enabling maintenance of homeostasis and aiding in the response to external stimuli. Most hormones are released in an intermittent pulsatile fashion in quantities sufficient to meet anticipated needs. An acute reduction of body mass for competition and chronic weight cycling represents both an acute and chronic physiological stressor (Brownell et al., 1987; Burge et al., 1993; Fogelholm, 1994) and as such are likely to elicit an adaptive endocrine response (Strauss et al., 1985). A wide variety of metabolic and energy regulating hormones are likely to be affected through chronic weight restriction and weight cycling. The glucocorticoids, androgens, and growth hormone/IGF-1 are some of the primary hormones which may be affected and are to be focused on in this review of literature and research project.

3.1. The Glucocorticoids/Cortisol

The glucocorticoids, of which cortisol is considered the most biologically relevant, are a group of corticosteroid hormones produced by the adrenal cortex and are involved in the response to physical, psychological and physiological stress (Borer, 2003; Hill et al., 2008; Levine et al., 2007). Cortisol is a low molecular weight lipophilic molecule which penetrates tissue cells through a process of passive diffusion. It is released in response to a cascade of hormonally mediated reactions, beginning with the release of corticotrophin releasing hormone (CRH) from the hypothalamus and culminating in the release of cortisol from the cortex of the adrenal gland (Levine et al., 2007).

Control between these hormones is achieved through a classic negative feedback mechanism. Cortisol has a biological half life of approximately 80 minutes and release undergoes diurnal variation with highest levels present in the early morning. Secretion also undergoes 7 – 15 spontaneous increases throughout the day, which are generally associated with food intake.

The primary function of cortisol is to restore homeostasis following stress, including the physiological stress caused by fasting and malnourishment (Bergendahl et al., 1996). This is achieved primarily through increased gluconeogenesis and the breakdown of adipose lipids and lean mass proteins so increasing circulating glucose and free fatty acid (FFA) concentrations. This action provides energy and substrate to the cells for use in homeostatic restoration (Djurhuus et al., 2004; Levine et al., 2007). Cortisol is also involved in mood alteration and the encouragement of *ad libitum* food intake (Borer, 2003).

The catabolic action of cortisol in times of stress has many negative physiological consequences, including muscle wasting (Fitts et al., 2007), thinning of the skin (Borer, 2003) and a loss of cartilage and bone matrix (Abad et al., 2001; Canalis, 1996). Cushings syndrome is an endocrine disorder characterized by high circulating levels of cortisol. Common symptoms include an increase in centrally distributed fat, muscle and bone thinning, and a number of psychological disorders. Body composition effects appear similar between those with adult or childhood onset of this condition (Di Somma et al., 2002) and it has been suggested that adverse body composition effects may continue long after successful treatment of this condition (Abad et al., 2001).

Given that cortisol is the primary catabolic hormone in the body homeostasis can occur only if a balance is achieved between its action and that of its anabolic counterparts, including the androgens and growth hormone and associated growth factors such as the IGF's (Kraemer, 2000) the specific role of which will be discussed later in this section.

3.2. The Androgens/Testosterone

The androgens refer to a group of sex hormones and are considered to be one of the primary anabolic agents in the body. Androgens are C-19 steroids which are secreted

primarily from the testes in men and the adrenals in both men and women, although they may also be locally synthesized in the peripheral tissues. There are a number of androgens, including dehydroepiandrosterone (DHEA), DHEA-sulphate and androstenedione, however testosterone is currently considered to be the most biologically relevant (Venken et al., 2008). The androgens, and testosterone in particular, are involved in the promotion of protein synthesis in tissues which have androgen receptors although testosterone is also capable of exerting an effect through the estrogen receptors α and β following aromatization to 17 β estradiol by the P430 aromatase enzyme. Testosterone's anabolic effects include growth of muscle mass and strength, which occur in a linear dose response fashion (Woodhouse et al., 2003) and through the process of hypertrophy (Sinha Hakim et al., 2002), and increased bone density and strength (Romeo & Ybarra, 2007; Sanyal et al., 2008; Venken et al., 2008). Testosterone also appears to have a role to play in the regulation of sexual (Isidori et al., 2005) and cognitive (Beachet et al., 2006) function.

Forty-four – sixty-five % of testosterone in circulation is bound to the protein sex hormone binding globulin (SHBG). This is a plasma glycoprotein, the gene of which is located on chromosome 17p13.1 and whose primary function is to bind certain androgens and estrogens thereby disabling and regulating their action. In addition SHBG is capable of binding to a specific receptor, inducing a signalling cascade mediated through a G protein (Nakhla et al., 1999) which culminates in an increase in cytoplasmic cyclic AMP so inducing lipolysis. The binding of steroids to SHBG interferes with the binding of this molecule to its receptor (Kahn et al., 2002) and higher concentrations of SHBG are associated with a decrease in free testosterone availability. SHBG levels are known to increase with age, and have been suggested as being involved in the muscle and bone loss associated with aging (Khosla et al., 1998). Decreases in total and bioavailable testosterone and an increase in SHBG are mediated through increases in the pituitary gonadotropins follicle stimulating hormone (FSH) and lutenising hormone (LH) (Feldman et al., 2002), which act primarily as regulatory agents in a number of anabolic and reproductive processes. In addition, there appears to be an inverse relationship between SHBG and IGF-1, IGF-11 and IGFBP3 (Pfeilschifter et al., 1996). These growth factors have a pleiotropic anabolic effect, which will be discussed in greater detail later in this section.

Long term caloric restriction without malnutrition has been shown to reduce serum total testosterone and to increase SHBG levels (Cangemi et al., 2010). In addition, testosterone has been shown to be reduced in a group of wrestlers during the competitive season (Strauss et al., 1985) and in male athletes undergoing high levels of endurance training (Hackney et al., 1988). Reduced circulating sex hormone concentrations during times of energy imbalance have been suggested as occurring in an attempt by the body to contain anabolic processes as the synthesis of large complex molecules from smaller simple ones requires relatively large amounts of energy (Wade et al., 1996).

3.3. Growth hormone, IGF-1 and the Somatomedin Hypothesis

Growth hormone is a pleiotropic anabolic hormone which acts on many of the tissues of the body to induce a positive nitrogen balance and encourage protein synthesis. Its actions are in many ways mediated and regulated by that of the growth factor insulin-like growth factor-1 (IGF-1), although the relationship between the two is quite complex, as will be discussed in the forthcoming section. The mechanisms by which these hormones act is called the “somatomedin hypothesis”. The somatotrophic axis refers to the hypothalamus, the pituitary gland and the liver, with the hypothalamus acting as the control centre which mediates the actions of the other two. The original somatomedin hypothesis suggests that growth hormone, which is secreted from the pituitary gland cannot work directly on its target tissue but instead stimulates the release of hepatic IGF-1 which travels through the circulation to exert an effect on the target tissues (Le Roith et al., 2001).

While elements of this hypothesis have stood up over the years a number of studies display inconsistencies in its design. For example, IGF-1 is expressed in most tissues of the body and may work in an autocrine/paracrine as well as endocrine fashion (Eicher et al., 1993). In addition, while it is clear that the actions of GH and IGF-1 are interlinked in many ways, it also seems that both these elements have independent actions. It seems that growth hormone’s primary anabolic function is to promote protein synthesis while IGF-1 acts both to reduce protein degradation and to increase protein synthesis (Le Roith et al., 2001).

3.3.1. Growth Hormone

Growth Hormone is a cytokine peptide hormone containing 91 amino acids (Clark, 1997), which are synthesized and stored primarily in the anterior pituitary gland. Growth hormone releasing hormone (GHRH), which is released from the hypothalamus, causes the release of GH from the pituitary gland and it is regulated by a classic negative feedback system involving itself, its mediator IGF-1 and somatostatin or the “growth hormone inhibiting hormone” (GHIH) (Smith, 2005). GH may also be synthesized in a number of extra-pituitary sites suggesting a local paracrine/autocrine action although this effect appears to be small in comparison to its pituitary derived IGF-1 mediated endocrine action (Clark, 1997).

The primary function of GH is to stimulate cell growth and reproduction and to induce lipolysis through cAMP mediated events. This function appears to occur at the transcriptional level (Guk-Chor Yip & Goodman, 1999). Growth hormone’s lipolytic effect is thought to occur through both direct and indirect mechanisms and to result in an increased concentration of circulating free fatty acids so sparing glycogen stores (Gibney et al., 2007). GH secretion in humans and rodents is highly pulsatile in nature, the actual pattern of which is gender specific. GH’s secretory pattern appears to have a significant impact on its metabolic action, with irregular “male” pattern pulsatility conveying a more potent anabolic effect (Jaffe et al., 2002) with a higher biological activity for the generation of hepatic IGF-1 than the more regular “female” pattern of release (Giannoulis et al., 2005).

As with all hormones and endocrine agents, the secretion and action of GH does not occur alone, but in combination with a number of other hormones, which act together in a complimentary and counter-regulatory fashion in order to ensure homeostasis. Leptin appears to have a direct role to play in somatotrope function as indicated by the presence of the Ob receptor on the pituitary. Evidence that GH secretory burst interval and frequency are strongly and independently predicted by leptin concentration (Misra et al., 2005) suggests that growth hormone’s effect may be dependent on the extent of available energy within the body. Abnormalities of the GH-IGF-1 system have been reported in humans in chronic energy imbalance, and low IGF-1 with high GH in adults suffering from anorexia nervosa has been suggested as occurring as a result of a

nutritionally acquired GH resistance (Counts et al., 1992; Misra et al., 2005; Stoving et al., 1999).

GH and testosterone have similar effects in that they are both potent anabolic hormones and GH's pulsatile secretory pattern appears to be at least in part mediated through the action of testosterone (Painson et al., 2000). Testosterone has the potential to stimulate GH release (Weissberger & Ho, 1993) although these hormones have both independent and additive effects, suggesting actions via distinct pathways (Gibney et al., 2005). The catabolic action of the glucocorticoids appears to cause a suppression of GH secretion and IGF-1 activity (Rajaram et al., 1997). Cortisol and GH have opposite effects on protein metabolism (Fitts et al., 2007; Moller & Norrelund, 2003) but similar, though additive lipolytic actions (Djurhuus et al., 2004).

3.3.2. Insulin-like Growth Factor-1

The IGFs, or insulin-like growth factors are a group of pleiotropic growth factors, whose effects are in many ways mediated through the action of growth hormone (Woelfle et al., 2003). They were named “insulin-like” because of their similarity to insulin in that they too have the ability to stimulate glucose uptake into the liver. The IGF group is composed of the ligands IGF-1 and 2 and insulin. IGF-1 and 2 are peptides of 70 and 67 amino acids respectively. The IGF system is sensitive to metabolic alterations (Haspolat et al., 2007; Kari et al., 1999; Thissen et al., 1994) and their abundance, along with receptor sensitivity and synthesis is controlled in order to reflect tissue requirements. These growth factors appear to have a key role to play in the processes that link nutrition and growth (Frystyck et al., 1999).

The complexity of the IGF system is demonstrated by the presence of six individual and specific binding proteins, which have a number of IGF dependent and independent roles to fulfil (Rajaram et al., 1997) and are capable of modulating IGF induced cell proliferation in both a positive and negative fashion depending on homeostatic requirements. Delayed growth during short term acute caloric restriction appears to be mediated through a reduction in IGFBP1 (Frystyck et al., 1999) whose main metabolic role is to bind and inactivate unbound IGF, so acting as an *in vivo* inhibitor of IGF-1 action (Rajaram et al., 1997).

Longer term chronic energy restriction, as is evident in those suffering from anorexia nervosa induces a decrease in IGFBP3 (Counts et al., 1992) which is recognised as providing the main reservoir of accessible IGF-1 to the body (Rajaram et al., 1997). It has been suggested that the regulation of hepatic IGF-1 in relation to energy availability is achieved primarily at the post transcriptional level (Frystyck et al., 1997) and that this is influenced by the actions of many hormones, including insulin, growth hormone, the sex steroids, the glucocorticoids and the thyroid hormones (Rajaram et al., 1997). It is thought that transient adaptations of the GH/IGF-1 system in response to nutritional and energy deprivation reflect the need to conserve resources for the maintenance of more basic and essential body functions (Counts et al., 1992).

Summary

A number of endocrine factors related to reproduction and metabolism appear to be affected by the extent of energy availability within the body. It is possible that endocrine function in jockeys may be affected through chronic weight cycling. It has been suggested that jockeys may have low bone mass (Warrington et al., 2009b). This finding may have particular consequences for this group given the high risk nature of the sport (Hitchens et al., 2009; Waller et al., 2000). Endocrine disturbances, which may occur as a result of chronic weight cycling and acute body mass reduction may impact on osteogenic function in this group. This theory, and the regulation of bone mass and integrity will be discussed in the next section.

4. Bone

The skeletal system comprises of cartilage, ligaments and bone/osseous tissue which join together to provide a strong flexible framework for the body, which enables movement through the provision of rigid levers. Bone also has a protective role to fulfil within the body as it encloses a number of organs, such as the brain, lungs, spinal cord, heart, pelvic viscera and bone marrow. The strength and resilience of bone allows fulfilment of this function, forming a protective “cage” around the more vulnerable organs. In addition to its structural and protective role, bone also has a wide ranging and vital metabolic role to fulfil including:

Blood Formation

The majority of blood and immune system cells are formed in the bone marrow. This is a soft hemopoietic tissue that occupies a bone’s medullary cavity and consists of a mesh of reticular tissue saturated with immature blood cells and adipocytes (Saladin et al., 1999b)

Electrolyte Balance

The majority of total body calcium and phosphate is stored within the bone tissue, which provides a reservoir of minerals for the human body (Peacock, 2010).

Acid-base balance

Bone acts as a physiological buffer which protects against pH change through the absorption and release of alkaline salts (Sebastian et al., 1994).

Detoxification

Bone has the ability to remove and store certain heavy metals and foreign substances from the body which may later be slowly released into the bloodstream for safe excretion (Saladin, 1999b).

4.1. The Anatomy of Bone

Osseous tissue is essentially a connective tissue in which the matrix has been hardened by the deposition of calcium, phosphate and other minerals. It is composed of

approximately 1/3 organic and 2/3's inorganic matter. The organic component is composed largely of collagen and various protein/carbohydrate complexes such as glycosaminoglycans, proteoglycans and glycoproteins. Hydroxyapatite is the principal component of the inorganic compartment. This is a crystallized calcium-phosphate salt, which also contains lesser amounts of other elements, such as magnesium, sodium, potassium, fluoride, sulphate, carbonate and hydroxyl ions (Saladin, 1999b). The combination of organic and inorganic matter, or collagen and mineral provides the bone with a combination of strength and resilience as minerals resist compressive forces, while collagen resists tension. Bone tissue also contains blood vessels, which penetrate bone through minute holes called nutrient foramina; bone marrow and cartilage, adipose, nervous and fibrous tissue.

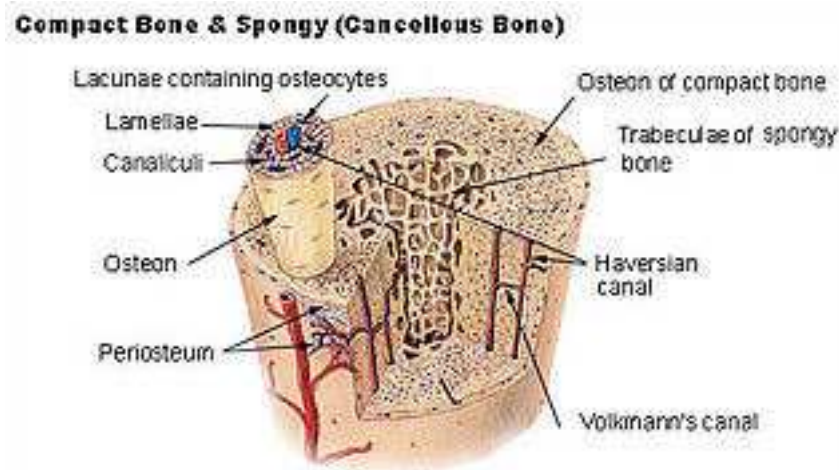


Figure 2.2: The Microscopic Structure of Bone

There are two major types of bone; cortical and trabecular bone. Approximately 90% of the skeleton is composed of cortical bone, the main function of which is to provide mechanical support. The basic structural units of cortical bone are called osteons, which contain layers of matrix concentrically arranged around a central canal, known as the Haversian canal. Osteons are connected to each other and to the nutrient foramina by canaliculi known as Volkmans canals which allow communication and transportation throughout the bone. Trabecular bone contains a mesh of slender rods called trabeculae, the spaces between which are filled with bone marrow. Osteons are unnecessary in trabecular bone as the latticed structure ensures that all blood vessels are in close proximity with the osteocytes, which represent bones specialised

communication and transportation cells. At first glance it appears that the trabeculae are arranged in a random order. In fact, they develop along the bone's line of stress, so imparting maximum strength while adding minimum weight (see figure 2.3). Trabecular bone is always enclosed by a layer of cortical bone. The microscopic structure of bone tissue is illustrated in figure 2.2.

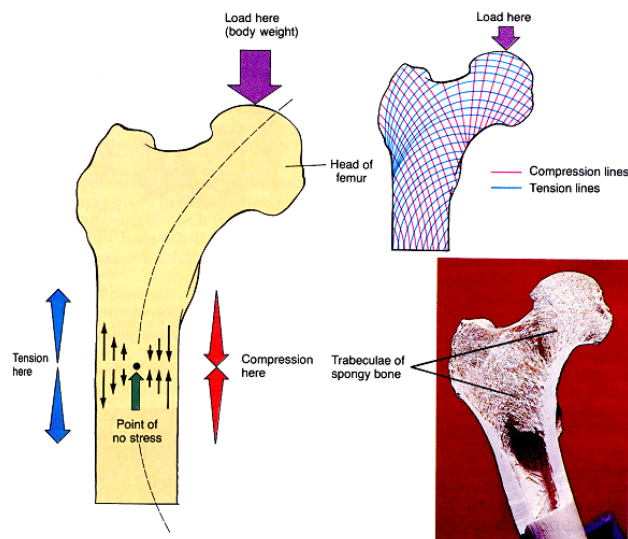


Figure 2.3: Trabecular Bone

4.2. The Physiology of Bone

Bone's metabolic function, along with processes concerned with maintenance, growth and remodelling ensure that bone remains a highly metabolically active tissue throughout its lifetime. Bone contains four types of specialised cells which enable it to fulfil its metabolic function (Saladin, 1999b; Khan et al., 2001). These are:

Osteogenic Cells

Cells formed from embryonic fibroblasts which have the ability to differentiate into osteoblast cells. They occur in the endosteum, the inner layer of the periosteum and within the haversian canals.

Osteoblasts

These are the principle mediators of bone formation. Osteoblasts aid in the synthesis of organic matter and bone mineralization. When activated these cells produce collagen

fibers which wind around the osteon in a helical pattern. Crystallization of collagen occurs through a positive feedback process whereby the fiber becomes encrusted with calcium phosphate. Osteoblastic cells are non-mitotic, however numbers increase rapidly in response to stress due to osteogenic cell differentiation.

Osteoclasts

These are the principle bone resorption cells and are located primarily on the bone surface. Osteoclast cells attach to the bone surface via a ruffled border which acts to form a subosteoclastic bone resorbing compartment. When activated these cells secrete hydrogen ions into the extracellular space via hydrogen pumps. Chloride ions follow by a process of electrical attraction so filling the space between the osteoclast and bone with a strong hydrochloric acid which dissolves bone minerals. Osteoclasts also secrete the enzyme acid phosphatase/cathepsin which induces a breakdown of collagen fibers.

Osteocytes

These cells are former osteoblasts which have become embedded in the bone matrix. Osteocytes are connected via gap junctions which allow for transportation of nutrients and waste throughout the bone itself and to and from the circulatory system. These cells also allow communication between the osteoblasts and osteoclasts regarding maintenance of bone integrity. Osteocytes reside in cavities within the bone known as lacunae which are connected to each other by slender channels called canaliculae.

4.2.1. Bone Modelling and Remodelling

Bone can grow in one of two ways: 1) interstitial growth, which refers to the adding of internal matrix and 2) appositional growth, which is the development of additional bone on its outer surface.

Modeling refers to the process whereby new bone is synthesized in order to maximise stiffness and minimise deformation. It is the dominant process in the growing skeleton. Remodeling however is the dominant process which occurs in the mature skeleton (Brandi, 2009). It is an organized bone cell activity based on the basic multicellular unit (BMU) whereby bone may replace itself so allowing growth and repair. During this process osteoblast and clast activity is triggered by microdamage caused by

repeated bone strains (Martin & Seeman, 2008). In response to this, osteoclasts cause breakdown or resorption of the damaged area. This process triggers a responsive stimulation of osteoblastic activity enabling the production of replacement bone (Khan et al., 2001). The osteocyte and canaliculae system provides a means of communication between bone cells allowing this coordinated process occur. Mature bone is capable of appositional growth only, as there is insufficient space for interstitial growth. Macromodelling refers to the adding of regional bone mass, so improving geometric strength and properties. Micromodelling refers to change in trabecular orientation to best withstand the direction of strain (Khan et al., 2001) (see figure 2.3). The exact molecular signalling mechanisms by which bone integrity is mediated remain unclear (Rubin et al., 2006) and represents an area of bone research which warrants further investigation.

4.2.2. Biochemical markers of bone turnover

Bone remodelling is a dynamic process. Measurement of specific markers of bone turnover allows analysis of the state of the bone modelling process. Examination of biochemical markers of bone turnover is said to be useful in the prediction of the rate of bone loss (Garnero et al., 1999; Proteau et al., 2006) and fracture risk (Garnero et al., 2000; Romeo & Ybarra, 2007), although biochemical markers of bone turnover appear to be limited in their ability to predict individual bone mass variability (Marcus et al., 1999). Results from these and other studies suggest that examination of such markers may be useful as a complement to the information provided by bone mass assessment but should not be considered as a surrogate marker (Dogan & Pasaci, 2002).

The main formation markers currently utilised include bone specific alkaline phosphatase and osteocalcin which are secreted by the osteoblasts and the extension peptides of procollagen type 1, which have both amino and carboxy terminals (P1NP and P1CP). Pyridinoline, deoxypyridinoline and collagen degradation products such as amino and carboxy telopeptides of collagen cross links (NTX and CTX) represent the main measurable bone resorption markers (Dogan & Pasaci, 2002). These are secreted into the circulation and renally cleared and may therefore be measured in either the serum or the urine (Khan et al., 2007).

Assessment of biochemical markers of bone turnover may be particularly useful when considering intervention efficacy as they provide a more immediate indication of bone remodelling changes than BMD alone (Camozzi et al., 2007; Pagani et al., 2005). In addition bone turnover markers represent a more systemic representation of bone response than does a single BMD measurement (Chailurkit et al., 2001). While it is not currently possible to identify any one marker which has the sensitivity and specificity to accurately diagnose osteoporosis, biochemical markers of bone turnover have a valuable contribution to make to the overall diagnosis of bone health, particularly when examined within the context of other risk factors (Dogan & Pasaci, 2002; Garnero et al., 2000; Garnero et al., 1999).

4.3. Influences on bone health

Optimal bone strength and functionality are highly individual and dependant on a number of determining factors. Bone must be strong enough to withstand the stresses of daily living without succumbing to fracture. Bone mass and architecture in excess of that dictated by the habitual strain experienced may hinder fluid movement however and be inefficient in terms of the energy requirements of growth, maintenance and use (Bass et al., 2005; Calbet et al., 1999). While much of bone mass and strength is genetically predetermined there are a number of modifiable dietary and lifestyle factors which have a role to play in the development of bone strength above its genetically predetermined baseline level. These factors will be discussed in the following sections.

4.3.1. Genetics

It has been suggested that baseline levels of bone strength, size and quality in humans are determined *in utero*, and that these baseline qualities remain unchanged throughout the organism's postnatal life (Bass et al., 2005). The genetic influence on bone regulation is summarised in Table 2.1. Bone fracture, which is the clinical outcome of insufficient bone strength, appears to display the lowest heritability (Ferrari, 2008) and it is thought that environmental factors such as occurrence, mechanism and response to falls may be the over-riding factor to consider here (Kannus et al., 1999).

Table 2.1: Heritability (h^2) of osteoporosis-related phenotypes

Phenotype	Heritability (%)
Bone Mineral Density	50 – 80%
Hip Geometry	70 – 85%
Bone Turnover (Biochemical Markers)	40 – 70%
Bone Micro-Architecture	50 – 60%
Fracture	25 – 48%

Source: Ferrari, 2008

Skeletal development is extremely complex, with numerous genetic and environmental influences (Cusack & Cashman, 2003; Ralston, 2007). A number of gene polymorphisms and quantitative trait loci have been identified as having a contribution to make toward skeletal health and fracture susceptibility. These include those associated with the vitamin D receptor, collagen type 1 α 1, oestrogen receptor α , transforming growth factor β 1, lipoprotein receptor related protein 5, sclerostin, TCIRG1 and CLCN7 (Cusack & Cashman, 2003; Ralston, 2007). Individual polymorphisms however appear to have minimal effects on bone's adaptive response (Judex et al., 2004) and actual fracture risk (Ferrari, 2008), demonstrating the complexity of this system.

Individual genetic make-up exerts an influence on bone structure through a number of pathways, including: a direct effect on bone size and geometry; indirect effects on bone mass, muscle strength and activity and a modification of osteogenic cell sensitivity to mechanical loading (Karasik & Kiel, 2008). Muscle and bone are considered to be two parts of the same functional units, and stem from a common precursor, namely the mesenchymal stem cell. Muscle mass and function determine the extent of mechanical loading to which a bone is exposed (Frost, 2003; Roblin, 2009) and have a role to play in the prevention of falls and attenuation of forces due to its dynamic stabilizing capacity. Sarcopenia appears to coexist with osteopenia (Solomon & Bouloux, 2006) and it appears that muscle and bone share common genetic determinants which allow maintenance of appropriate proportions of muscle and bone relative to physical demands (Karasik & Kiel, 2008). The numerous influences

involved in bone regulation add to the complexity of elucidation of a specific genotype associated with bone regulation and fracture risk.

4.3.2. Body Composition

Absolute body mass has been shown to be a strong positive predictor of bone mineral density (Gerdher et al., 2003; Michaelsson et al., 1996; Semanick et al., 2005) and has been suggested as a suitable selection factor for bone mass screening (Michaelsson et al., 1996). Absolute body mass consists of three compartments, namely fat, lean and bone tissue. Both the fat and lean compartments have a contribution to make toward the regulation of bone mass and the relationship between these variables will be discussed in the following section.

4.3.2.1. Lean Mass

It seems that lean mass is the more relevant measure of body composition to consider when assessing the impact of body mass on bone (Arimatsu et al., 2009; Burr, 1997; Semanick et al., 2005; Wang et al., 2005) as its mass and action provides the mechanical loading by which bone is regulated (Bass et al., 2005; Frost, 2003). The effect of lean mass and physical activity on bone mass and architecture will be discussed in greater detail in section 4.3.3.

4.3.2.2. Fat Mass

The role of fat mass in the development of bone mass and architecture is somewhat more equivocal and research has yielded some conflicting results. A large body mass appears to convey a protective effect on bone health (Gerdhem et al., 2003; Gomez-Ambrosi et al., 2008; Michaelsson et al., 1996; Semanick et al., 2005), likely due to the increased mechanical loading which it places on the skeleton, the effect of which will be discussed in section 4.3.3. In addition, release of a number of adipose tissue factors or adipokines have been suggested as having a role to play in maintenance of bone health (Gomez-Ambrosi et al., 2008), while factors secreted from the skeleton, termed “osteokines” may also be involved in adipose tissue regulation. The main adipokines cited as being involved in bone tissue regulation are leptin, adiponectin, resistin, visfatin, IL-6 and TNF α . The osteokines which may have an effect on adipose tissue are osteopontin, osteocalcin, osteonectin and osteoprotegerin/RANKL. The

relationship between the bone and adipose organs has been termed the bone adipose axis (Gomez-Ambrosi et al., 2008).

Converse to evidence indicating a positive impact of fat mass on bone is the suggestion that there may be a degree of pathophysiological linkage between disorders of the bone and adipose tissue, namely osteoporosis and obesity (Zhao et al., 2007a). These disorders appear to share common genetic and environmental influences with suboptimal nutritional and physical activity habits known to exert an influence on both. Furthermore, both adipocytes and osteoblasts derive from a common progenitor (the mesenchymal stem cell) and it has been suggested that differentiation may be directed toward one over the other (Zhao et al., 2007b).

In support of this contention are data from studies which report that once the contribution of body mass to increased mechanical loading has been accounted for, fat mass *per se* shows a negative relationship with bone mass (Zhao et al., 2007a). Examination of bone mass and body composition in a large cohort of Chinese men and women (n = 13,970) showed a significantly higher odds ratio for development of osteoporosis and osteopenia, and of suffering a non-spinal fracture in those participants with a higher % body fat, after adjusting for the potential confounding influence of body mass, age and physical activity. In addition placement of subjects in five kg strata demonstrated a negative correlation between fat and bone mass at any given body mass range (Hsu et al., 2006). Results from this study support the hypothesis that fat mass itself has a negative impact on bone once the contribution of mechanical loading is accounted for (Hsu et al., 2006; Zhao et al., 2007a).

Further complicating the relationship between bone and fat mass however is evidence that both age and physical activity habits may dissociate the relationship between the two (Reid et al., 1995; Wearing et al., 2006). Reid et al., (1995) identified fat mass as one of the strongest predictors of bone mass in a group of sedentary premenopausal women, but not in a group of premenopausal women who exercised regularly. A review by Wearing et al., (2006) reported that fat mass is negatively correlated with fracture risk in adults and it was suggested that this may occur as a result of increased BMD (Gerdhem et al., 2003; Michaelsson et al., 1996; Semanick et al., 2005) and the greater “cushioning” effect of increased adipose tissue (Wearing et al., 2006) although

an adipose induced reduction in peak force alone may be incapable of preventing fracture (Robinovitch et al., 1995). In contrast, it has been reported that adiposity is associated with a two-fold greater risk of wrist fractures in children (Goulding et al., 2000a). This was attributed to lifestyle factors contributing to high adiposity in children, such as low physical activity levels and a resultant lack of balance and coordination (Wearing et al., 2006). Controversy exists as to whether or not fat mass has a positive impact on bone mass in obese children. A study examining obese (n=103) and non-obese (n=132) children showed that obesity was related to significantly higher whole body bone area and BMC adjusted for height and vertebral bone density (Leonard et al., 2004b). This finding was in contrast to that of Goulding et al., (2000b) who reported significantly lower vertebral BMC for bone area after adjustment for height, weight and Tanner staging. Differences in methods of bone mass adjustment may account for the differing results reported (Goulding et al., 2000b; Leonard et al., 2004b).

4.3.3. Bone and its Physical Environment

Physical activity has long been cited as one of the key determinants of bone strength and quality (Andreoli et al., 2001; Morel et al., 2001; Nordstrom et al., 1998; Schoenau, 2006) and bone is said to adapt and develop in order to cope, without undue fatigue or fracture, to the habitual loads which are placed on it (Bass et al., 2005). Although baseline bone variables are genetically predetermined (Cusack & Cashman, 2003; Ferrari, 2008) the extent of development of bone strength, mass and geometry is ultimately determined by the physical environment to which it is exposed, provided all nutritional and endocrine influences are in place (Bass et al., 2005). The mechanostat theory is widely proposed as a potential mechanism whereby the skeletal system adjusts and adapts in accordance with its physical environment so enabling it to cope with the typical peak voluntary muscle loads (TPMVLs) placed on it.

4.3.3.4. The Response of Bone to Mechanical Loading

Bone appears to have two mechanical set points, or thresholds: one which “switches on” the mechanostat and the other which “switches it off” (Frost, 2004). A minimum effective strain (MES) (the “on” switch) must be applied to bone in order to elicit an

adaptive response, while lower levels of physical activity and mechanical stimulation (the “off” switch) result in the suspension of this process (Schoenau, 2005). It appears that there is a site specific response of bone to the mechanical loads placed on it (Skerry, 2006), although physical activity may also convey a more generalized component to bone adaptation, likely due to the action of the synergistic muscles (Nevill et al., 2003).

Mechanical bone competence is defined as the “ability of bone to cope with the stresses which are habitually placed on it” (Frost, 2003). It is widely acknowledged that both muscular (Robling, 2009) and gravitational (Judex & Carlson, 2009) forces represent the primary sources of mechanical loading on bone. Muscle forces, caused by intentional skeletal contractions place a strain on the bone to which they are attached (Frost, 2003), while gravitational forces, generated in response to impact with an external object may increase the extent of bone strain provided by muscular contractions (Andreoli et al., 2001). Controversy exists however as to whether muscular or gravitational forces predominate in the response of bone to its mechanical environment (Kohrt et al., 2009).

The strain provided by mechanical loading is sensed by the osteocyte cells, which convey this information to the specialised bone remodelling unit, namely the osteoblast and osteoclast cells, which were described in section 4.2. It is thought that microscopic matrix deformations in response to the strain administered by mechanical loading may induce fluid flow through the matrix, in response to the creation of pressure gradients within the interstitial and canalicular spaces (Khan et al., 2001). It appears that it is fluid shear stress and flow and not matrix deformation *per se* which actually cause stimulation of the osteocytes and initiation of the bone remodelling process (Judex et al., 2006; Qin et al., 2003) and the extent of bone remodelling reflects the extent of fluid displacement throughout the bone matrix.

4.3.3.5. Sports Participation and Bone Regulation

The adaptive response of the skeleton to the loading to which it is habitually exposed may best be demonstrated through removal of this stimulus. Mechanical unloading as incurred through weightlessness (Vico et al., 2000) or immobilization (Rubin &

Lanyon, 1987; Zerwekh, 1998), invariably culminates in skeletal demineralization. Participation in sport on the other hand represents an ideal model through which to examine the effects of different types of physical activities on bone mass and regulation. Weight bearing activities which provide peak impact forces on the bone appear to provide the greatest osteogenic stimulus (Calbet et al., 1999; Morel et al., 2001; Nichols et al., 2003; Wittich et al., 1998). For example, athletes participating in high intensity high impact activities such as rugby (Elloumi et al., 2009), soccer (Wittich et al., 1998), volleyball (Calbet et al., 1999), gymnastics (Pollock et al., 2006) and combat sports (Andreoli et al., 2001) all display enhanced bone mass in comparison to those involved in lower impact activities such as road cycling (Nichols et al., 2003; Warner et al., 2002) and swimming (Taaffe et al., 1995). In particular it has been said that those activities which provide peak forces in unusual directions may represent the most potent osteogenic stimulus to the body (Morel et al., 2001).

Although the bone related benefits of sports participation appear certain, research examining those athletes involved in the sport of running display some paradoxical findings. Running has been associated with both higher (Brahm et al., 1997; Rector et al., 2008) and lower (Zanker & Cooke, 2004) bone mass than either non-athletic controls or athletes from different sports including cyclists (Rector et al., 2008). It has been suggested that above a certain threshold of training volume, physical activity may in fact represent a threat to skeletal integrity (Maimon et al., 2004). Research indicates that disturbances to bone regulation identified in those involved in high volumes of training may not occur as a result of exercise *per se* however, but depend on whether or not the energy expended during training and competition allows a state of energy balance be maintained (Zanker & Swaine, 2000). The effect of insufficient energy intake on bone health will be discussed in greater detail in section 4.3.4. Briefly however, energy availability refers to the extent of energy available to the body to perform all other functions once the energy expended in physical activity is accounted for (ADA, 2009). Endocrine (Loucks & Thuma, 2003) and osteogenic (Ihle & Loucks, 2004) function appear to be disrupted at a threshold of energy availability of 30 kcal·kgLBM·day⁻¹ (Ihle & Loucks, 2004; Loucks, 2003; Loucks & Thuma, 2003) and it is possible that high training volumes, culminating in a high degree of energy expenditure may represent a threat to the skeletal system if this threshold is not met through sufficient dietary intake.

The bone health of weight category athletes has been suggested as being placed at risk as a result of the severe energy restrictions and physiological stress which accompanies an acute reduction in body mass (Proteu et al., 2006; Walberg Rankin, 2006; Warrington et al., 2009). The high impact loading and mechanical strain induced by certain sports such as gymnastics may however convey an osteogenic stimulus which may potentially counteract and over-ride the negative influence of energy deficiency (Zanker et al., 2004), although this will of course depend on the extent of energy availability. A rapid reduction in body mass in preparation for competition has been shown to compromise osteogenic function in a group of elite judoists, as evidenced by a shift in bone turnover toward a primarily resorptive state (Proteau et al., 2006). Weight regain between competitions, coupled with the high impact nature of judo seemed to be reflected by an overall osteogenic balance favouring bone formation however, while bone mass was shown to be enhanced in comparison to a group of controls. Based on these findings it was concluded that high impact exercise may convey a protective effect on bone mass, despite the negative osteogenic effects of rapidly reducing body mass (Proteau et al., 2006). The hypothesis that participation in a high impact sport may convey a protective effect on bone health in weight category athletes is supported by a study examining a group of female boxers who were reported to display high levels of bone mineral density in comparison to a control group despite having low levels of body fat, high energy expenditure and a high incidence of oligomenorrhea (Trutchnigg et al., 2008).

4.3.4. Nutritional Influences on Bone

Nutrition is a key modifiable lifestyle factor involved in bone regulation and is capable of exerting a systemic influence which impacts on the skeleton as a whole. Nutrition exerts a dual effect on bone, both through the provision of substrate and the preservation of usual endocrine and metabolic function. A number of aspects of nutritional intake are suggested as being fundamental to maintenance of skeletal homeostasis. In particular it appears that sufficient caloric intake, appropriate intake of key micronutrients and a balanced intake between acidic and alkaline foods are the main dietary components known to impact on bone health (Ihle & Loucks, 2004; MacDonald et al., 2004; Prentice et al., 2006).

4.3.4.1. Energy Intake

It has been proposed that the main modifying effect of diet and nutrition on bone healthy and quality is whether or not the diet of the individual allows a state of energy balance to be maintained (Cobb et al., 2003; Zanker & Cooke, 2004). As mentioned in the previous section (4.3.3) energy availability refers to the amount of energy available to the body to perform all other essential homeostatic functions after the energy expended in physical activity has been accounted for (ADA, 2009). Energy imbalance and disordered eating have been reported to significantly affect bone health in female runners (Cobb et al., 2003; Zanke & Swaine, 2000) in accordance with the female athlete triad. This condition has been described as the interrelationship among energy availability, menstrual function and bone mineral density which may have clinical manifestations including eating disorders, functional hypothalamic amenorrhea and osteoporosis (Nattiv et al., 2007). It is thought that inadequate energy intake and the accompanying energy deficit may alter the action of a number of key endocrine factors known to affect bone health. These include the reproductive and metabolic hormones and associated growth factors such as growth hormone and IGF-1 (Haspolat et al., 2007; Misra et al., 2003a; Strauss et al., 1985). The action of these agents was discussed in section 3 and their specific impact on bone will be reviewed in section 4.6.

Bone turnover may be disrupted in favour of resorption at an energy availability below $30 \text{ kcal} \cdot \text{kgLBM}^{-1}$ (Ihle & Loucks, 2004), which is the minimum threshold of energy availability cited as necessary to maintain metabolic function in women (ADA, 2009; Loucks & Thuma, 2003). Recommended minimum thresholds do not appear to have been reported for males but are likely to be similar. In addition, extensive weight loss which occurs as a result of energy deficiency may cause an altered response and action in a number of factors including the adipokines leptin and adiponectin. These factors have been said to have a key role to play in bone loss during times of reduced energy availability (Zanker & Cooke, 2004) along with the aforementioned endocrine agents.

Further evidence of the effect of caloric restriction, which appears to impact primarily on the cortical bone compartment (Hamrick et al., 2008), on bone health was provided in a study examining the relationship between bone density and resting metabolic rate in a group of female ballet dancers (Kaufman et al., 2002). In this study, a strong positive relationship was shown between bone density and RMR. It was suggested that

the reduction in RMR observed may have occurred in response to chronically low energy intake (Hall, 2006). Energy restricted bone mass loss may be reversible once normal feeding is commenced however, as evidenced by an increase in bone mass and a shift of bone turnover toward a formative state following enrolment in an anorexia nervosa recovery program (Bolton et al., 2005).

4.3.4.2. Micronutrients

Adequate intake of key micronutrients has long been cited as a determinant of bone strength and quality (Brown & Josse, 2002; Miller et al., 2001). In particular, calcium and Vitamin D have been suggested as the primary micronutrients known to affect bone health, while additional elements such as potassium, magnesium, ascorbic acids and essential fatty acids may also have a key role to play (Chen et al., 2006; MacDonald et al., 2004; Sebastian, 1994).

Calcium

Calcium is the principle mineral contained within bone mass and as such has a vital structural role to fulfil within the bone tissue (Brandi, 2009). In addition calcium is considered a pleiotropic metabolic agent, with functions in nerve conduction, muscle contraction, cell adhesion and blood coagulation (Miller et al., 2001). Fulfilment of this essential metabolic role in times of calcium imbalance may impact on the structural integrity of bone as bone provides a calcium reservoir for use in times of imbalance (Peacock, 2010). A decrease in circulating calcium levels, triggers activation of receptors on the parathyroid gland, which in turn cause the release of parathyroid hormone (PTH). This hormone aims to restore circulating calcium homeostasis primarily through an increase in osteoclast activity and the liberation of calcium from bone. Calcitonin, released from the thyroid gland acts as an antagonist to the action of parathyroid hormone.

Research regarding the effects of calcium intake on bone is somewhat conflicting. Reid et al., (2008) demonstrated a beneficial effect of 1200 mg·day⁻¹ of calcium on BMD and bone turnover in healthy older men. In addition associations have been demonstrated between dietary calcium intake and the bone formation marker osteocalcin (Earnshaw et al., 1997), while calcium intake has been reported as a significant predictor of femoral neck BMD change during the menopausal transition

(MacDonald et al., 2004). In contrast, no relationship was shown between calcium intake and absolute BMD in these studies (Earnshaw et al 1997; MacDonald et al., 2004), nor have high intakes been indicated as having any effect on fracture risk (Nieves et al., 2008). Increased calcium as provided through milk supplementation has been shown to have no effect on bone mass in a group of young Chinese women (Woo et al., 2007). Dairy products are popularly cited as one of the key determinants of bone health due to the high calcium content of these foods (Miller et al., 2001). A review of the available literature indicates however that the majority of studies conducted have shown a nonsignificant effect of dairy food intake on bone. Those purporting to show a favourable outcome were confounded by a small effect size and a suggestion that high intakes of dairy foods may represent a surrogate for other modifiable lifestyle behaviors such as nutritional knowledge and positive exercise, smoking and alcohol habits (Weinsier & Krumdieck, 2000).

Vitamin D

Vitamin D, or cholecalciferol is actually a hormone, though it is more widely recognized as a micronutrient and has a key role to play in bone health (Mosekilde, 2005). Vitamin D influences bone through a stimulatory effect on calcium and phosphate absorption from the small intestine and by reducing urinary calcium and phosphate excretion (Salasin, 1999b). Insufficient vitamin D levels cause stimulation of parathyroid hormone (PTH) release which induces bone resorption (Mosekilde, 2005). Vitamin D is naturally synthesized from UV exposure, however if sufficient sunlight is unavailable or avoided supplementation may be necessary. Recent research suggests that 97.5% of people in the UK and Ireland may require supplementation in order to maintain even conservative 25(OH) D levels (25nmol l^{-1}) (Cashman et al., 2008). Vitamin D insufficiency, as occurred in response to UV deprivation following an expedition to Antarctica has been shown to impact on calcium and PTH levels as well as bone turnover and bone mass (Iuliano-Burns et al., 2009). Winter time is associated with a reduction in serum vitamin D status and an associated increase in serum PTH and resorptive activity. This finding was associated with an increased proportion of falls resulting in fracture (Pasco et al., 2004) which may reflect the direct and indirect effect of vitamin D on muscle function (Mosekilde, 2005).

Other Micronutrients

Sufficient intake of potassium, magnesium (Ilich et al., 2008; Jones et al., 2001; MacDonald et al., 2004; New et al., 2004) and ascorbic acid/vitamin C (Maimon et al., 2008; Simon & Hudes, 2001) have all been suggested as being essential for preservation of bone regulation and health. The presence of Vitamin C appears to be required for the process of collagen formation (Franchesi, 1992), while potassium and magnesium may have a role to play in the provision of a suitably neutral pH environment. Modern diets tend to be high in animal proteins and processed foods, which generate fixed acids, primarily in the form of sulphuric acid. If a sufficient quantity of alkaline forming foods is not ingested, the skeleton may be required to provide neutralization from its reservoir of basic calcium salts (Sebastian et al., 1994). Dietary supplementation of potassium bicarbonate has been shown to neutralize dietary acid production resulting in a positive effect on mineral balance and bone turnover (Sebastian et al., 1994).

4.3.4.3. Additional Influences

Strong evidence is available supporting a link between fruit and vegetable consumption and bone health (Chen et al., 2006; MacDonald et al., 2004; New et al., 2000), likely due to the high quantity of essential micronutrients and alkaline chemicals contained within these foods.

Some disparity exists as to the role of fatty acids on bone health. A negative relationship has been reported between bone and mono and polyunsaturated fatty acids (MacDonald et al., 2004) and it was suggested that this may be due to a reduction in calcium absorption through the formation of calcium-fatty acid soaps. Interestingly however saturated fatty acids were not shown to have an impact and it was thought that this may be due to their abundance in dairy products (MacDonald et al., 2004). In contrast to this however is evidence from a large cross-sectional study on a rural Chinese population (n = 12,055) which suggested that seafood, which was a large dietary component of the studied population was associated with increased BMD in women (Zalloua et al., 2007). It seems that this is due to the high content of essential fatty acids contained within these foods. Essential fatty acids may enhance the effects of vitamin D so facilitating calcium absorption from the gut. A diet high in n-3 fatty acids has been shown to reduce the rate of bone loss following ovariectomy in an

animal model (Sun et al., 2003). This appeared to occur as a result of decreased osteoclastogenesis due to downregulation of RANKL on activated T-cells and inhibition of NF- κ B activation in osteoclast precursors.

It appears that nutritional intake has a secondary effect on bone when considered in comparison to factors such as genetics and menopausal state (Earnshaw et al., 1997; MacDonald et al., 2004). It is clear however that many aspects of dietary intake have an important role to play in development and maintenance of skeletal integrity. In particular it seems that a calorically sufficient diet with a high composition of fruit and vegetables and dairy products may be most beneficial to bone (Chen et al., 2006; Ihle & Loucks, 2004; Miller et al., 2001). It is important to consider the complex make up of different food types when examining the effects of single macro and micro nutrients on bone health. All foods contain a variety of biologically active compounds and the attribution of a health effect to a single nutrient may in fact mask the effects of many other known or unknown components contained within a particular food, or non-nutritional lifestyle behaviours associated with intake of a particular type of food (Chen et al., 2006).

4.3.5. Hormonal Status and Bone

The effect of mechanical loading must be considered in relation to the metabolic/hormonal state of the body which may influence bone cell sensitivity to mechanical stimuli (Burr, 1997). Muscular loads appear to have the greatest contribution to make toward development of bone health (Arimatsu et al., 2009; Frost, 2003; Wang et al., 2005) and the musculoskeletal system is commonly regulated, with vitamin D, testosterone, cortisol, growth hormone and IGF-1 all cited as the most relevant endocrine factors to consider in the modification of muscle mass (Solomon & Bouloux, 2006) as well as bone. The action of many of these endocrine factors appear to be affected by the extent of available energy within the body (Bergendahl et al., 1996; Hackney et al., 1988; Haspolat et al., 2007) and so may be disrupted within jockeys, who appear to operate in a chronic state of energy deficiency (Dolan et al., 2010; Labadarios, 1988; Leydon & Wall, 2002). The primary functions and actions of the glucocorticoids, androgens and GH/IGF-1 were discussed in section 3. The specific effects of these endocrine agents on bone health will be discussed in this section.

4.3.5.1. Cortisol and Bone

Prolonged exposure to endogenous or exogenous glucocorticoids has a detrimental effect on bone mass and quality (Abad et al., 2001; Di Somma et al., 2002; Weinstein et al., 1998). This appears to occur through an effect on both aspects of bone turnover (bone resorption and formation) (Canalis, 1996; Di Somma et al., 2002) with a reduction in bone formation induced through a reduction in osteoblast birth rate and an increase in mature osteoblast apoptosis the predominant effect (Canalis, 1996). Indirectly cortisol may also influence bone regulation through an effect on calcium absorption and excretion (Canalis, 1996), an inhibition of gonadotropin production (Canalis, 1996) or an inhibitory effect on hepatocyte growth factors (Blanquaert et al., 2000).

Cortisol is released in response to starvation, as observed in anorexia nervosa, likely through an increase in secretory burst frequency. This appears to impact bone mass through a suppression of bone formation (Canalis, 1996; Misra et al., 2004).

4.3.5.2. Sex Steroids and Bone

Both male and female sex steroids (the androgens and estrogens) are known to have a role in bone health (Slemenda et al., 1996; Vandershuerren et al., 2003; Venken et al., 2008) and receptors for both are expressed on bone (Ohlsson & Vandenput, 2009; Venken et al., 2008). The female sex hormones (the estrogens) are thought to have a more primary influence on bone health (Khosla et al., 1998), although the androgens also have an integral and direct role to play (Sims et al., 2006; Vandershuerren et al., 2003). Traditionally it has been suggested that the androgens as the “male” hormones stimulate bone mineral acquisition during puberty, while the estrogens or “female” hormones inhibit it and appear to induce closure of the epiphyseal plates, so terminating longitudinal bone growth (Juul et al., 2001). This theory is supported by the findings of Lorentzon et al., (2005) who concluded that estrogens reduce and androgens increase cortical bone size and that this may be the cause of sexual dimorphism of cortical bone geometry (Lorentzon et al., 2005). It has been suggested that estrogen may exert a biphasic dose dependent effect as it appears to be stimulatory at lower levels as is evident in males and early female puberty and inhibitory at higher doses as seen in late female puberty and female adulthood (Khosla et al., 1998).

Testosterone's primary bone action is to increase periosteal bone formation so increasing bone size (Lorentzon et al., 2005; Romeo & Ybarra, 2007), as well as exerting an indirect action through its effect on muscle development (Vandershuerren et al., 2003). Hypogonadism causes the release of both bone formation and resorption markers with a greater increase shown in resorptive activity resulting in a net loss of bone mass and strength (Venken et al., 2008). Increased resorptive activity reported in hypogonadism appears to occur as a result of a down-regulation of osteoclast activity but not number (Sanyal et al., 2008).

Follicle stimulating hormone (FSH) and lutenising hormone (LH) are pituitary derived glycoproteins which act synergistically to regulate a number of anabolic and reproductive processes including androgen and estrogen release. Both FSH and LH release is controlled by pulses of gonadotropin releasing hormone (GnRH) and evidence is available suggesting a key role for these hormones in the regulation of bone health (Xu et al., 2009). There appears to be a strong inverse relationship between FSH and bone mass in amenorrheic women (Devleta et al., 2004) and it has been suggested that FSH may have a direct effect on bone resorption through a stimulatory action on osteoclast formation and function (Sun et al., 2006). The gonadotropins may also exert an indirect influence on bone through their regulatory effect on gonadal function and reproductive hormone release.

Sex hormone binding globulin (SHBG), is the principal regulator of testosterone and estrogen activity and is thought to have a role to play in the regulation of bone mass (Slemenda et al., 1996) and in osteoporotic fracture risk (Khosla et al., 1998) through an effect on testosterone bioavailability (Ongphiphadanakul et al., 1995).

4.3.5.3. GH/IGF-1 and Bone

The GH/IGF-1 axis has an integral role to play in longitudinal bone growth and the attainment of peak bone mass in childhood and adolescence (Bex & Bouillon, 2003), and in the maintenance of adult bone mass (Mukherjee et al., 2004). It is unclear whether growth hormone's influence on bone is direct or mediated through the action of IGF-1 (Bex & Bouillon, 2003) although IGF-1 has been suggested as being the

principal mediator of GH at the bone tissue level (Jurimae & Jurimae, 2006). GH deficiency is associated with decreased bone mass and increased fracture risk although it is not known whether actual volumetric density is decreased (Bex & Bouillon, 2003). Sex specific effects of GH on bone mass may be related to the pulsatile release of this hormone, as it appears that pulsatile infusion in GH deficient adults may have a more potent effect on markers of bone formation and resorption than does continuous infusion (Jaaffe et al., 2002).

There appears to be a consistent association between IGF-1 and bone mass (Jurimae & Jurimae, 2006; Nindl et al., 2008; Rajaram et al., 1997). It appears that the primary effect of the GH/IGF-1 axis is to induce an effect on osteoblast cell proliferation (Ernst & Froesch, 1988). IGF-1 may also exert an influence on osteoclast activity. In particular IGF-1 impacts on the action and expression of osteoprotegerin (OPG) and the OPG ligand (Zhao et al., 2008). Activation of the OPG ligand results in a rapid differentiation of osteoclast precursors to mature bone resorptive cells. The binding of OPG to its ligand receptor inhibits this action however and so bone resorptive activity is largely dependent on the balance between the two (Hofbauer et al., 2000). Research indicates that IGF-1 has a role to play in regulation of this balance through mediation of stromal cell expression of these molecules (Mrak et al., 2007). Given its apparent role in the regulation of both bone formation and resorption it has been suggested that IGF-1 may aid in the mediation of the complex coupling process of bone remodelling (Rubin et al., 2002c; Ueland, 2004). Growth hormone also appears to have a direct IGF-1 independent role in the regulation of OPG expression (Mrak et al., 2007).

IGF-1's action on bone appears to be primarily mediated through its binding protein IGFBP5. This protein has been suggested as having a high affinity for hydroxapatite and as such may act to fix and protect the IGF's to the skeletal matrix (Campbell & Andress, 1997). It also appears that this binding protein has an IGF independent effect on osteoblast (Campbell & Andress, 1997) and osteoclast (Kanatani, et al., 2000) activity.

4.3.5.4. Leptin and Bone

There appears to be a strong positive association between leptin and bone (Gordeladze & Reselan, 2003; Hamrick et al., 2008; Kaufman et al., 2002) which appears to occur

through a direct effect on osteoblastic bone formation (Steppan et al., 2000). The relationship between leptin and bone density in humans is somewhat equivocal however (Gomez-Ambrosi, 2008). It has been suggested that the effect of leptin on bone mass may be dependent on its effect on fat mass (Jurimae & Jurimae, 2006). The relationship between bone and fat mass is discussed in greater detail in section 4.3.2. Despite the presence of the OB receptor on osteoblast cells it is possible that leptin's primary osteogenic effect is related to the extent of energy availability (Gomez-Ambrosi et al., 2008) and as such may exert a positive (Kaufman et al., 2002) or negative (Hsu et al., 2006) influence on bone regulation.

4.3.6. Alcohol and Smoking

Evidence of the effect of alcohol consumption on bone health is somewhat contradictory. Moderate intake of alcohol appears to have no effect (Williams et al., 2005) or a positive effect on bone mass (Ilich et al., 2008; Ilich et al., 2002). High alcohol intakes do however seem to be detrimental to bone health, as evidenced by a net bone loss in a group of skeletally mature rats fed alcohol, and this appears to occur primarily through a reduction in bone formation (Sibonga et al., 2007). The effect of alcohol on bone may be direct, or to occur indirectly as a result of an associated energy imbalance (Chakkalakal et al., 2002; Sibonga et al., 2007). Smoking also appears to represent a threat to bone health (Korkor et al., 2009; Tamaki et al., 2009) potentially through reduced osteoblastic activity in response to the toxic effects of smoking, although precise mechanisms remain to be determined (Tamaki et al., 2009).

Summary

It is clear that numerous factors are involved in the regulation of bone strength and integrity. Disruptions to any of these may compromise bone's structural integrity resulting in fracture. A common consequence of disordered bone regulation (osteoporosis) will be discussed in the following section, along with an analysis of the primary means of bone mass and strength assessment (DXA scanning).

4.4. Bone Disorders

When the mature body is in its usual homeostatic state the skeletal system is capable of simultaneously maintaining both structural and metabolic function. Disease, trauma and stress may however compromise skeletal integrity, leading to a variety of disorders, of which osteoporosis, which is a bone disorder characterized by low bone mass may be the most relevant to consider here.

4.4.1. Osteoporosis/Osteopenia

Osteoporosis is defined as an asymptomatic systemic bone disease characterized by low bone mass and micro-architectural deterioration of bone tissue with a consequent increase in bone fragility and susceptibility to fracture (Khosla et al., 2008). Essentially, osteoporosis is a condition whereby mechanical bone competence is incapable of maintaining bone integrity without fracture. It was traditionally viewed primarily as a postmenopausal female disorder however the recent literature suggests that osteoporosis also represents a significant health concern for men (Khan et al., 2007; Seeman et al., 2006). Development of osteoporosis is largely dependent on the extent of peak bone mass attained (Mora & Gilsanz, 2003) which occurs largely in the first two decades of life (Bachrach, 2007), and the subsequent rate of bone loss (Hansen et al., 1991). Recent research suggests that peak bone mass of the lumbar spine and total body is attained between the ages of 20 and 23 years in males and 18 and 20 years in females (Boot et al., 2009). This finding contrasts with that previously reported by Szulc et al (2000) who suggested that peak BMD of the total hip and its components is achieved by the age of 25 while peak BMD of the lumbar spine and total body was achieved by the ages of 29 and 37 years respectively (Szulc et al., 2000).

Male osteoporosis is defined as a heterogeneous entity with multiple underlying causes. The main primary and secondary causes of osteoporosis are presented in Table 2.2.

Table 2.2: Primary and Secondary Causes of Osteoporosis

Primary Osteoporosis:

- ⇒ Age Related Osteoporosis
- ⇒ Idiopathic Osteoporosis

Secondary Osteoporosis:

- ⇒ Alcoholism
 - ⇒ Glucocorticoid Excess (endogenous or exogenous)
 - ⇒ Hypogonadism
 - ⇒ Hyperparathyroidism
 - ⇒ Gastrointestinal Disorders
 - *Malabsorption syndromes*
 - *Inflammatory bowel disease, gluten enteropathy*
 - *Primary biliary cirrhosis*
 - *Post gastrectomy*
 - ⇒ Hypercalcuria
 - ⇒ Chronic obstructive pulmonary disease
 - ⇒ Post-transplant osteoporosis
 - ⇒ Neuromuscular diseases
 - ⇒ Systemic Diseases
 - *Rheumatoid Arthritis*
 - *Multiple myeloma*
 - *Other malignancies*
 - *Mastocytosis*
 - ⇒ Medication/Drug Related
 - *Glucocorticoids*
 - *Anticonvulsants*
 - *Thyroid hormone*
 - *Chemotherapies*
 - ⇒ Lifestyle Choices
 - *Cigarette smoking*
 - *Sedentary lifestyle*
-

Source: Khosla et al., 2008.

4.5. Assessment of bone health

In order to allow safe and accurate diagnoses of osteoporosis be made it is essential that validated and reliable means of bone quality assessment are available. Accurate assessment of bone quality is difficult however, as bone quality encompasses a number of aspects, most of which are highly specific to the individual in question. Four key determinants of bone strength have previously been identified. These are:

- ⇒ The properties of bone as a tissue, including its yield point, stiffness, ultimate strength and fatigue life.
- ⇒ The amount of micro-damage present within the bone.
- ⇒ The quantities and types of tissue in the bone (mass).
- ⇒ Architecture and geometry (Frost, 2003).

Bone mass assessment through dual energy x-ray absorptiometry (DXA) scanning is the current measurement of choice for diagnosis of osteoporosis (ISCD Writing Group, 2004b; Khosla et al., 2008; Lewiecki et al., 2008). Functions and limitations of this diagnostic tool will be discussed in this section.

4.5.1. Dual Energy X-Ray Absorptiometry (DXA)

DXA scanning contains two sources of x-ray energy which are directed towards the bone. Transmission of this energy is dependent on the amount of bone mass present as mineral absorbs a greater degree of radiation than either protein or adipose tissue, so providing a relative indication of the amount of tissue present in the body (Cummings et al., 2002). The radiation dose delivered by DXA scanning is extremely low, rendering it one of the safer and more practical means of measuring bone mass (Sturtridge et al., 1996). Areal BMD, as measured by DXA scanning is defined as the mineral mass of the bone (bone mineral content/BMC) divided by its projection area in a given direction. It has been suggested that vertebrae L1-L4 and both proximal hips should be measured during a DXA scan, and the lowest T score of the lumbar spine or any of the three hip regions of interest (femoral neck, trochanter and proximal hip) may be used for diagnosis (Hamdy et al., 2002).

There is a strong correlation between BMD and the strain required to break a bone and this is the basis upon which diagnostic criteria are formed (Abrahamson et al., 2006; Cummings et al., 2002). A bone mass score T score greater than or equal to 2.5 SD below the mean is considered osteoporotic, although it should be noted that this particular value has no inherent biological value as BMD is considered a continuous risk factor with no one cut off point capable of distinguishing between high and low risk individuals (Cummings et al., 2002). Current WHO guidelines were developed based on data collected from Caucasian women over 50 years of age. There is

currently no criterion for densitometric diagnosis of osteoporosis in younger men (ISCD Writing Group, 2004a). Prediction of fracture risk based on BMD scores in this group is difficult as fractures tend to be trauma based rather than as a result of fragility. Z scores, which represent the deviation of bone mass from the age-matched mean, have been suggested as being more appropriate for use in males aged 20 – 64 who do not have any osteoporotic risk factors (ISCD Writing Group, 2004a). Discordance between T and Z scores for males in their 20's along with variance in the means of Z score calculation (Carey et al., 2009) may have implications for the clinical interpretation of this measure however. It has been suggested that the addition of clinical risk factors (Kanis et al., 2007) and analysis of bone turnover (Camoszi et al., 2007) may enhance the fracture prediction ability of DXA scanning.

Although widely used there are a number of limitations associated with the use of DXA scanning. BMD is not a mechanical characteristic *per se* (Melton et al., 2005) but may be considered a surrogate for other factors such as bone size (Center et al., 2004). In addition bone mineral density represents a two dimensional measure ($\text{g}\cdot\text{cm}^{-2}$), while bone volume is a three dimensional entity and considerable errors in assessment may be incurred due to the inability of DXA scanning to measure bone depth (Hogler et al., 2003; Molgaard et al., 1997). It has been suggested that adjustment for the confounding influence of stature may be required so to enhance the accuracy of the diagnosis made (Hogler et al., 2003; Molgaard et al., 1997; Prentice et al., 1994). One difficulty with this technique however is that while this may provide a more accurate measure of bone health, particularly in children (Leonard et al., 2004a) results cannot be validated against a meaningful reference value as can BMD (Fewtrell et al., 2005).

Summary

Bone mass assessment using DXA scanning appeared to reveal low bone mass in a group of Irish jockeys (Warrington et al., 2009). This finding has particular implications for this group in light of the high risk nature of the sport (Hitchens et al., 2009; Turner et al., 2002). Suitable osteogenic interventions may be required and one of the experiments of this thesis involved an assessment of the suitability of whole body vibration therapy as an osteogenic intervention in jockeys. The osteogenic

potential of this intervention and *in vivo* evidence to date will be discussed in the following section.

4.6. Vibration Therapy

Whole body vibration platforms provide an oscillatory stimulus to the musculoskeletal system, with osteogenic effects apparently demonstrated in animal (Christienesen & Silva, 2006; Garman et al., 2007; Maddalozzo et al., 2008; Rubinacci et al., 2008; Sehmisch et al., 2009; Xie et al., 2006, Rubin et al., 2002a; Rubin et al., 2002b) and human (Gilsanz et al., 2006; Rubin et al., 2004; Rubin et al., 2006; Vershueren et al., 2004; Ward et al., 2004; Xiang Yan et al., 2008) studies. Oscillating, or vibration platforms have been cited as the most effective alternative to drug therapy in recovering post menopausal bone loss (Rubin et al., 2006) with vibration appearing to microscopically stress the bone so causing a responsive increase in bone strength.

4.6.1. Vibration as a Natural Stimulus

Tissue vibrations are a natural part of all sporting and daily activities, the extent of which depends on the natural frequency of the vibratory stimulus and the damping characteristic and capacity of the affected tissue. Two biomechanical variables have been suggested as being indicative of vibration intensity, namely amplitude and frequency. Amplitude refers to the extent of the oscillatory motion of the vibration which manifests as the peak to peak displacement. This can be measured in mm, or in accordance with g, the gravitational constant of the earth (9.81 m s^{-2}). Vibration frequency is measured in hertz and is recorded as the repetition rate of the oscillatory cycles (Cardinale & Wakeling, 2005). Individual characteristics may alter the response to and effect of the vibratory stimulus. Active muscle provides a damping effect, so attenuating vibratory forces (Wakeling et al., 2002) while it also appears that peak aerobic power may be related to the response of the body to the vibratory stimulus (Maikala et al., 2006) in that the greater the physical capacity of the body the greater the capacity to absorb and attenuate vibratory stimuli and stress. The vibration platforms available today offer a broad range of amplitudes and frequencies and the safety and application of these devices may vary accordingly. Low magnitude, high

frequency platforms were used in this study however and so this review will focus on the effects of these particular platforms.

Exposure to whole body vibration (WBV) appears to cause an up-regulation of many physiological responses. For example oxygen uptake, heart rate and oxygen pulse response have all been shown to be increased during exposure to WBV (Maikala et al., 2006). Occupational exposure to vibration has been suggested as having a detrimental effect on those habitually exposed to it and is said to be associated with lower back pain (Rozali et al., 2009). This is likely due to the chronic nature of occupational exposure. The potential beneficial effects associated with short term bouts of WBV are likely to be achieved through similar mechanisms as are the detrimental outcomes associated with chronic exposure (Maikala et al., 2006). Vibratory stimuli should be considered as a physiological stressor and as such intensity must be individually and functionally applied if the desired results are to be achieved without adverse effects.

4.6.2. Vibration and Bone

The mechanical stimulation of bone relates to interdependence between strain cycle number, magnitude and frequency rather than by the magnitude of strain alone. It has been suggested that the accumulative effect of a low magnitude strain, applied at a very high frequency, as is the case with the WBV platforms used in this study may allow safe augmentation of the skeleton at strain levels far below those required to cause damage to bone (Judex et al., 2006; Rubin & Lanyon, 1987).

It is unlikely that bone cells are capable of directly sensing the matrix deformation induced by the vibratory stimulus as the magnitude of mechanical strain is quite low. It seems however that WBV may exert an effect on fluid pressure gradients within the lacunar canalicular system. Increased intramedullary pressure is a byproduct of matrix deformation and it may in fact be the resultant fluid flow which provides the stimulus required to generate an adaptive response (Judex et al., 2003; Qin et al., 2003). The fluid pressure gradient in the lacunar canalicular system is affected by signal frequency (Turner et al., 1994) and it has been proposed that this signal may be large enough in WBV to cause stimulation of the osteocytes (Qin et al., 2003) so eliciting an adaptive

response. Stimulation of the osteocytes, osteoblasts and bone lining cells by this mechanism may then have an impact on bone matrix production through the action of intermediary mechanisms such as growth factors, prostaglandins and other mediators (Turner et al., 2004). It appears therefore that WBV may mimic the action of matrix deformation without actually applying the strain magnitudes necessary for deformation to occur (Qin et al., 2003).

4.6.3. *In Vivo* Evidence from Animal and Human Studies

Vibratory stimuli have been indicated as being safely and effectively transmitted to the hip and spine of the human body through the plantar surface of the foot while standing on a WBV device (Kiiski et al., 2008; Rubin et al., 2003a), however its adaptive effect on the musculoskeletal system in humans remains to be conclusively determined due to the limited number of studies available and differences in experimental design and analysis.

A number of studies are available which suggest that WBV may be anabolic to Gilsanz et al., 2006; Xiang Yan et al., 2008) or protective (Rubin et al., 2006; Ward et al., 2004) of bone in human subjects. Rubin et al., (2004) assessed the impact of a low magnitude (0.2g), high frequency (30 Hz) WBV intervention on BMD of the hip, spine and distal radius in a group of postmenopausal women. This study suggested a positive intervention effect as illustrated by an attenuation in bone loss, but no bone gain in the treatment group, indicating a protective but not anabolic osteogenic influence.

In a similar study, Xiang Yan et al., (2008) examined the BMD response to six months of vibration therapy at 30 Hz and 5mm amplitude in a group of postmenopausal osteoporotic Chinese women. The study findings showed a positive osteogenic effect of intervention with a greater increase demonstrated in lumbar bone mass over femoral bone mass. It was suggested that this finding may be due to the direction of the vibratory transmission as the vibratory stimulus travelled cranially along the longitudinal axis of the body in the same direction as the lumbar bone. That the direction of vibratory stimulus may have had a role to play in the adaptive response was supported by the work of Ward et al., (2004), who demonstrated an increase in tibial but not vertebral BMD following a WBV intervention in a group of disabled

children. The lack of vertebral response was attributed to the abnormal stance of the disabled children which was thought to have affected transmission of the vibratory stimulus (Ward et al., 2004). Research in animal studies suggests a site specific effect of WBV with a greater effect demonstrated at sites adjacent to the vibratory stimulus (Xie et al., 2006). This finding was not supported by the work of Christianesen & Silva, (2006) however who hypothesized that differing strain thresholds on individual bones may account for the site specific response to WBV.

Further evidence supporting the potentially osteogenic role of WBV in the human skeleton was provided in a study by Gilsanz et al who indicated that a six month intervention of 10 minutes WBV, five days a week at 30 Hz and 0.3g was capable of enhancing the musculoskeletal system of young women with low BMD. The authors reported significant increases in cancellous bone of the lumbar vertebrae and cortical bone of the femoral midshaft by 2.1 and 3.4% respectively and a 4.9% increase in paraspinous muscle cross sectional area (Gilsanz et al., 2006).

Many of the human studies which promote the osteogenic effect of WBV indicate that this intervention may be most effective in those who have low initial BMD (Gilsanz et al., 2006) or who are in some way physically impaired, be it through age (Rubin et al., 2004; Xiang Yan et al., 2008) or disability (Semler et al., 2008; Ward et al., 2004). Two possible explanations have been proposed to explain the enhanced osteogenic effects in those with low initial BMD: 1) increased signal conductivity as a result of lighter bones and 2) increased sensitivity in bones prone to disuse (Gilsanz et al., 2006). In support of this view is evidence from animal studies which displayed enhanced sensitivity to a vibratory stimulus following ovariectomy (Rubinacci et al., 2008; Sehmisch et al., 2009), a procedure known to induce rapid bone loss and which has been said to provide an appropriate model for the examination of osteoporosis in humans (Lelovas et al., 2008). In addition it appears that genetic predisposition to low bone mass is accompanied by increased sensitivity to anabolic stimuli (Judex et al., 2002). Based on the available research it has been suggested that WBV may be more suited as a rehabilitative device rather than as a preventative treatment (Rubin et al., 2006). In support of this contention is evidence that vibrations applied to the right leg of 19-week-old anaesthetised female mice in the absence of weight bearing was effective at increasing trabecular bone formation in the metaphysis and at enhancing

cortical bone morphology of the epiphysis. Results from this study provided evidence that WBV may be potentially anabolic to those who cannot tolerate weight bearing activity such as patients with spinal injuries or muscular dystrophy (Garman et al., 2007).

Whether WBV is capable of being anabolic and osteogenic to those who do not have low initial BMD, or are not in any way physically impaired would appear to have yet to be validated. Recent animal studies have shown however that WBV is anabolic to healthy growing bone as indicated by improvements shown in bone mass in a group of physically active growing young mice subjected to three weeks of WBV at 45 Hz and 0.3g (Xie et al., 2006). It appeared that the new bone laid down in response to WBV in this study may have been of a comparable quality to that which was already there, although possibly of a lesser maturity. WBV has been shown to be ineffective however at enhancing any aspect of bone mass, quality or turnover in a group of young, healthy adults in a study by Torvinen et al., (2003), although an improvement in jump height did suggest a degree of neuromuscular adaptation to the vibratory stimulus. Further research may be required so to assess the effect of WBV on healthy bone, and to examine whether it is capable of conveying an anabolic as well as protective effect in humans.

The osteogenic potential of vibration therapy appears to be strongly dependent on compliance (Rubin et al., 2004). Gilsanz et al., (2006) indicated a direct dependence of efficacy on compliance. This study did report however that brief daily exposure to WBV is all that is required, with just two minutes per day suggested as the cut-off threshold after which no further improvements were noted (Gilsanz et al., 2006). These results suggest that bone's biological response to WBV may be triggered and not accumulated, a suggestion which is supported in the study by Ward et al., (2004), who showed no evidence of a relationship between efficacy of intervention and compliance and which cited this as being indicative of minimal influence of duration of intervention on changes in proximal tibial BMD. A study by Kiiski et al., (2008) which examined the transmission of WBV to the human skeleton appeared to support this notion. Bone's osteogenic response to loading appears to become saturated after 36 load cycles (Rubin & Lanyon, 1984). The typical vibration protocol delivers loads far in

excess of this and so it was suggested that the majority of vibration stimuli may be delivered in vain (Kiiski et al., 2008).

4.6.4. Potential Impact on Lean and Adipose Tissue

An additional purported benefit of WBV is that it appears to enhance the entire musculoskeletal system and not just bone tissue. Gilsanz et al., (2006) showed an increase in muscle mass following a six month vibration therapy intervention. As lean tissue is strongly correlated with bone mass (Arimatsu et al., 2009; Wang et al., 2005) and given the importance of the musculoskeletal system in the prevention and amelioration of fall severity, this is a key benefit of WBV interventions and provides evidence of a more holistic anti-osteoporotic therapy. In support of this suggestion was a pilot study which examined the effects of WBV in a group of eight children with osteogenesis imperfecta and which showed apparently enhanced musculoskeletal force and mobility following a six month intervention. While data from this study can be considered to be quite preliminary they provide an indication of the potentially therapeutic effects of WBV (Semler et al., 2008).

In addition to the anabolic potential for the musculoskeletal system, animal models have suggested that WBV may be capable of reducing body mass (Sehmisch et al., 2009) through a slowing of fat acquisition (Maddalozzo et al., 2008) and inhibition of adipogenesis (Rubin et al., 2007). The study by Madalozzo et al., (2008) examined the effect of a 12 week WBV intervention at 6mm and 30 – 50 Hz interspersed with rest periods, and showed a reduction in fat mass, but no change in lean mass. No differences in food intake were observed between the experimental and control groups and it was suggested that an increase in energy expenditure achieved through WBV without a concomitant increase in food intake may explain the attenuated weight gain shown in the vibrated rats.

Supporting this study was evidence that 15 weeks of WBV at 0.2g and 90 Hz, for five days a week was associated with a decrease in fat mass in a group of 40 male, seven-week-old mice (Rubin et al., 2007). Evidence from this study suggests that reduced adiposity was achieved through an attenuation of fat precursor cell differentiation. Considering that WBV has previously been suggested as being anabolic to both bone

and muscle (Gilsanz et al., 2006) it was suggested that WBV may enhance the musculoskeletal system by encouraging non-committed mesenchymal stem cell precursors toward a connective tissue lineage (Rubin et al., 2007).

Summary

Results from this review of literature appear to suggest that jockeys are subject to many of the challenges typically associated with weight category athletes, although detrimental impacts may be exacerbated in this group due to the length and intensity of the racing season. Chronic energy restriction, accompanied by acute weight loss practices appear to be prevalent within this group. The associated hypo-hydration and energy deficiencies are likely to have an impact on a range of parameters in jockeys, including physiological, metabolic, osteogenic and cognitive function. There appears to be a dearth of population specific research available however which examines this issue. The aim of this research project therefore was to examine these parameters in a group of jockeys. The results of the four experiments undertaken are presented in chapters three – six.

Chapter Three

Study One: The Effects of a Four% Reduction in Body Mass in 48 Hours on Physiological and Cognitive Function in Jockeys



3.1. Abstract

Aim: To examine the impact of a four % body mass reduction within 48 hours on physiological and cognitive function in a group of professional jockeys. **Methods:** Eight jockeys (23.8 ± 7 yrs; 1.68 ± 0.5 m; 58.24 ± 5.3 kg and 20.74 ± 1.74 kg m⁻²) and eight age and BMI matched male controls (22.2 ± 3.2 yr; 1.73 ± 0.03 m; 66.9 ± 7.9 kg and 22.2 ± 2.9 kg m⁻²) took part in this study. Participants were required to perform a maximal incremental cycle ergometer test to volitional fatigue with physiological function assessed through measurement of respiratory metabolic measures, heart rate, lactate and RPE. Motor response, decision making, executive function and working memory were assessed using a computerized cognitive test battery. Following baseline testing jockeys were required to reduce their body mass by four % using the means typically adopted in preparation for racing, and to return 48 hours later for repeat-testing. The control group completed the same test protocol 48 hours apart, whilst maintaining usual dietary and activity habits between the tests. **Results:** The jockey group significantly reduced their body mass by 3.6 ± 0.9 % ($p < 0.01$), whilst the control subjects showed no significant change in body mass between test trials. Peak values for VO_2 (l min⁻¹), RER ($\text{VCO}_2 \cdot \text{VO}_2^{-1}$), lactate (mmol l⁻¹) and heart rate (b min⁻¹) did not change between the tests for either group. In contrast, peak power achieved significantly decreased for the experimental group (213 ± 27 Vs 186 ± 23 Watts, $p < 0.01$), whilst no significant difference was observed for the control group between the two tests. The jockeys showed higher VO_2 and heart rate at a number of submaximal workloads ($p < 0.05$). No differences were identified for the control group for any submaximal variable. No changes were identified for any cognitive variable in the jockey group following a four % reduction in body mass **Conclusion:** Acute weight loss of the magnitude typically undertaken by jockeys in preparation for competition appears to lead to impaired physiological function which may impact on the performance and possibly the safety and well-being of jockeys.

3.2. Introduction

Making weight for sports refers to the practice of rapidly reducing body mass prior to competition (Fogelholm et al., 1993) and appears to have a negative impact on athletic performance if body mass loss is greater than that which is physiologically tolerable to the body (Brownwell et al., 1987). It has been suggested that any body mass loss necessary for competition should be undertaken gradually over a period greater than seven days and mass should comprise of adipose tissue (Fogelholm, 1994). In contrast rapid weight loss (< seven days) may be to the detriment of work capacity and performance (Burge et al., 1993; Filaire et al., 2001; Umeda et al., 2004). Rapid weight loss practices have previously been shown to have a detrimental impact on athletic performance in a number of weight category sports, including judo (Filaire et al., 2001; Umeda et al., 2004); boxing (Hall & Lane, 2001), lightweight rowing (Burge et al., 1993) and wrestling (Oppliger et al., 1996).

Rapidly reducing body mass prior to competition appears to be an integral feature of horse-racing (Dolan et al., 2010; Labadarios, 1988; Leydon & Wall, 2002; Moore et al., 2002). One of the key challenges facing jockeys is the pressure of making weight and staying at weight throughout the protracted racing season. Evidence suggests that the primary methods used by jockeys to make weight for racing are dehydration by a number of mechanisms, including the use of saunas and exercising while wearing heavy clothing or sweat suits, along with severely restricted fluid and food intake (Dolan et al., 2010). Such methods of weight regulation are consistent with research conducted in other weight category sports such as wrestling, boxing and judo (Burge et al., 2001; Oppliger et al., 1996) and are known to have an impact on athletic performance and physiological and cognitive function. Detriments to both physical and mental function following a rapid reduction in body mass likely occur as a result of associated dehydration and energy deficiency. The detrimental effects of dehydration on both physiological (Coyle et al., 2004; Ebert et al., 2007; Sawka et al., 2007) and cognitive (Cian et al., 2001; Gopinathan et al., 1988; Tomporowski et al., 2007) function are well documented. In addition, restricted dietary intake and an associated energy deficient state are known to impact on exercise performance (De Souza & Williams, 2004; Nattiv et al., 2007; Oliver et al., 2007). It is likely that rapid weight loss practices may exert a similar detrimental impact on both physiological and

cognitive function in jockeys. There appears to be a dearth of population specific research detailing the impact of such practices on athletic function and performance in this group however and so the aim of this study was to examine the impact of rapid weight loss on physiological and cognitive function, work capacity and performance in a group of professional jockeys.

3.3. Methods

Aims and Objectives:

The aim of this study was to determine the extent of body mass reduction habitually undertaken by jockeys and to examine the effect of a four % reduction in body mass in 48 hours on physiological and cognitive function in a group of professional jockeys.

Objective One:

To assess the typical magnitude of weight loss adopted by jockeys in preparation for racing using a self report questionnaire.

Objective Two:

To identify weight loss practices used through the maintenance of a food and weight loss diary throughout the period of weight reduction.

Objective Three:

To examine the impact of a four % reduction in body mass on physiological function in a group of jockeys, as assessed through performance on an incremental cycle ergometer test to volitional fatigue.

Objective Four:

To examine the impact of a four % reduction in body mass on cognitive function as assessed through performance on a laptop based cognitive test battery.

Hypothesis:

That reducing body mass by the magnitude indicated and time allowed would have a negative impact on both physiological and cognitive function in this group of jockeys.

3.3.1. Preliminary Questionnaire

Prior to implementation of this study, a self report questionnaire was completed by 24 jockeys (10 flat and 14 national hunt) at racecourses around Ireland. A copy of this questionnaire is included in appendix C and protocol for the main study was

determined based on the results attained. Response to this questionnaire provided an evaluation of typical body mass on racing and non-racing days. Participants provided information regarding typical non-racing and racing body mass, and the greatest amount of mass lost prior to a race. Typical and lowest riding mass was calculated as a percentage of usual self reported non-riding mass. Participants also provided information regarding the typical amount of time given to achieve the stipulated weight standard.

3.3.2. Study Design Overview

Eighteen male participants were recruited to take part in this study (nine jockeys and nine age, gender and BMI matched controls). All experimental and control participants were required to report for three separate trials: 1) habituation trial 2) baseline trial and 3) experimental trial. Trials two and three were conducted 48 hours apart and consisted of a series of tests of physiological and cognitive function as well as hydration and body composition assessment. The jockey group were required to reduce their body mass by four % of their baseline measure in the 48 hours between test trials. The control group were instructed to maintain usual dietary and physical activity habits between test trials (see figure 3.1). In addition all participants were required to maintain a food diary for the 48 hours between test trials, and to record any additional methods used to actively reduce body mass. Ethical approval for this study was granted by the Dublin City University Research Ethics Committee.

3.3.3. Participants

Nine professional and apprentice full-time jockeys volunteered to participate in this study. Experimental participants were recruited via advertisement at Irish racecourses and telephone recruitment. Any participant for whom the required body mass loss caused them to weigh-in lower than minimum stipulated weight standards (Flat: > 52.7 kg; National Hunt: > 62 kg) was excluded from the study. Nine age and BMI matched males were also recruited, via email advertisement within Dublin City University, to act as a control group. Prior to testing all participants provided written informed consent and completed medical screening forms. Any participant who was absolutely contraindicated from exercise participation was excluded from the study. All

participants were requested to abstain from alcohol and from any unusual and strenuous physical activity for 24 hours prior to collection of baseline data.

3.3.4. Anthropometric Assessment

3.3.4.1. Body Mass and Stature

Body mass was assessed in minimal clothing and reported to the nearest 100g using a portable digital scales (Salter, Germany). Stretched stature was measured to the nearest mm using a portable stadiometer (Seca, Leicester Height Measure).

3.3.4.2. Body Composition

Harpenden skinfold callipers (Cambridge Scientific Industries, UK) were used to measure double thickness subcutaneous adipose tissue on the right side of the body. The following 7 skinfold sites were used: Biceps, triceps, subscapular, suprailiac, abdominal, mid-thigh, medial calf. All measurements were taken in accordance with published guidelines (ACSM, 2006). A minimum of three repeated measures were taken at each site, with additional measures taken if any measurement varied by more than 1mm. Order of measurement was repeated on a rotation basis. Body density was estimated using the regression equation based on the work of Withers et al (1987). The sum of skinfolds was converted to an estimation of body density (BD) using the equation:

$$BD = 1.0988 - 0.0004 * \text{sum of seven skinfolds}$$

Body density was then converted to % body fat (BF) using the equation proposed by Siri, (1956)

$$BF = (495/BD) - 450$$

3.3.5. Hydration Assessment

Hydration status was assessed through measurement of urine specific gravity (Usg) using the TS400 refractometer (Leica Microsystems, Germany) which compares the weight of urine to that of distilled water. This means of assessment has been suggested as a practical and reliable means of hydration status evaluation (Oppliger et al., 2005). A value of 1.020 was accepted as the threshold of euhydration with values \geq to this taken as being indicative of a dehydrated state (Armstrong et al., 2005; Oppliger & Bartok, 2002; Sawka et al., 2007).

3.3.6. Incremental Cycle Ergometer Test

3.3.6.1. Respiratory Measures

Physiological function was assessed through performance on a Monark ergomedic 828E cycle ergometer (GIH, Sweden) using a continuous incremental step test to volitional exhaustion. The test protocol began with a two minute warm up at 60 watts followed by three minute stages commencing at 60 watts and increasing in 30 watt increments until volitional exhaustion. Gas exchange was measured via indirect calorimetry using the Innocor Metabolic Cart (Innovision, Denmark). Oxygen uptake (VO_2), expired carbon dioxide (VCO_2) and the respiratory gas exchange ratio (RER) were continuously recorded throughout the test by the photoacoustic spectroscopy method. The system was calibrated according to manufacturer specifications prior to each test session and included a test of gas flow, gas delay and ambient air composition.

3.3.6.2. Heart Rate Measurement

Heart rate was measured continuously throughout the test via telemetry using a Polar heart rate monitor (Polar Vantage NV, Port, Washington, NY) and recorded at the end of each three minute stage.

3.3.6.3. Lactate Accumulation

Blood lactate was analysed at the end of each three minute stage using a portable hand held device (Lactate Pro, Arkray Factory Inc, Finland). Measurement range for this device is 0.8 – 23.3 mmol l⁻¹. Precision coefficient of variation for normal level lactate samples was 3.2% and accuracy testing against the enzymatic method provided a correlation coefficient of $r = 0.9988$. Blood obtained from earlobe puncture was applied to the test strip containing potassium ferrocyanide and lactate oxidase, causing the production of ferrocyanide and pyruvate. The application of an electrical voltage across the strip caused ferrocyanide oxidation, the current created from which is directly proportional to blood lactate concentration.

3.3.6.3. Rating of Perceived Exertion (RPE)

A subjective rating of perceptual effort was measured according to Borg's rating of perceived exertion scale, which ranges from 6 to 20 (7 = very very easy; 19 = very very hard) (Borg, 1982).



Illustration 3.1. Physiological Assessment

3.3.7. Cognitive Function

Cognitive function was assessed using a specifically designed computer based cognitive test battery which was developed specifically for use in this study and in

conjunction with the sports psychology department in the University of Limerick. Four specific tests were included:

1. Simple Reaction Time (SRT)

Simple reaction time was measured as time in ms taken to respond to the appearance of a single coloured disk on screen and provides a measure of speed of motor response (Erlanger et al., 2003).

2. Choice Reaction Time (CRT)

Choice reaction time was measured as time in ms taken to respond to the appearance of one of two coloured discs and evaluates speed of decision making (Erlanger et al., 2003).

3. Stroop Test (ST)

The stroop test was used to test executive function, which is the ability of the brain to react to novel situations outside of the domain of the “automatic” psychological processes. This was measured as the time taken to react to both a baseline and interference test trial (Sibley et al., 2006).

4. Rapid Visual Information Processing (RVIP)

RVIP was recorded as time in ms taken to respond to a sequence of three odd or three even numbers from the appearance of a series of digits between 1 and 9. The test lasted for three minutes and the number of correct responses, and errors made were recorded, as well as time taken to respond. This test provides a measure of working memory (Smit and Rogers, 2000) which is an executive aspect of short term memory involved in the interim integration, processing, disposal and retrieval of information.

3.3.7.1. Habituation Trial

Each specific cognitive function test was preceded by a brief preparatory trial. Habituation to the test protocol was achieved through completion of the entire test battery three times prior to baseline testing. Efficacy of habituation was tested through a repeated measures ANOVA, whereby no further learning effect was deemed to have

occurred if no significant difference ($p > 0.05$) was identified between habituation trial three and the baseline trial. Data are presented in both their uncorrected and corrected forms. Uncorrected data represent the mean response time to each test excluding any simple reaction time response $> 500\text{ms}$ and any choice reaction time response $> 750\text{ms}$. Corrected data refer to the mean response time taken following removal of any response time which varied more than two standard deviations from the mean.

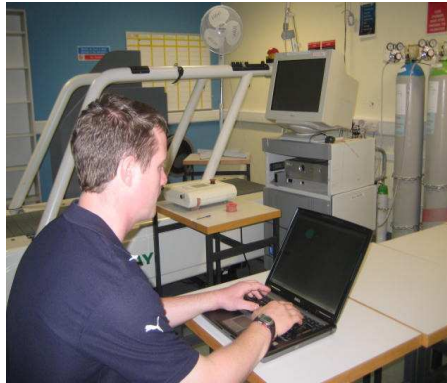


Illustration 3.2. Cognitive Function Assessment

3.3.8. Nutritional Analysis

Participants were asked to maintain a detailed food diary for the 48 hours of weight reduction/maintenance (see appendix E). Precise instructions were provided to each participant regarding the details to be recorded in this diary. The completed food diaries were analysed using a validated dietary analysis package (Dietplan 6.3, Forestfield Software Ltd, Sussex, UK). Food portion sizes were estimated using standard UK references. Resting metabolic rate was estimated according to the equation proposed by Mifflin et al (1990), i.e.

$$RMR = 66.47 + (13.75 * Weight \text{ kg}) + (5 * Ht \text{ (cm)}) - 6.76 * age \text{ (yr)}$$

Estimated RMR was then used to calculate a physical activity level (PAL) from the ratio of reported energy intake to estimated RMR (Goldberg et al., 1991). Participants were also asked to record any additional strategies used to actively reduce body mass within this diary.

3.3.9. Statistical Analysis

Normality of data distribution was tested using the Shapiro Wilks test. Differences between the groups were identified using an independent samples t-test, or Mann Whitney U test depending on data distribution. Pre-post within group differences were assessed using a paired sample t-test or Wilcoxon signed ranks test. Significance was accepted at the level of $p < 0.05$.

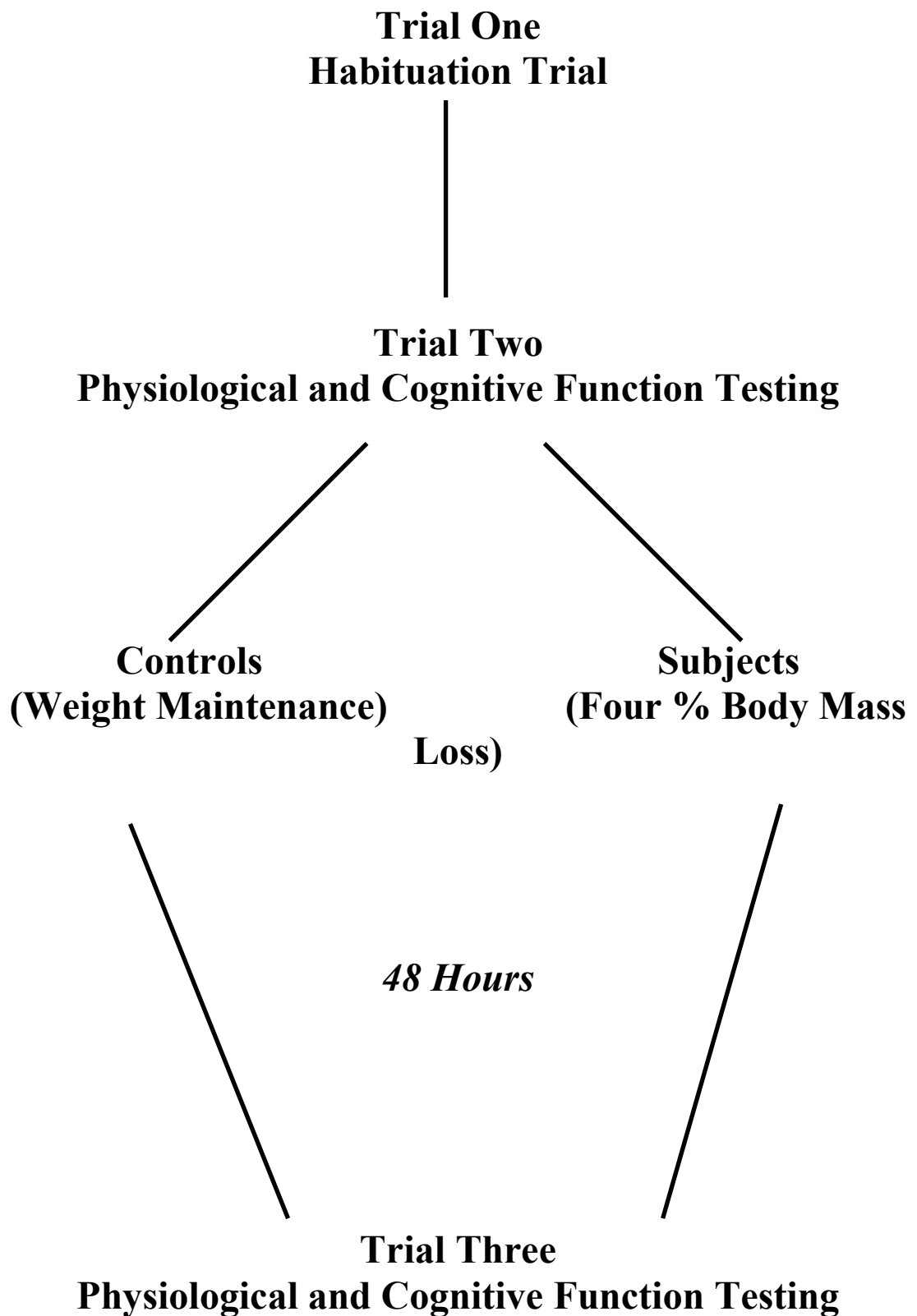


Figure 3.1: Schematic Representation of Experimental Design

3.4. Results

3.4.1. Preliminary Questionnaire

A summary of the body mass regulation questionnaire is presented in Table 3.1. Briefly, typical riding mass was reported to be $3.6 \pm 2.4\%$ below non-riding mass ($2.2 \pm 1.4\text{kg}$). The greatest amount of body mass lost previously for a race was $3 \pm 1.4\text{kg}$ ($5.3 \pm 2\%$ below self reported non-riding mass; range: 2.3 – 10.5%). Participants were typically provided with 34 ± 13 hours notification of the required weight standard (range 24 – 72). The least amount of notification provided as to the required body mass was 19 ± 8 hours (range 3 – 24). No differences were reported between the flat and national hunt groups for any body mass loss parameter ($p > 0.05$).

Table 3.1: Weight Loss in Jockeys

	Total (n = 24)	Flat (n = 10)	National Hunt (n = 14)
Non Riding Mass (kg)	-	$53.8 \pm 3^{**}$	$64.3 \pm 3.5^{**}$
Riding Mass (kg)	-	$51.6 \pm 2.2^{**}$	$62.2 \pm 2.4^{**}$
Mass Loss (kg)	2.2 ± 1.4	2.3 ± 1.1	2.04 ± 1.7
Range (kg)	0 – 5.5	0.5 – 4.1	0 – 5.5
Mass Loss (%)	3.6 ± 2.4	4.2 ± 1.9	3.1 ± 2.5
Range (%)	0 – 7.9	0.9 – 7	0 – 7.9
Largest Mass Loss (kg)	3 ± 1.4	2.6 ± 0.9	3.3 ± 1.7
Range (kg)	1.4 – 7.3	1.4 – 4.1	1.4 – 7.3
Largest mass Loss (%)	5.3 ± 2	5.4 ± 1.5	5.3 ± 2.3
Range (%)	2.3 – 10.5	3.4 – 7.6	2.3 – 10.5

*Data presented as mean \pm SD, ** $p < 0.01$*

3.4.2. Descriptive Data

Descriptive and anthropometric data of the subjects are presented in Table 3.2. Groups were age, gender and BMI matched. A number of differences were illustrated in indices of body composition between the groups (see Table 3.2). Mean jockey body mass was

reduced by 2.1 ± 0.5 kg between the test trials, equating to a mean loss of 3.6 ± 0.9 %. Control participants maintained mean body mass between test trials.

Table 3.2: Descriptive Data

	Jockeys (n = 9)		Controls (n = 9)	
	<i>Baseline</i>	<i>Retrial</i>	<i>Baseline</i>	<i>Retrial</i>
Age (yrs)	24 ± 7	-	22 ± 3	-
Height (m)	1.68 ± 0.05	1.67 ± 0.05	$1.74 \pm 0.03^{\diamond}$	$1.73 \pm 0.03^{\diamond}$
Body Mass (kg)	58.2 ± 5.3	$56.2 \pm 5.3^{**}$	$66.9 \pm 7.9^{\diamond}$	$66.9 \pm 7.3^{\diamond\diamond}$
BMI ($\text{kg}\cdot\text{m}^{-2}$)	20.74 ± 1.74	$20.03 \pm 1.7^{**}$	22.23 ± 2.9	$22.1 \pm 2.8^{\diamond}$
Sum of Skinfolds (mm)	51.14 ± 8	50.34 ± 8	$74 \pm 25.1^{\diamond}$	74.4 ± 26.2
Body Density	1.078 ± 0.003	1.079 ± 0.003	$1.069 \pm 0.01^{\diamond}$	1.069 ± 0.01
Body Fat (%)	9.03 ± 1.4	8.9 ± 1.4	$12.97 \pm 4.3^{\diamond}$	13.07 ± 4.6

*Data presented as mean \pm SD, $^{\diamond}p < 0.05$ between groups, $^{\diamond\diamond}p < 0.01$ between groups, $^*p < 0.05$ between trials, $^{**}p < 0.01$ between trials*

3.4.3. Urine Specific Gravity (Usg)

No baseline differences in hydration status, as measured by urine specific gravity were demonstrated between the groups. Both groups were categorised as euhydrated at the baseline trial. Jockey urine density significantly increased following the reduction in body mass from 1.019 ± 0.004 to 1.028 ± 0.005 ($p < 0.01$), demonstrating a high degree of dehydration following the four % reduction in body mass (see figure 3.2). All experimental participants returned to complete the second trial in a state of dehydration ranging from 1.020 – 1.036. Control participants maintained a state of mean euhydration between test trials.

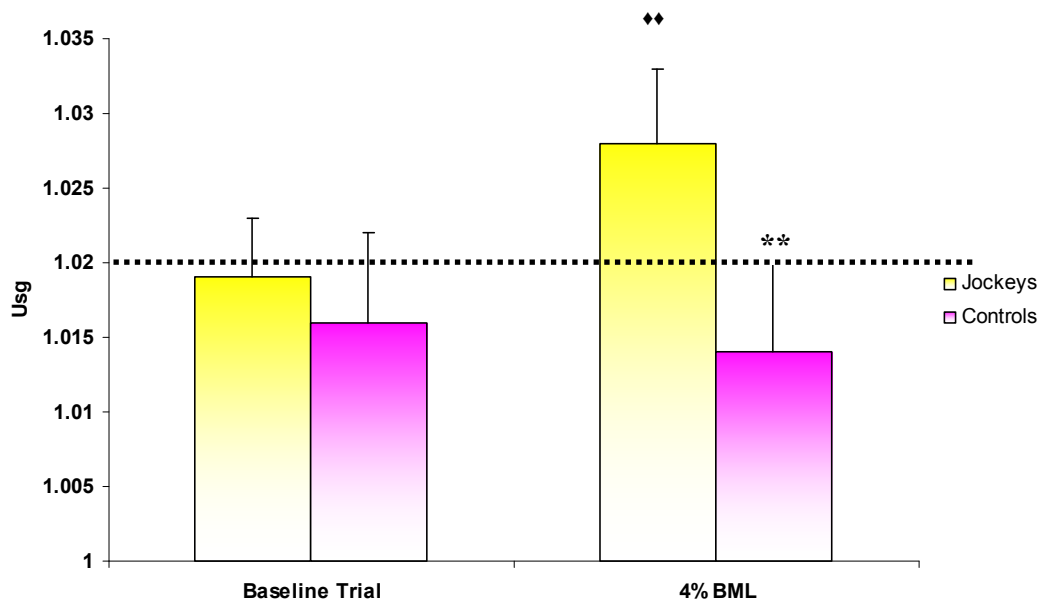


Figure 3.2: Hydration Change Between Trials

♦ $p < 0.05$ between groups, ♦♦ $p < 0.01$ between groups, * $p < 0.05$ between trials, ** $p < 0.01$ between trials denotes dehydration threshold (1.020) (Sawka et al., 2007)

3.4.4. Cycle Ergometer Test Data

3.4.4.1. Resting and Peak Physiological Data

A summary of the resting and peak physiological values are presented in Table 3.3. No differences were identified for any resting variables either between trials or between groups, although a trend toward a significantly decreased respiratory exchange ratio (RER) following a four % reduction in body mass was identified in the jockey group ($p = 0.076$).

A number of differences were shown between the groups for absolute peak values. In particular control participants had a significantly higher VO_2 ($\text{l}\cdot\text{min}^{-1}$) and peak power output (PPO) than the jockey group. In contrast VO_2 ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and relative PPO ($\text{watts}\cdot\text{kg}^{-1}$) were not significantly different between the groups. No differences were shown for any peak physiological variable between the test trials for the control group. Peak power output, expressed both as watts and $\text{watts}\cdot\text{kg}^{-1}$ was significantly reduced

following weight reduction in the jockey group (see Table 3.3). In addition, a trend toward a significantly increased peak heart rate was identified between trials in the jockey group ($p = 0.061$). One experimental participant's physiological results were excluded from the analysis due to technical faults on the retrial and so $n = 8$ for cycle ergometer test results.

Table 3.3: Resting and Peak Physiological Values

	Jockeys (n = 8)		Controls (n = 9)	
	<i>Baseline</i>	<i>Retrial</i>	<i>Baseline</i>	<i>Retrial</i>
<i>Resting Data</i>				
VO₂ (l·min⁻¹)	0.283 ± 0.07	0.307 ± 0.072	0.328 ± 0.04	0.345 ± 0.06
VO₂ (ml·kg⁻¹·min⁻¹)	4.79 ± 1.08	5.45 ± 1.41	4.93 ± 0.60	5.18 ± 0.84
RER	0.88 ± 0.1	0.79 ± 0.11	0.9 ± 0.14	0.87 ± 0.1
Heart Rate (b·min⁻¹)	62 ± 5	64 ± 9	64 ± 6	63 ± 6
Lactate (mmol·l⁻¹)	1 ± 0.2	0.86 ± 0.09	0.93 ± 0.15	0.913 ± 0.125
<i>Peak Data</i>				
VO₂ (L·min⁻¹)	2.71 ± 0.29	2.65 ± 0.304	3.07 ± 0.29 [†]	3.18 ± 0.29 ^{††}
VO₂ (ml·kg⁻¹·min⁻¹)	46.37 ± 3.72	47.23 ± 6.29	46.6 ± 7.97	48.16 ± 7.67
RER	1.21 ± 0.17	1.15 ± 0.138	1.2 ± 0.11	1.22 ± 0.12
Lactate (mmol·l⁻¹)	10.09 ± 2.67	9.3 ± 3.7	11.93 ± 3.7	12.08 ± 2.28
Heart Rate (b·min⁻¹)	176 ± 11	184 ± 5	187 ± 9	184 ± 5
Time to Exhaustion	20.16 ± 2.7	17.52 ± 0.8 ^{**}	23.47 ± 1.6 [†]	23.46 ± 0.69 ^{††}
Workload (watts)	213 ± 27	186 ± 23 ^{**}	246 ± 16 [†]	246 ± 19 ^{††}
Relative workload (watts·kg⁻¹)	3.6 ± 0.42	3.28 ± 0.48 ^{**}	3.7 ± 0.36	3.7 ± 0.4

*Data presented as mean ± SD, [†]p < 0.05 between groups, ^{††}p < 0.01 between groups, * p < 0.05 between trials, ^{**}p < 0.01 between trials.*

3.4.4.2. Submaximal Data

In contrast to the peak respiratory, heart rate and lactate values achieved a number of physiological variables revealed significant differences at set submaximal workloads

throughout the second test trial in the jockey group. Examination of submaximal data at set workloads (60, 90, 120 and 150 watts) displayed significant differences between trials on a number of variables. Results for VO_2 ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), heart rate and blood lactate response are presented in figures 3.3 – 3.5. Subjective reporting of perceived exertion (RPE) was also shown to be significantly increased at 150 watts ($p < 0.05$). No differences were shown within the control group for any variable at any set workload.

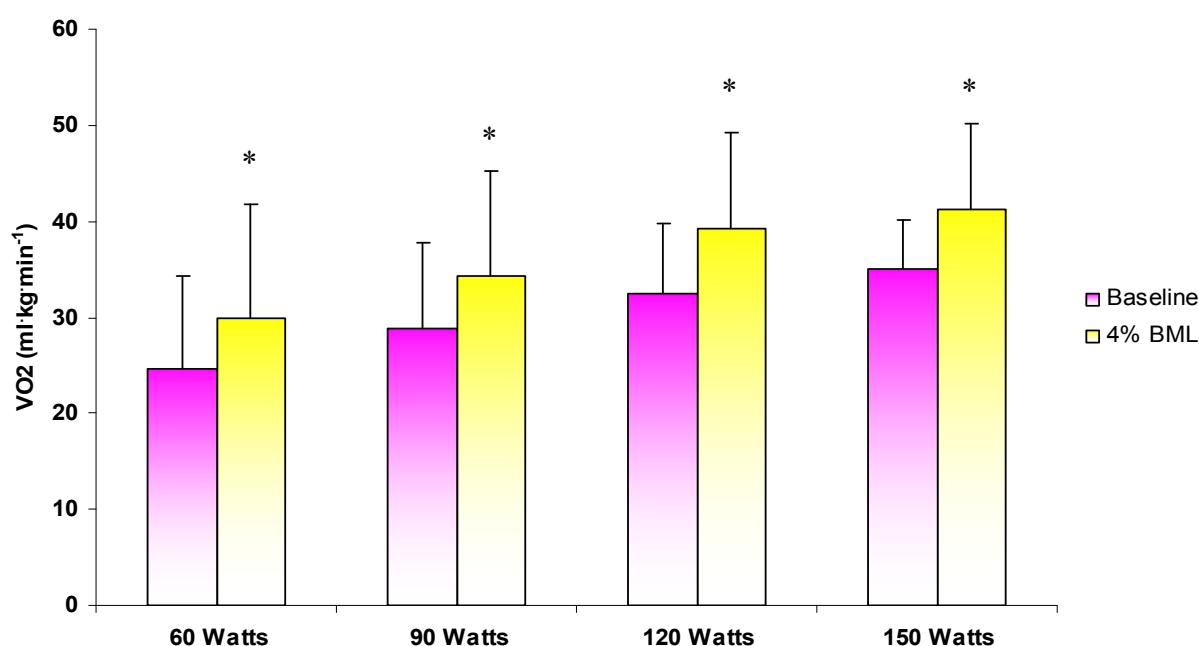


Figure 3.3: Exercise Economy (VO_2 $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) response for Jockey Group

Data presented as mean \pm SD, * $p < 0.05$

Changes in exercise economy for the jockey group following the four % body mass loss are presented in figure 3.3. Submaximal VO_2 response ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) was shown to be significantly elevated at each set workload ($p < 0.05$) examined indicating an increased oxygen demand at each submaximal workload.

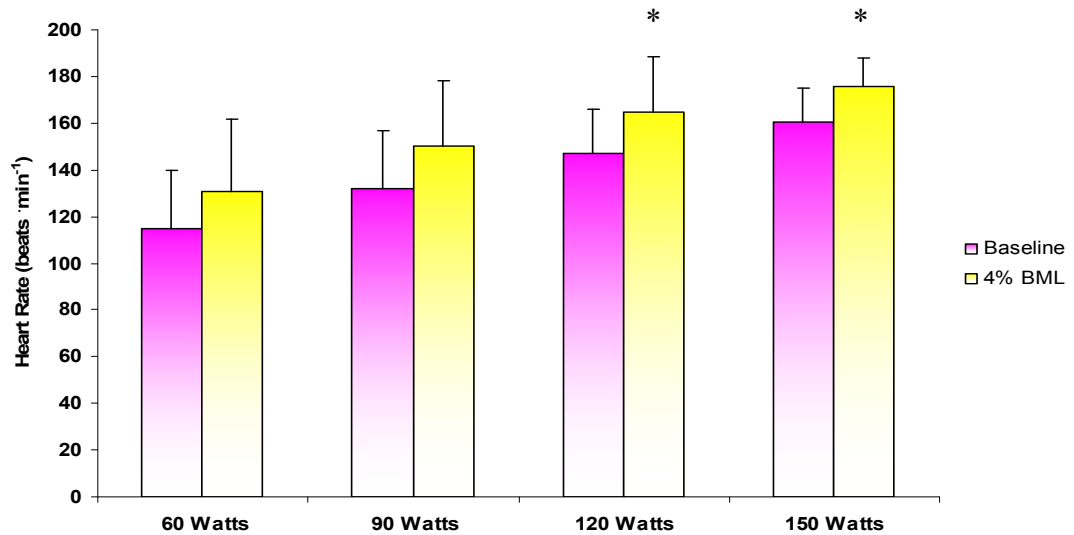


Figure 3.4: Heart Rate Response During Submaximal Exercise for Jockey Group
*Data presented as mean \pm SD, * $p < 0.05$*

Heart rate response was elevated at each submaximal workload and this was shown to be significant at 120 and 150 watts ($p < 0.05$) following a four % reduction in body mass (see figure 3.4). In addition a trend toward significance was shown between test trials at 90 watts ($p = 0.069$).

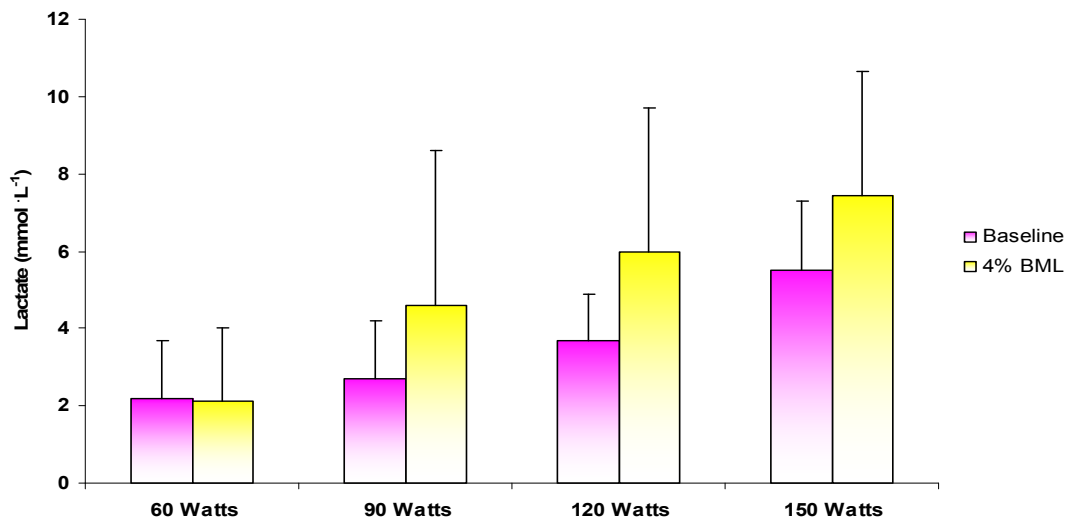


Figure 3.5: Blood Lactate Response During Submaximal Exercise For Jockey Group
*Data presented as mean \pm SD, * $P < 0.05$*

The blood lactate response during submaximal exercise was elevated at 90, 120 and 150 watts, however at no workload was this shown to be significant. A trend toward elevated lactate accumulation at 120 and 150 watts was however identified ($p = 0.063$ and $p = 0.060$ respectively).

Examination of submaximal data at relative workloads (20, 40, 60 and 80% baseline PPO) revealed similar results and data are presented in appendix D.

3.4.5. Cognitive Data

3.4.5.1. Uncorrected and Corrected Cognitive Results

Uncorrected cognitive results are presented in Table 3.4. Differences between the groups were apparent on a number of tests, with control participants demonstrating a significantly faster response time on the 1st phase of the simple reaction time test and the stroop baseline and interference tests. Experimental subjects did not display any significant cognitive changes between trials. Control participants showed a significant reduction in response time to the second phase of the simple reaction time test, the second phase of the choice reaction time test and the baseline stroop test between test trials (see Table 3.4).

Table 3.4: Mean Uncorrected Cognitive Scores

	Jockeys		Controls	
	n = 9		n = 9	
	Baseline	Retrial	Baseline	Retrial
Simple Reaction Time (ms)	356 ± 29	349 ± 59	322 ± 32	316 ± 33
<i>SRT 1 (ms)</i>	326 ± 22	329 ± 39	309 ± 29 [♦]	306 ± 30
<i>SRT 2 (ms)</i>	335 ± 30	334 ± 39	321 ± 27	303 ± 23*
Choice Reaction Time (ms)	490 ± 86	562 ± 218	495 ± 133	410 ± 81
<i>CRT 1 (ms)</i>	440 ± 77	448 ± 70	417 ± 62	395 ± 72
<i>CRT 2 (ms)</i>	470 ± 55	450 ± 68	434 ± 117	412 ± 66*
Stroop Baseline (ms)	927 ± 128	898 ± 78	830 ± 133 [♦]	778 ± 116*
<i>Stroop Interference (ms)</i>	1022 ± 178	1049 ± 155	878 ± 123 ^{♦♦}	840 ± 103
<i>Interference (ms)</i>	95 ± 95	151 ± 144	48 ± 90	62 ± 59
RVIP (ms)	528 ± 109	547 ± 74	425 ± 151	484 ± 89
<i>Hits (n)</i>	15 ± 5	14 ± 6	16 ± 4	18 ± 4
<i>Misses (n)</i>	9 ± 5	10 ± 6	8 ± 4	6 ± 4
<i>False Hits (n)</i>	17 ± 30	11 ± 12	3.2 ± 4.6	2 ± 3

*Data presented as mean ± SD, [♦]p < 0.05 between groups, ^{♦♦}p < 0.01 between groups, *p < 0.05 between trials, **p < 0.01 between trials*

Correction of cognitive scores through the removal of any response time greater than two standard deviations from the mean removed baseline differences between the groups as indicated by the uncorrected scores (see Table 3.4). No additional significant changes were identified following correction of the cognitive data results, which are presented in appendix D.

3.4.5.2. Individual Cognitive Data Values

Individual responses of the jockey group to the simple reaction time test, choice reaction time test, stroop baseline and interference and rapid visual information processing tests and RVIP accurate response rates are presented in figures 3.6 – 3.11. No visual trends toward changes in cognitive response following a four % reduction in body mass were identified for any parameter.

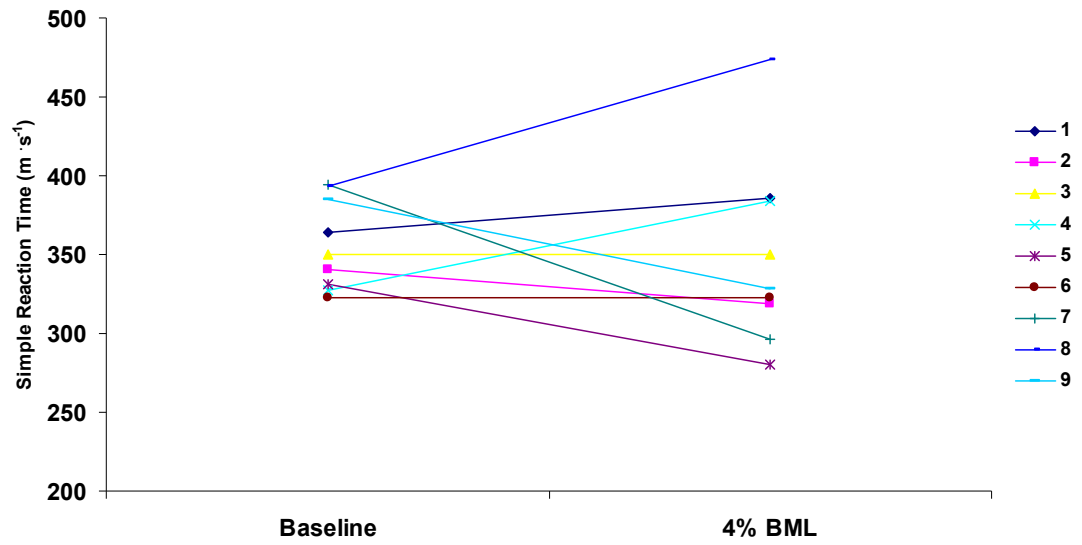


Figure 3.6: Individual Jockey Simple Reaction Time

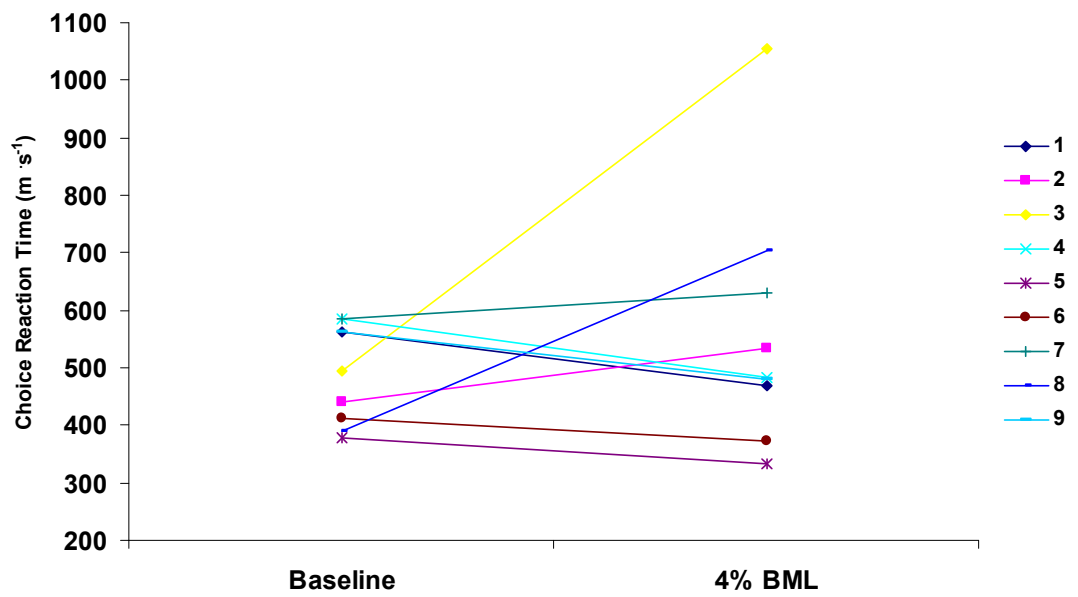


Figure 3.7: Individual Jockey Choice Reaction Time

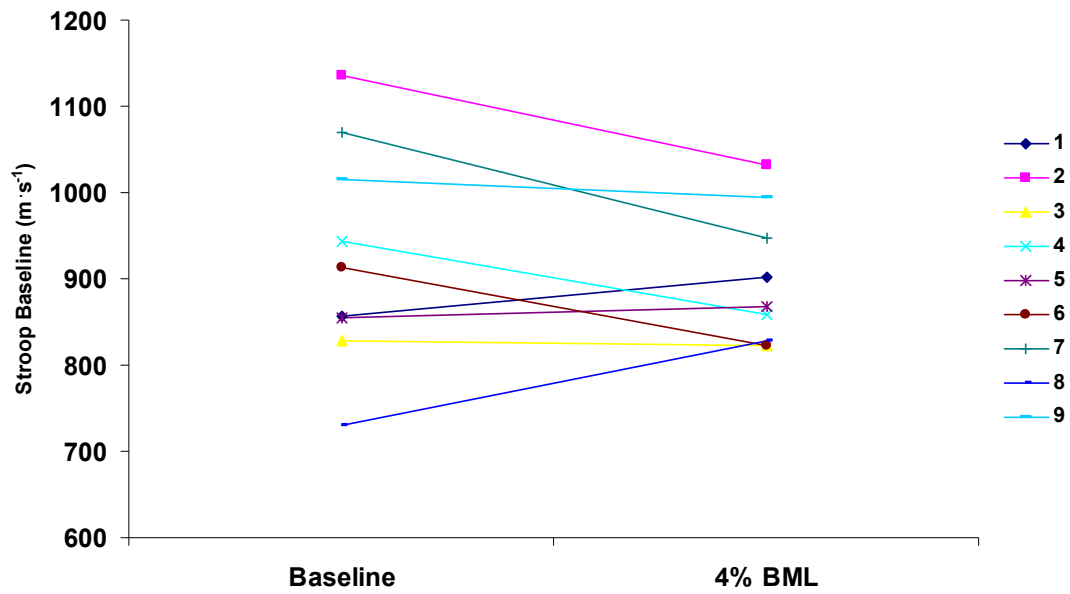


Figure 3.8: Individual Jockey Stroop Baseline Response

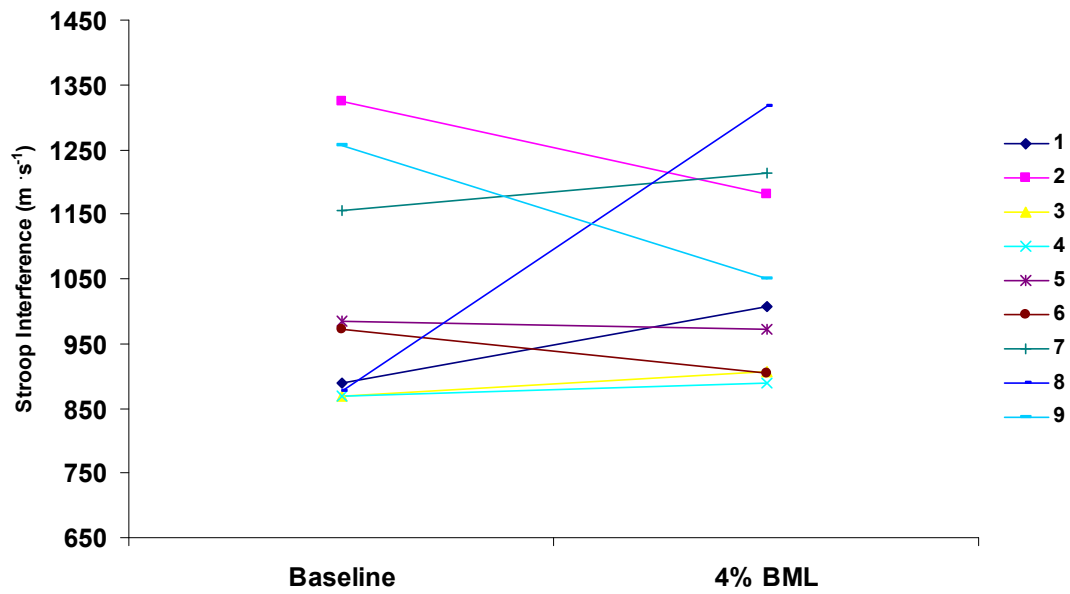


Figure 3.9: Individual Jockey Stroop Interference Response

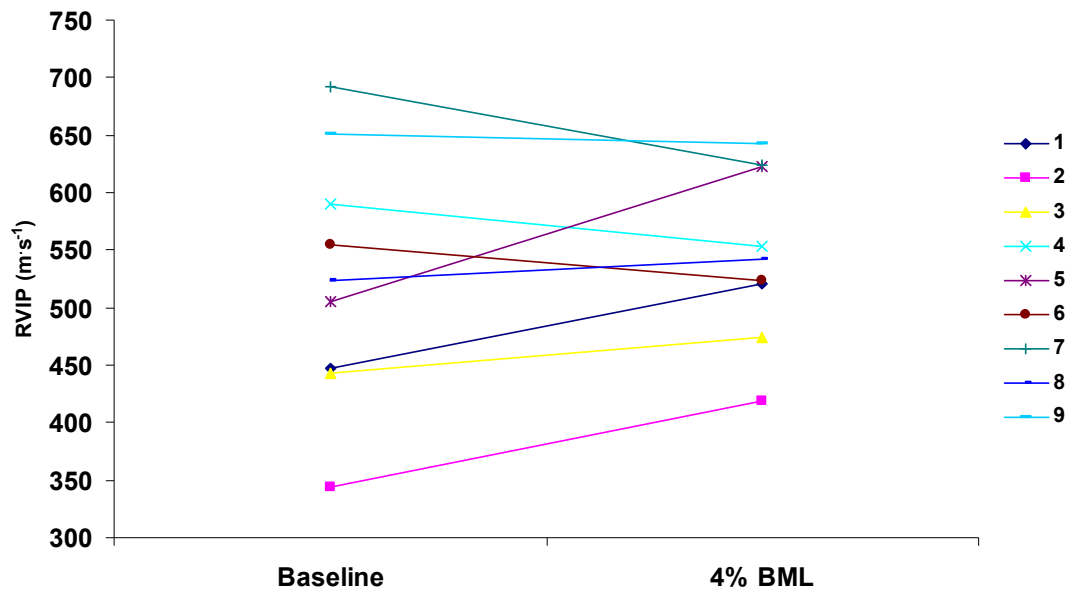


Figure 3.10: Individual Jockey Rapid Visual Information Processing

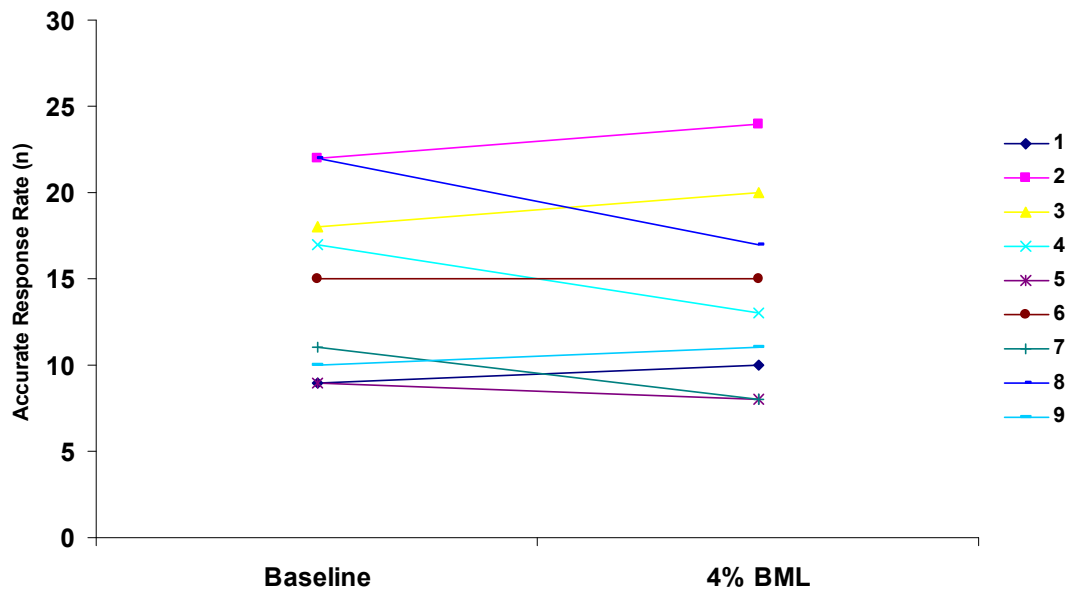


Figure 3.11: Individual Jockey RVIP Accurate Response Rate

3.4.6. Nutritional Information

Estimated baseline resting metabolic rate (RMR) was 1544 ± 74 and 1705 ± 89 kcal·day⁻¹ for jockey and control participants respectively. Retrial RMR values were 1515 ± 75 and 1705 ± 88 kcal·day⁻¹ for jockey and control participants respectively, demonstrating a significant decrease for the jockey group between trials ($p < 0.01$), but no change in the control group.

Percentage breakdown of macronutrient composition was not different between the groups but experimental participants consumed significantly less of all macronutrients in comparison to control participants. In addition total energy intake was significantly reduced in the 24 hours prior to the second trial for the experimental group in comparison to the preceding 24 hours. Mean subject total energy intake was sufficient to support a physical activity level (PAL) of 0.6 ± 0.22 when considered relative to estimated RMR. When broken down into day one and day two intakes were sufficient to support a PAL of 0.95 ± 0.45 and 0.27 ± 0.2 respectively. For a detailed breakdown of macronutrient intakes between test trials see Appendix D.

The jockeys took in significantly less of all measured micronutrients than control participants excluding carotene, biotin and Vitamin E. Mean jockey micronutrient intake did not reach recommended lower threshold intakes (LTI) for most micronutrients including potassium, calcium, copper, zinc, selenium, iodine, riboflavin, folate and vitamin C. All micronutrient data are presented in Appendix D.

3.4.7. Additional Measures Employed to Actively Reduce Body Mass

All participants reported restricted fluid and energy intake as the primary means of body mass reduction ($n = 9/100\%$). Sixty Seven % of participants ($n = 6$) used additional acute means of body mass reduction. Forty four % of participants ($n = 4$) employed active dehydrating methods such as exercising while wearing heavy clothing to induce sweating. Thirty three % of participants ($n = 3$) used passive dehydration mechanisms (sweating induced through the use of saunas or hot baths).

3.5. Discussion

Results of this study appear to indicate that a four % reduction of body mass in 48 hours increased the submaximal cardiovascular strain and reduced work capacity as determined by peak power output (PPO) during a maximal cycle ergometer test in a group of jockeys. In contrast, no such decrements were shown in cognitive performance. Low energy intake during the weight loss period and a significant increase in urine specific gravity between trials suggest that low energy intake and a high degree of dehydration are likely to have had a role to play in the impairment to physiological performance demonstrated in this study.

Results from the self reported weight loss questionnaire suggests that typical riding body mass is significantly lower than that which the participants indicated as their everyday non-riding body mass (2.2 ± 1.4 kg, $p < 0.01$), reflecting a decrease of $3.6 \pm 2.4\%$ from self reported non riding body mass ($p < 0.01$). Although moderate restrictions in energy intake have been shown to induce body mass loss without an associated performance decrement (Zachwieja et al., 2001), body mass loss of the magnitude demonstrated here, and within the short time ranges reported (34 ± 13 hours) is likely to comprise body water, muscle glycogen stores and possibly lean muscle mass (Umeda et al., 2004) all of which may convey a negative impact on athletic performance (Burge et al., 1993; Filaire et al., 2001). The magnitude of weight loss selected for this study was based on the findings of this questionnaire. The jockey group were required to reduce body mass by four % in the 48 hours between trials adopting those methods that were “typically used to make weight for racing”. It was thought that this protocol would most closely represent the challenges typically encountered by this population.

Participants reduced body mass by 2.1 ± 0.5 kg within the forty eight hour time frame allocated, representing a 3.6 ± 0.9 % decrease from baseline. It appears that dehydration induced decrements to both physiological (Baker et al., 2007; Casa et al., 2000; Filaire et al., 2001; Sawka et al., 2007) and cognitive (Cian et al., 2001; Gopinathan et al., 1988; Tomporowski et al., 2007) function may occur at a threshold of 2% body mass loss and so it was hypothesized that the protocol used here may

induce impairments to both physiological and cognitive function. This hypothesis was accepted for physiological function, but rejected for cognitive function.

No changes in subcutaneous body fat, as estimated by skinfold measurement, were reported between the trials. Furthermore, results from the hydration and nutritional analysis suggest that body mass losses in the jockey group, may have been predominantly derived from body water and substrate stores. Mean Usg levels between trials showed a significant increase from 1.019 ± 0.004 to 1.028 ± 0.005 ($p < 0.01$). A Usg of 1.020 has previously been suggested as being indicative of dehydration (Armstrong et al., 2005; Oppliger & Bartok, 2002; Sawka et al., 2007) and to represent a body mass loss of approximately 2% (Casa et al., 2000). The high levels of dehydration reported here following a rapid reduction in body mass are consistent with those previously reported in a group of flat jockeys on a competitive race day (Warrington et al., 2009).

Results of the nutritional analysis revealed an extremely low caloric intake in the jockey group during the period of body mass reduction, with severe deficiencies noted in virtually all major macro and micro nutrients. Reported energy intake in the weight loss group over the two days was found to be insufficient to support even the most basic of metabolic processes (Mifflin et al., 1990) let alone match the daily energy demands of an active jockey preparing for competition. Analysis of the breakdown of nutritional intake revealed a mean intake of just 422 ± 315 kcal ($0.27 \pm 0.2\%$ of estimated RMR) in the 24 hours prior to weigh-in, with 20% of subjects ($n = 2$) abstaining absolutely from all food and drink. In addition 67% of participants ($n = 6$) used additional weight loss techniques, which focused primarily around active and passive dehydration. Such methods of rapid weight loss are in agreement with those previously reported as regularly used by jockeys (Dolan et al., 2010; Labadarios, 1988; Leydon & Wall, 2002; Moore et al., 2002).

Physiological function and work capacity were shown to be impaired by the reduction of body mass observed in the weight loss group, as was evidenced by a decrease in peak power output (PPO) in the second trial ($p < 0.01$). Examination of the cycle ergometer test data showed a higher absolute VO_2 ($\text{L} \cdot \text{min}^{-1}$) and workload (watts) in the control group. When these data were expressed relative to body mass (VO_2 ,

ml·kg⁻¹·min⁻¹ and watts·kg⁻¹) differences between the groups were shown to be non-significant, demonstrating similar relative aerobic and power capacities between the groups. No significant change for any resting respiratory, heart rate or lactate measure were shown between trials for either the jockey or control group (see Table 3.3) although RER did show a trend toward decrease following the four % reduction in body mass in the jockey group ($p = 0.076$). Decreased RER when in a dehydrated state has previously been demonstrated (Armstrong et al., 2006; Cheung & McLellan, 1998) indicating a potential alteration to substrate metabolism and a shift toward increased lipid oxidation. No further changes in RER were demonstrated throughout the test however.

Despite the significant decrease in PPO, no changes were demonstrated for any of the peak physiological variables studied, in agreement with previous research (Cheung & McLellan, 1998; Ebert et al., 2007; Moquin & Mazzeo, 2000). A trend toward increased peak heart rate in the retrial was reported ($p = 0.061$), showing that a similar or greater magnitude of physiological strain was experienced in both trials, despite the reduction in max power output. Reduced body mass alone may contribute to decreased power output, however a significant decrease in relative peak workload achieved (watts·kg⁻¹, $p < 0.01$) indicates that additional influences were present which affected performance in this study. It is likely that both dehydration and low energy intake had a role to play in the impairment to aerobic function demonstrated which is in accordance with previous research (Casa et al., 2000; Cheung & McLellan, 1998; Coyle, 2004; Ebert et al., 2007; Oliver et al., 2007; Sawka et al., 2007).

Examination of submaximal physiological data revealed a significant increase in both oxygen uptake (ml·kg⁻¹·min⁻¹) and heart rate response at set workloads demonstrating an increase in the submaximal physiological strain experienced by the jockeys following a four % reduction in body mass. In addition lactate accumulation appeared to be elevated as evidenced by a mean increase at 90, 120 and 150 watts, which showed a trend toward significance at 120 and 150 watts ($p = 0.063$ and 0.060) respectively. A large variability in retrial lactate response may have affected the statistical power of the tests used. Differences in physical capabilities and in response to exercise following rapid weight loss may in part explain the wide variability in lactate response shown here. In addition observational evidence indicated a rapid increase in lactate

accumulation at quite low exercise intensities in a number of jockeys. Further research may be required so to further investigate lactate response following weight loss in jockeys. An increase in cardiovascular and physiological strain when exercising in a dehydrated state is well documented in the literature (Armstrong et al., 2006; Ebert et al., 2007; Moquin & Mazzeo, 2000). A number of postulated mechanisms have been proposed to explain the decline in aerobic exercise capacity when exercising in a dehydrated state, including a reduction in plasma volume limiting blood supply to the working muscles (Coyle, 2004) or an anticipatory reduction in muscle force production in response to increased core body temperature (Tucker et al., 2004) which is a reported consequence of exercising in a dehydrated state (Ebert et al., 2007; Morris et al., 2005; Watson et al., 2005). The precise mechanisms of dehydration induced impairments to aerobic exercise performance remain to be determined.

Cognitive function was not shown to be affected in this study. Previous research has suggested that many aspects of cognitive and psychological function are affected by dehydration (Cian et al., 2001; Ritz & Berrut, 2005; Tomporowski et al., 2007) and that this may occur at similar thresholds to physiological impairments (2%) (Gopinathan et al., 1988). In particular it appears that more complex cognitive processes, involving multiple aspects of the cognitive domain may be more consistently affected by dehydration (Wilson & Morley, 2003) and so it was expected that performance on the stroop and RVIP tests in particular may have been impaired following the weight reduction protocol used here. Aspects of cognitive function, including the ability to memorise, recall, draw a design, identify a randomly scattered number or combined number and letter sequence and accuracy of task performance, reaction and response time have been shown to be affected in a group of jockeys on a competitive race day in comparison to a non-race day (Labadarios, 1988). The decline in performance was related to the amount of weight lost in preparation for the race meeting. Short term memory and mood state has been shown to be negatively affected by a 5% loss of body mass for competition in a group of competitive wrestlers (Choma et al., 1998). No such decrements to cognitive performance were observed in the current study. This would appear to suggest that speed of motor response; decision making; executive function and working memory were maintained following the four % reduction of body mass in this group of jockeys.

A number of possible explanations are available which may have contributed to this finding. In particular, a major limitation of many of the available studies involving dehydration and cognitive function is that the methods used to induce dehydration, namely heat and exercise (Cian et al., 2001; Gopinathan et al., 1988; Tomporowski et al., 2007) may be considered as individual physiological stressors, making isolation of the specific effects of dehydration difficult (Grandjean & Grandjean, 2007). Cognitive testing in this study was conducted prior to the exercise test, in an attempt to control for the confounding influence of exercise on cognitive performance. That no differences in cognitive performance were identified in this study is in agreement with the work of Szinnai et al., (2005) who showed no change to cognitive function following dehydration of 2.6% induced by 24 hours of water deprivation. In addition, cognitive function in the study by Cian et al., (2001) was shown to be normalised 3.5 hours after dehydration induced through either passive exposure to heat or treadmill exercise, despite the continued level of hypo-hydration experienced. Results from these studies suggest that dehydration *per se* may not be the primary limiting factor in cognitive performance, but that the methods used to induce dehydration may contribute to observed decrements. Results of these studies (Cian et al., 2001; Szinnai et al., 2005) appear to support the lack of change in cognitive function reported in the present study.

Control participants showed an improved performance on a number of cognitive function tests in the second trial (see Table 3.4) which may indicate an insufficient habituation protocol. Statistical analysis of the third habituation trial and baseline cognitive test data revealed no significant differences between tests however. Further research may be required so to more fully evaluate the effects of a rapid reduction in body mass on cognitive performance as results from the present study and surrounding literature appear to be quite contradictory.

3.6. Limitations

A number of limitations are present within this study which may have affected interpretation of results. Food diaries, while practical for small group samples (Bingham et al., 1985) can be inaccurate and participants may under-report or misinterpret intake (Black, 2000, Deakin, 2006). Although physiological function was clearly shown to be impaired following body mass loss it is not clear whether this attenuation of physiological capacity will translate into actual performance decrements, as the extent of the contribution of the aerobic energy system to racing performance remains to be identified. In addition, it is likely that both dehydration and low energy intake had a role to play in the impairments to physiological function and work capacity observed. The study design employed did not however allow examination of the independent influences of these parameters and so it is unknown whether dehydration or low energy intake had a predominant effect. Changes in control cognitive response between test trials suggest that the habituation protocol used may have been somewhat ineffective so affecting interpretation of results. Due to logistical and methodological issues, the sample size in this study was small, which may have affected identification and interpretation of subtle changes in physiological and cognitive function.

3.7. Conclusion

Results from this study clearly demonstrate an impairment to aerobic exercise performance following a four % reduction in body mass in 48 hours as evidenced by a decrease in peak power output and increased submaximal cardiovascular strain experienced during an incremental cycle ergometer test to volitional fatigue. In contrast, no such differences were observed for cognitive function. The rapid weight regulation practices typically adopted by the jockeys, which focus predominantly on dehydration and a low energy intake, are the likely cause of much of this impairment to physiological function although precise mechanisms remain to be identified. Impaired physiological function is likely to impact on racing performance and so rapid reductions in body mass should be avoided if possible prior to competition.

Chapter Four

Study Two: A Comparison of Bone Mass between Professional Jockeys, Elite Amateur Boxers and Age, Sex and BMI Matched Controls



4.1. Abstract

Aim: The aim of this study was to compare bone mass between two groups of professional jockeys (flat: n=14; national hunt: n=17), other weight category athletes (elite amateur boxers: n=14) and a group of age, sex and BMI matched controls (n=15).

Methods: All subjects underwent DXA scanning for assessment of bone mass. Measurements were made of the total body, lumbar spine and femoral neck. Body composition and the relative contribution of fat and lean mass were extrapolated from the results of the total body scan. All data were analysed in accordance with differences in body size and composition, in particular height, lean mass, fat mass and age. **Results:** Both groups of jockeys displayed significantly lower bone mass than either the boxers or control groups for a number of variables measured including total body BMD, total body BMC (minus the head), L2 – 4 BMD and femoral neck BMD and BMC. Adjustment of bone data revealed that differences in body size and composition in particular height and lean mass, may partly explain differences identified between the groups, but that additional factors were present which may have influenced bone mass in the different groups. **Conclusion:** Bone mass appears to be reduced in both jockey groups which may have particular implications for these athletes in light of the high risk nature of the sport. The high intensity, high impact training associated with amateur boxing appeared to convey an osteogenic stimulus on these weight category athletes. While differences in body composition between the groups had a key role to play in these findings, additional factors appear to be present which contributed to the differences in bone mass reported. It is suggested that gravitational, nutritional and hormonal factors may in part explain a portion of the variance demonstrated here.

4.2. Introduction

The original research, from which this project stemmed, appeared to indicate that jockeys have lower bone mass than would be expected for males of this age (Warrington et al., 2009). A number of potential mechanisms to explain this finding were proposed, including insufficient dietary and nutritional intake (Dolan et al., 2010), a lack of a strong osteogenic stimulus associated with participation in horse-racing (Alfredson et al., 1998; Cullen et al., 2009) and the low body mass associated with this population (Warrington et al., 2009). The finding of low bone mass in this group was however based on World Health Organisation T scores, which were formulated based on Caucasian women over 50 years of age and so may have limited application to a younger male athletic group (ISCD Writing Group, 2004a). In addition bone mineral density as indicated by DXA scanning is unable to fully account for differences in body size, with a tendency toward under and over estimation in those of smaller and larger stature respectively (Molgaard et al., 1997; Prentice et al., 1994). This occurs due to an inherent technical inability to measure bone depth and therefore true volumetric bone density. While the study by Warrington et al., (2009) provided some very interesting data, further research was required so to assess whether bone mass is actually reduced in this group, and if so to more fully elucidate the potential mechanisms involved.

Jockeys and amateur boxers are examples of weight category athletes and both must “weigh-in” at a designated body mass in order to compete. In theory these athletes may face similar issues regarding weight control. In practice however the challenges which they encounter are quite different. The main difference between horse-racing and the majority of other weight category sports such as boxing is that while weight loss occurs in all cases, boxers are required to weigh-in prior to competition only. This weigh-in may take place up to 12 hours before the competition, thereby allowing the athlete time to replenish energy and fluid stores depleted when making weight (Slater et al., 2007; Slater et al., 2006). This opportunity is not afforded to jockeys who are required to weigh-in immediately before and after each race that they ride. The situation is further compounded by the virtue that jockeys race at weight throughout the week, and in many cases over a 10-12 month period. In contrast, boxers appear to have a defined competitive season which may last approximately four - six months and

contains a small number of major competitions so allowing these athletes to regain some body mass between competitions and in the off-season. In addition boxers compete in a set weight category allowing a certain amount of structure be applied to the achieving and maintenance of the required body mass. Jockeys are required to align their weight with the mount which they are riding in each individual race, which may be as many as five to seven races per day. The large variability and lack of predictability related to the specific weight targets which jockeys must meet can result in rapid weight loss and chronic weight cycling which may increase the physiological and metabolic strain placed on this athletic population.

Bone is a living dynamic tissue which has a number of structural and metabolic functions to fulfil within the body. It has been suggested that the severe energy restrictions which accompany acute weight loss may have consequences for the bone health of the individual (Proteau et al., 2006; Walberg Rankin, 2006). This may be due to calorific and micronutrient deficiencies and also the hormonal readjustments which may occur as a result of energy deficiencies and increased stress on the body (Bass et al., 2005; Walberg Rankin, 2006; Zanker et al., 2000).

Given the preliminary data which suggest that bone mass in jockeys may be reduced (Warrington et al., 2009), the aim of this study was to establish whether true differences exist by comparing bone mass in a group of flat and national hunt jockeys with another group of weight category athletes (elite amateur boxers) and age, gender and BMI matched controls.

4.3. Methods

Aims and Objectives:

The aim of this study was to assess differences in bone mass and body composition in two different types of weight category athletes (professional jockeys and elite amateur boxers) and a group of age, gender and BMI matched controls.

Objective One:

To compare bone mineral density (BMD), bone mineral concentration (BMC) and bone area (BA) of the four groups as indicated by DXA scanning.

Objective Two:

To normalise bone mass results to reflect the size and stature of the groups so to identify whether differences exist independently of variations in body composition.

Hypothesis:

That differences in bone mass may exist between the groups, with bone mass being shown to be reduced in the two jockey groups and enhanced in the boxer group and that these findings may exist independently of variations in body stature and composition.

4.3.1. Research Design Overview

The aim of this study was to assess differences in bone mass and body composition in two different types of weight category athletes (jockeys and boxers) and a group of age, sex and BMI matched controls. All subjects received a total body; lumbar spine and proximal hip DXA scan for assessment of bone mineral concentration, bone area, fat and lean mass. All data was adjusted to account for differences in body size and composition. Ethical approval for this study was granted by the Dublin City University Research Ethics Committee. All participants provided written informed consent and medical history prior to participation in this study. Anyone with a reported medical condition known to affect bone health was excluded from this study.

4.3.2. Participants

Sixty male participants were recruited to take part in this study. These included 31 professional jockeys (14 flat and 17 national hunt); 14 elite amateur boxers and 15 controls. Boxers were recruited from those individuals who compete in weight categories corresponding to the weight ranges within which jockeys compete. All participants were matched for age, gender and BMI. In addition the national hunt jockeys, boxers and controls were matched for body mass. It was not possible to match body mass of all groups as flat jockeys compete in a distinctly different weight range than national hunt riders and so are required to maintain a lower body mass than their national hunt counterparts. The weight classifications for each group can be summarised as follows:

Flat Jockeys (FJ)

Jockeys who compete in races of 5 – 20 furlongs (1 furlong = 0.201 km) and consist of a run with no obstacles. Weight allocations for flat jockeys range from 52.7 – 64 kg.

National Hunt Jockeys (NH)

NH races are at least 3.2 km long, throughout which the horse must jump a number of fences or hurdles. Weight allocations for national hunt jockeys range from 62 – 76kg.

Boxers:

Amateur boxing is a high intensity combat sport contested between two people who fight with their fists for three 3-minute rounds separated by 60 seconds recovery. The winner is determined either by a scoring system based on the number of scoring punches or by “knock out”. Amateur boxers currently compete in 11 different Olympic weight classes ranging from 48.2 kg to over 91.4 kg in the superheavyweight classification. All subjects in the boxing group were members of the Irish Amateur National Squad.

Controls

Recreationally physically active age and BMI matched males were recruited to act as a control group in this study.

4.3.3. Dual Energy X-Ray Absorptiometry (DXA)

Bone mass was determined by dual energy x-ray absorptiometry (DXA) scanning using the GE Lunar Prodigy Advance Scanner (GE Medical Systems, UK). Scans were performed in order to measure bone mass of the total body, lumbar spine (vertebrae L2 - 4) and femoral neck. Positioning for all scans was completed in accordance with manufacturer instructions. For the total body scan participants were centred and squared in the centre of the table with ankles and knees taped together and scanner laser light positioned approximately 3cm above the head. Lumbar spine scans were taken from L5 – T12. Legs were positioned upright on the provided foam block to lessen curvature of the spine. Scanner laser lights were positioned midway between the iliac crest and ASIS along the midline of the body. Femoral neck was measured after placing the hips in an internally rotated position and positioning the laser light five - six cm below the greater trochanter along the mid line of the thigh. Bone mineral density (BMD) scores were reported as grams of absolute bone mineral content (BMC) per cm² of projected bone area (BA). The relative contributions of fat and lean mass were extrapolated from the results of the total body scan.



Illustration 4.1. GE Lunar Prodigy Advance DXA Scanner

Bone mineral apparent density (BMAD) was calculated so to provide an estimation of volumetric bone density, using previously described equations (Boot et al., 2009; Kroger et al., 1992).

$$L2 - 4 \text{ BMAD} = ((BMD^2)/BMC) * (4\pi^{-1} * width)$$

$$Femoral Neck \text{ BMAD} = ((BMD^2)/BMC) * (4k\pi^{-1})$$

(where k = 1.5cm, i.e. the fixed length along the femoral neck)

4.3.4. Statistical Analysis

Data were analysed using SPSS for windows version 17.0. Distribution of data was assessed through use of the Shapiro Wilks test. Any variable which did not meet parametric assumptions was log transformed so to normalise data distribution. One way independent samples ANOVA was used to identify differences between the groups for all parameters. General linear modelling using univariate analysis of covariance (ANCOVA) was used to identify significant covariates of total body, lumbar spine (L2 – 4) and femoral neck BMC and BA, with lean mass, height, age and fat mass included as potential covariates in each case. All variables were log transformed for this analysis. Identified variables were entered stepwise into a linear regression model and prediction equations were elucidated from the results. “Dummy variables”, whereby a standard value was applied to each group, were formulated in order to represent interaction effects and to identify group specific effects. Bone mass data were assessed in order to allow normalisation for potential confounding variables which DXA scanning is unable to account for, as previously described (Prentice et al., 1994). All data were transformed to their natural log and linear regression was used whereby bone area, height and body mass were simultaneously regressed onto total body BMC so to assess the relative contribution of each as previously described (Molgaard et al., 1997; Prentice et al., 1994). Height was then individually regressed onto BMC and BA and the resultant regression equation was used as the appropriate power coefficient by which to adjust stature in each case.

4.4. Results

4.4.1. Exploratory Data Analysis

Tests of normality indicated that the majority of variables met the assumptions of normal distribution. A number of variables however did not, including age, FMI, LMI, fat mass, L2 – 4 BMD and L2 – 4 BMC. These variables were therefore log transformed, which rendered them suitable for parametric testing as indicated by the Shapiro Wilks test. All data are presented in their raw state. Outliers were identified and examined using normality plots of distribution. One subject from each of the national hunt group and control group represented significant outliers for all bone mass variables and so were excluded from the data set (n = 58: 14 flat; 16 national hunt; 14 boxer and 14 control). All data were entered into the univariate analysis of variance in its log transformed state and power calculations were elucidated from the results.

4.4.2. Descriptive Data

Descriptive and anthropometric characteristics of the four groups are presented in Table 4.1. All groups were age, gender and BMI matched, with no significant differences apparent. Differences were shown between the groups for a number of other indices of body composition as illustrated in Table 4.1. Both boxers and controls had significantly higher lean mass than the flat jockey group. No differences were shown between the groups in relation to lean mass expressed relative to height ($\text{kg}\cdot\text{m}^{-2}$). The national hunt and control groups had a greater fat mass, FMI and % body fat than either the flat jockey or boxer group.

Table 4.1: Descriptive and Anthropometric Data:

	Flat (n=14)	National Hunt (n=17)	Boxers (n=14)	Control (n=15)
Age (yrs)	25 ± 7	25 ± 4	21 ± 2	23 ± 4
Height (m)	1.65 ± 0.06	1.72 ± 0.05*	1.74 ± 0.1*	1.79 ± 0.045* [†]
Body Mass (kg)	54.63 ± 3.6	64.3 ± 3.34*	65.3 ± 12.2*	69.18 ± 4.98*
BMI (kg·m⁻²)	20.18 ± 1.6	21.92 ± 1.2	21.56 ± 2.27	21.7 ± 1.88
Lean Mass (kg)	49.39 ± 3.8	53.74 ± 4.34	58.06 ± 8.3*	58.03 ± 5.12*
LMI (kg·m⁻²)	18.19 ± 1.3	18.25 ± 1.23	19.22 ± 1.6	18.17 ± 1.4
Fat Mass (kg)	4.43 ± 1.5	8.67 ± 3.9*	6.66 ± 4.1	9.26 ± 3.84*
FMI (kg·m⁻²)	1.65 ± 0.62	2.97 ± 1.46*	2.14 ± 1.07	2.94 ± 1.32*
Body Fat (%)	8.3 ± 2.9	13.8 ± 6.02*	9.8 ± 4.14	13.7 ± 5.06*

*Data presented as mean ± SD, * $p < 0.05$ from Flat; [†] $p < 0.05$ from National Hunt*

4.4.3. Bone Mass Characteristics

Bone mass characteristics of all groups are presented in Table 4.2. Groups consistently followed the same pattern for each variable measured, with flat jockeys displaying the lowest measure, followed by national hunt and control subjects, while the boxer group consistently displayed the greatest amount of bone. These differences reached significance between the groups for a number of variables, with the boxer and control group displaying significantly higher bone content than either of the two jockey groups in relation to total body BMD, total body BMC (minus the head), L2 – 4 BMD and femoral neck BMD and BMC (see Table 4.2). A tendency toward significance was shown between national hunt and control group for L2 – 4 BMC and L2 – 4 BA ($p = 0.07$ and 0.054 respectively).

Table 4.2: Bone Mass Characteristics

	Flat (n=14)	National Hunt (n=17)	Boxer (n=14)	Control (n=14)
TB BMD (g·cm⁻²)	1.09 ± 0.06	1.17 ± 0.05*	1.29 ± 0.1* [♦]	1.26 ± 0.06* [♦]
TB BMC (g)	2373 ± 255	2791 ± 226*	3268 ± 612* [♦]	3128 ± 327*
TB BMC (g) <i>(minus head)</i>	1941 ± 227	2314 ± 208*	2751 ± 560* [♦]	2649 ± 277* [♦]
TB BA (cm²)	2172 ± 163	2399 ± 147*	2516 ± 299*	2502 ± 147*
TB BA (cm²) <i>(minus head)</i>	1950 ± 161	2167 ± 145*	2285 ± 295*	2266 ± 142*
L2 – 4 BMD (g·cm⁻²)	1.10 ± 0.09	1.15 ± 0.1	1.48 ± 0.16* ^{♦†}	1.26 ± 0.14*
L2 – 4 BMC (g)	47.28 ± 6.9	51.76 ± 9	74.35 ± 15.1* [♦]	63.66 ± 11.24* [♦]
L2 – 4 BA (cm²)	42.72 ± 4.2	44.89 ± 5.3	49.84 ± 6.7*	50.13 ± 4.4* [♦]
L2 – 4 BMAD (g·cm⁻³)	0.14 ± 0.01	0.14 ± 0.01	0.18 ± 0.02* ^{♦†}	0.143 ± 0.014
FN BMD (g·cm⁻²)	1.05 ± 0.07	1.07 ± 0.11	1.25 ± 0.11* [♦]	1.19 ± 0.15* [♦]
FN BMC (g)	5.4 ± 0.55	5.76 ± 0.72	6.85 ± 0.82* [♦]	6.55 ± 0.7* [♦]
FN BA (cm²)	5.15 ± 0.4	5.39 ± 0.35	5.46 ± 0.32	5.5 ± 0.35
FN BMAD (g·cm⁻³)	0.39 ± 0.04	0.38 ± 0.05	0.44 ± 0.04 [♦]	0.42 ± 0.07

*Data presented as mean ± SD, * p < 0.05 from Flat; [♦] p < 0.05 from National Hunt; [†] p < 0.05 from Control*

4.4.4: Predictors of Bone Mass

General linear modelling was used to identify significant covariates of each of the bone mass variables ($p < 0.05$) (see Table 4.3). Lean mass (kg), height (m) and an interaction effect between lean mass and group were most consistently identified as covariates to the different bone mass variables.

Table 4.3: Significant Covariates of Bone Mass ($p < 0.05$)

Covariates	
Total Body BMC (g)	Lean mass (kg); height (m); group*lean mass (kg)
Total Body BA (cm ²)	Lean mass (kg); height (m); fat mass (kg); group*lean mass
L2 – 4 BMC (g)	Height (m); group*lean mass (kg)
L2 – 4 BA (cm ²)	Height (m); group*lean mass (kg)
FN BMC (g)	Lean mass (kg); age (yrs)
FN BA (cm ²)	Lean mass (kg)

All significant covariates were then entered into a stepwise linear regression model and prediction equations and R^2 values for each variable were calculated. These were:

Total Body Bone Mineral Content (g):

$$TBBMC = 4.763 + (LBM^{0.634}) - (LBMFlat^{0.024}) + (Ht^{1.204}) + (LBMBoxer^{0.020})$$

($R^2 = 0.814$)

Total Body Bone Area (cm²):

$$TBBA = 5.460 + (LBM^{0.425}) + (Ht^{1.044}) + (FM^{0.035}) - (LBMControl^{0.010})$$

($R^2 = 0.914$)

L2 – 4 Bone Mineral Content (g):

$$L2 - 4BMC = 2.239 + (Ht^{3.212}) + (LBMBoxer^{0.066})$$

($R^2 = 0.692$)

L2 – 4 Bone Area (cm²):

$$L2 - 4BA = 2.239 + (Ht^{3.212}) + (LBMBoxer^{0.066})$$

($R^2 = 0.692$)

Femoral Neck Bone Mineral Content (g):

$$FNBMC = -0.669 + (LBM^{0.816}) - (Age^{0.249})$$

(R² = 0.562)

Femoral Neck Bone Area (cm²):

$$FNBA = 0.284 + (LBM^{0.349})$$

(R² = 0.362)

4.4.5. Adjusted Bone Measures

Total body bone area was the only significant predictor of total body BMC when BA, height and body mass were considered simultaneously. Regression of height onto BMC and BA revealed a power coefficient of three and two respectively. Height adjusted bone mass results are presented in Table 4.4 and showed that the boxer group had significantly more BMC·Ht⁻³ than any other group (see figure 4.1). In addition, the boxer group had significantly more bone area per m² of height than any other group (see Table 4.4). A ratio of BMC: lean mass was calculated for each of the three bone areas scanned. Results are presented in Table 4.4 and show that both the boxer and control groups had significantly higher TBBMC·LM⁻¹ than the flat jockey group, while boxers showed significantly higher L2 – 4 BMC·LM⁻¹ than any other group. No differences were shown between the groups in relation to FNBMC·LM⁻¹.

Table 4.4: Adjusted Bone Measures

	Flat	National Hunt	Boxer	Control
BMC·Ht⁻³ (g·cm⁻³)	531 ± 54	552 ± 29	621 ± 67* [†]	548 ± 58
BA·Ht⁻² (cm·m⁻²)	799 ± 35	815 ± 26	834 ± 38 [†]	784 ± 43
TB BMC·LM⁻¹ (g·kg⁻¹)	48.1 ± 3.5	52.1 ± 4.4	56.0 ± 3.8*	54.0 ± 5.1*
L2–4BMC·LM⁻¹ (g·kg⁻¹)	0.96 ± 0.1	0.97 ± 0.2	1.27 ± 0.2* [†]	1.1 ± 1.5
FN BMC·LM⁻¹ (g·kg⁻¹)	0.109 ± 0.01	0.107 ± 0.01	0.118 ± 0.01	0.113 ± 0.02

*Data presented as mean ± SD, * p < 0.05 from Flat; [†] p < 0.05 from National Hunt; [‡] 0 < 0.05 from Control*

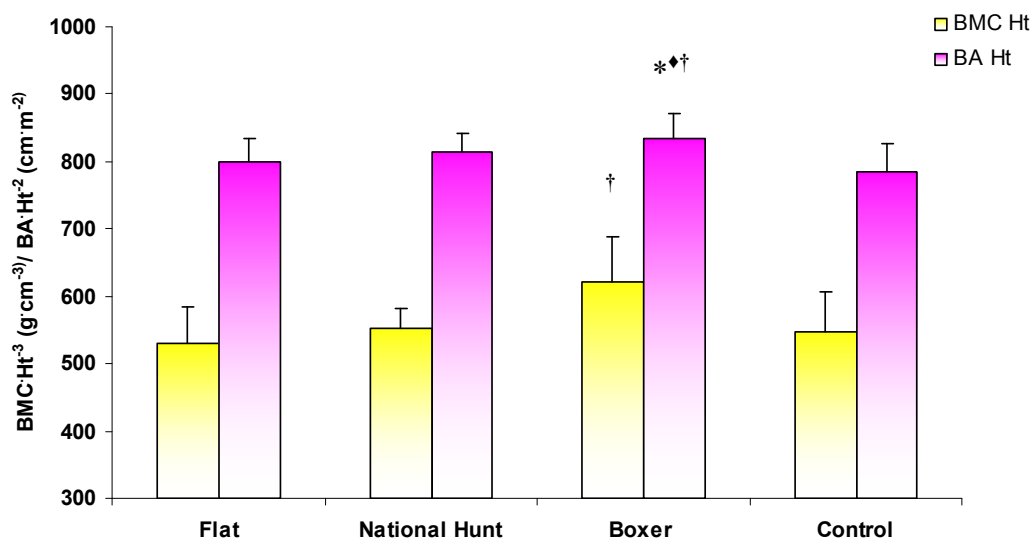


Figure 4.1: Total Body $BMC Ht^3$ and Total Body $BA Ht^2$

Data presented as mean \pm SD, * $p < 0.05$ from Flat; ♦ $p < 0.05$ from National Hunt; † $p < 0.05$ from Control

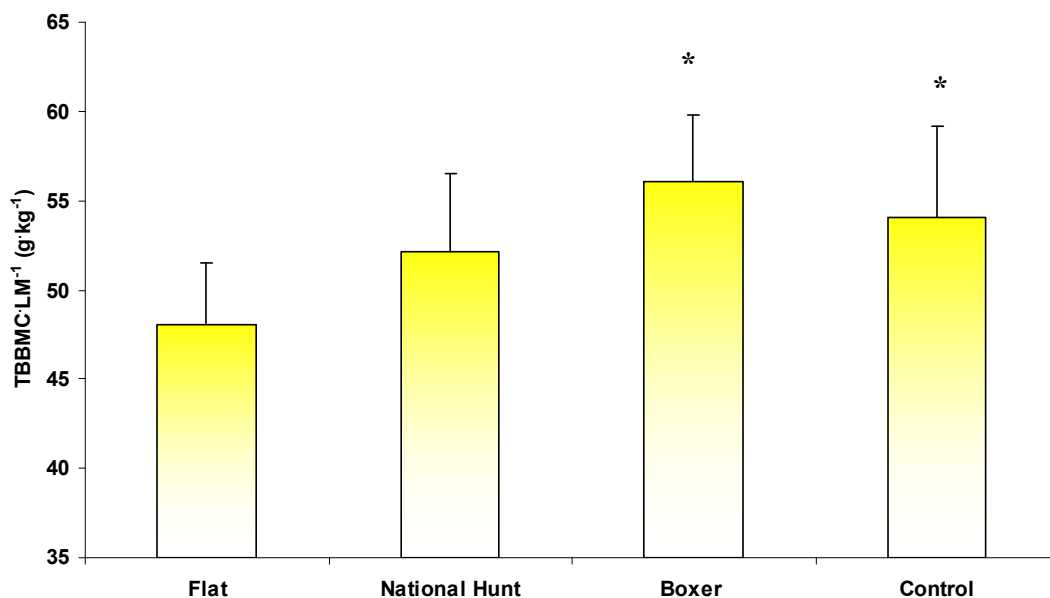


Figure 4.2: Total Body $BMC Lean Mass^{-1}$ ($g kg^{-1}$)

Data presented as mean \pm SD, * $p < 0.05$ from Flat.

4.5. Discussion

Participation in weight category sports poses a unique set of challenges to the athlete, the type and extent of which will ultimately depend on the specific demands of the sport and on the individual in question. Previous research reported apparently low bone mass in jockeys (Leydon et al., 2002; Warrington et al., 2009). Results from the present study appear to confirm the finding of low bone mass in jockeys. Differences in bone mass reported between the groups appeared to exist independently of differences in body size, suggesting that additional factors may be present.

Results from this study demonstrate that both jockey groups have lower bone mass than both the age and BMI matched boxer and control groups. These differences reached statistical significance for total body BMD and BMC (minus the head), L2 – 4 BMD and femoral neck BMD and BMC. Flat jockeys appeared to be most affected at all sites (see Table 4.2). Bone mass, as assessed through DXA scanning has previously been identified as the most relevant and predictive independent factor available for identification of fracture risk (Lewiecki et al., 2008; ISCD Writing Group, 2004). The finding of low bone mass in this group and assumed increase in fracture susceptibility (Brown et al., 2002) may have particular implications for jockeys, given the high risk nature of the sport (Hitchens et al., 2009; Waller et al., 2000) and may represent a major health and safety concern to the racing industry.

Although BMD has previously been identified as one of the most relevant and quantifiable determinants of fracture risk available (ISCD Writing Group, 2004) it is important to note that bone mineral density is a two dimensional measure (g cm^{-2}) and so cannot provide a true approximation of volumetric bone density due to its failure to detect bone depth (Prentice et al., 1994). In fact, it has been suggested that the protective effect of high BMD may not actually be due to bone density *per se*, but may be related to the biomechanical advantage which an increased cross sectional area may convey (Center et al., 2004). Estimation of volumetric bone density through the calculation of bone mineral apparent density (BMAD) aims to correct for this factor (Carter et al., 1992) and has been suggested as being less reliant on body size than BMD (Cvitejic & Korsic, 2004). Bone mineral apparent density (BMAD) was estimated in this group according to previously described calculations (Boot et al.,

2009; Kroger et al., 1992). Results revealed that the boxing group had significantly higher L2 – 4 BMAD than any other group, but that both jockey groups were similar to the control group, indicating that actual bone density differences between these groups may in fact be largely dependent on differences in body size.

In order to more fully explore the relationship between DXA derived measures of bone mass and other aspects of body composition, univariate analysis was used for identification of significant covariates of BMC and BA of the total body, lumbar spine and femoral neck. Lean mass, height and an interaction effect between group and lean mass were consistently identified as independent significant predictors of bone mass, particularly in the case of total body BMC. That lean mass consistently emerged as an independently significant predictor of bone mass is unsurprising given that lean mass and the muscular forces which this measure may represent are considered to characterize the extent of mechanical loading, by which bone is regulated (Frost, 2003). The consistent identification of a significant group*lean mass effect suggests that additional factors, independent of the effect of lean mass and height may be present however which have a role to play in explaining the differences in bone mass identified in this study. Stepwise regression, with total body BMC as the independent factor and including all significant covariates revealed that some unidentified factor, along with height and the amount of lean mass present appeared to enhance total body bone mineral content in the boxer group and to reduce BMC of the flat group.

A number of factors are present which influence bone regulation as discussed in chapter two (section 4.3). Nutritional and physical activity habits and their associated impact on hormonal status may be the most relevant modifiable environmental factors to consider in the interpretation of results reported here (Bass et al., 2005). The role of physical activity in the promotion of bone mass accrual is well documented and in particular it would appear that physical activities which place unusual strains on bone in different directions may present the greatest osteogenic stimulus to bone (Andreoli et al., 2001; Calbet et al., 1999; Morel et al., 2001; Wittich et al., 1998). Considering Newton's Second Law of Motion ($\text{force} = \text{mass} \times \text{acceleration}$) it is reasonable to assume that activities which provide acute acceleration and deceleration in different directions may place a larger strain on bone than those which do not. Boxing is primarily a speed and power type sport involving accelerated movement in multiple

directions (Ghosh et al., 1995; Lal Khanna & Manna, 2006) and may be considered typical of the type of sport known to convey an osteogenic benefit, likely accounting for the increased bone mass identified in this group. The high levels of bone mass identified in the boxing group in this study are consistent with that previously reported in groups of competitive boxers (Sabo et al., 1996; Trutschnigg et al., 2008). That an effect independent of lean mass and height was identified as being instrumental in the development of total body BMC in the boxing group is consistent with previous research which suggests that the osteogenic benefits associated with high impact activity may outweigh the benefits associated with lean mass alone (Rector et al., 2009). In contrast, horse riding has been suggested to convey low gravitational forces to jockeys (Cullen et al., 2009) and as such may not provide a strong osteogenic signal to the body.

The flat jockey group were shown to have lower total body bone mineral concentration than the effects of lean mass and height could account for. Recent research examining nutritional and lifestyle habits in a group of Irish jockeys highlighted a number of issues which may have a potentially detrimental impact on bone health (Dolan et al., 2010) and it is thought that these may have had a role to play in the low bone mass identified in this study. In particular low energy availability (Dolan et al., 2010) may have affected bone regulation in this group (Ihle & Loucks, 2004). In addition, low calcium and vitamin D intakes have been reported in jockeys and no participant in this study reported taking in the minimum recommendation of five portions of fruit and vegetables per day, with a mean intake of 0.9 ± 0.8 (range 0 – 2). Inadequate intake of fruit and vegetables may affect bone mass in one of two ways: 1) they are abundant in bone promoting vitamins and minerals, including vitamin K and C, potassium, magnesium and β -carotene, and 2). Fruits and vegetables represent a key neutralizing buffer to the human body due to their high alkaline content (Chen et al., 2004; MacDonald et al., 2004; New et al., 2000).

An interesting finding from this study was that control subjects appeared to have a lower total body cross sectional bone area than either of the two athletic groups once the effects of height, lean mass and fat mass were accounted for. A potential explanation for this finding may be that extended participation in sport caused increased periosteal apposition in both athletic groups, as a greater cross sectional area

may convey a biomechanical advantage to bone breaking strength (Carter et al., 1992). It is speculated however that poor dietary habits and a potential energy imbalance (Dolan et al., 2010) may have caused mineral accrual to lag behind periosteal apposition in the jockey group, so resulting in the low BMD values reported here and elsewhere (Warrington et al., 2009; Leydon & Wall, 2002).

DXA scanning has an inherent technical inability to measure true volumetric bone density and so differences in stature may influence results (Molgaard et al., 1997; Prentice et al., 1994). As previously suggested, regression analysis was used to examine the relative contributions of bone area, height and weight to total body BMC. Results of this analysis showed bone area to be the only significant predictor of TBBMC indicating that BMC expressed relative to bone area (BMD) may be the most relevant measure of bone mass to consider in this group. Given the results of the univariate analysis however, and the known effect of stature on DXA derived bone mass outcomes, BMC and BA were adjusted for height as previously described (Molgaard et al., 1997; Prentice et al., 1994). BMC expressed relative to height showed that the boxer group appeared to have significantly more bone per m^3 of stature than any other group (see Table 4.4). Examination of bone area Ht^{-2} was intended to provide an indication of the relative bone width of each group as previously described (Molgaard et al., 1997). Boxers were shown to have significantly higher $\text{BA} \cdot \text{Ht}^{-2}$ than the control group but were similar to both jockey groups for this variable. Examination of bone mineral content relative to lean mass showed that both the boxer and control group has higher total body $\text{BMC} \cdot \text{lean mass}^{-1}$ than the flat jockeys, while the boxers also displayed higher $\text{L2} - 4 \text{ BMC} \cdot \text{lean mass}^{-1}$ than any other group (see figure 4.2). Results of these adjusted bone mass measures lend support to the prediction equations generated, whereby it appears that many of the differences in bone mass observed between the groups may be accounted for by differences in height and the amount of lean mass present. Additional influences, independent of these factors do however appear to be present.

It has been suggested that weight cycling and the energy restrictions typically associated with weight category sports may place bone health of athletes at risk (Proteau et al., 2006; Walberg Rankin, 2006). Despite this, participation in high impact sports may convey a protective effect on bone mass; despite the negative osteogenic

effects of rapidly reducing body mass. Proteau et al., (2006) demonstrated a bone resorptive state in a group of elite judoists who were actively reducing body mass for competition. Weight regain between competition however, coupled with the high impact nature of judo appeared to be reflected by an overall osteogenic balance favouring bone formation and a high bone mass in comparison to controls, demonstrating the protective effect of high impact activity. This theory is supported by a study which examined a group of female boxers who were shown to have high levels of bone density in comparison to a control group despite displaying low levels of body fat, high energy expenditure and a high incidence of oligomenorrhea (Trutschnigg et al., 2008). These studies support the high bone mass results reported in the boxer group in this study.

4.6. Limitations

There are a number of limitations in this study which may have affected interpretation of results. DXA scanning though widely used provides an incomplete view of actual bone health and strength. Further research involving alternative bone scanning techniques such as quantitative computer tomography (QCT) may be of benefit as this technique provides a cross-sectional image of long bones and may provide a more comprehensive view of bone architecture and strength (Leonard et al., 2004a). Inferences have been made regarding the dietary and physical activity habits of jockeys and boxers and their potential impact on bone health. These variables were not directly measured however and so it is difficult to identify true causes of differences in bone mass. The sample size in this study is relatively small and results are indicative of this group only and may not be representative of these populations as a whole. The prediction equations generated within this study were useful in examination of the key contributors to bone mass in these groups. A larger sample size may be required however so to enhance the predictive ability of such equations.

4.7. Conclusion

The aim of this study was to compare bone mass between two groups of professional jockeys (flat and national hunt), elite amateur boxers and an age, gender and BMI matched control group. Results indicate that bone mass is significantly reduced in both the jockey groups. Given that low BMD represents one of the greatest risk factors for fracture susceptibility (Lewiecki et al., 2008), accompanied by the high risk nature of horse-racing (Hitchens et al., 2009; Turner et al., 2002), this finding may represent a substantial health and safety concern for the horse-racing industry. In contrast, the boxer group appeared to display enhanced bone mass through participation in their sport, which may in part be due to the high degree of mechanical loading associated with participation in this sport. It appears that bone mass in the flat jockey group is most affected, which may reflect the lower weight range assigned to these athletes. Further research may be required so to identify the interaction of genetic and lifestyle factors which may have influenced bone mass findings in these athletes.

Chapter Five

Study Three: An Analysis of Bone Mass, Turnover and Metabolism in Jockeys



5.1. Abstract

Aim: The aim of this study was to examine bone mass, turnover and endocrine function in a group of jockeys in comparison to an age, gender and BMI matched control group. **Methods:** 20 male jockeys (26 ± 3 yrs; 1.7 ± 0.07 m; 61.1 ± 5.4 kg and 21.36 ± 1.8 kg·m⁻²) and 20 age and BMI matched healthy male controls (24 ± 3 yrs; 1.78 ± 0.06 m; 69.5 ± 6.7 kg and 21.99 ± 1.62 kg·m⁻²) took part in this study. Early morning, fasted blood and second void urine samples were taken for determination of biochemical markers of bone turnover and related hormonal activity, including testosterone, sex hormone binding globulin, the gonadotropins and IGF-1. **Results:** Bone mass appeared to be reduced in the jockey group at all sites measured. (Total body BMD: 1.134 ± 0.05 Vs 1.27 ± 0.06 g·cm⁻³ for jockeys and controls respectively, $p < 0.01$). Bone resorptive activity was elevated in the jockey group as indicated by significantly higher urinary NTx/creatinine (76.94 ± 29.52 Vs 55.9 ± 13.9 nmol·mmol⁻¹ for jockeys and controls respectively, $p < 0.01$). SHBG levels were significantly higher in the jockey group (41.21 ± 9.77 Vs 28.24 ± 9.98 nmol·l⁻¹ for jockeys and controls respectively, $p < 0.01$). Univariate and regression analysis showed SHBG and IGF-1 to be independent predictors of bone mass at the total body and femoral neck respectively ($p < 0.05$). **Conclusion:** Low bone mass and an apparent increased rate of bone loss in the jockey group may have implications in terms of fracture risk for this population. Disrupted reproductive hormone function has been suggested as occurring in times of energy deficiency in an attempt to conserve available energy for more immediately essential processes. It is therefore proposed that the weight restrictive lifestyle apparently followed by jockeys may have impacted on reproductive hormone and anabolic processes in this group, leading to a reduction in bone mass. These findings may have important health and safety implications for jockeys and the horse-racing industry as a whole.

5.2. Introduction

Results from study two of this thesis (chapter four) and previous research (Warrington et al., 2009; Leydon et al., 2002) suggests that jockeys may have low bone mass as a result of participation in this weight regulation sport, a finding which may have serious consequences for this group in light of the high risk nature of the sport (Hitchens et al., 2009; Turner et al., 2002; McCrory et al., 2006). While some of the variance in bone mass identified may be accounted for by differences in lean mass and height, additional influences appear to be present which may affect bone regulation in this group. In particular it was proposed that the physiological stress associated with a chronically weight restricted lifestyle (Dolan et al., 2010) may have had a potential effect on osteogenic function in this group (Warrington et al., 2009).

The primary role of the endocrine system is to coordinate function between all other systems, enabling maintenance of homeostasis and aiding in the response to external stimuli. The endocrine system may be placed under considerable pressure by the physiological stress caused by insufficient energy intake (Bergendahl et al., 1996; Haspolat et al., 2007; Strauss et al., 1985). In particular, processes related to growth and reproduction may be disrupted in times of energy deficiency, in an attempt to preserve available energy for more immediate and essential processes (De Souza & Williams, 2004; Wade et al., 1996). As a result, action of key reproductive and anabolic hormones, including testosterone and growth hormone (GH), may be disrupted in times of energy deficiency (Misra et al., 2003b). In contrast, release of hormones involved in the body's response to stress and anxiety such as cortisol may be increased (Bergendahl et al., 1996).

Disrupted endocrine function in times of energy deficiency may have an adverse effect on bone mass and regulation, and bone turnover has been suggested as being adversely affected at a threshold of energy availability below $30 \text{ kcal} \cdot \text{kgLBM} \cdot \text{day}^{-1}$ (Ihle & Loucks, 2004). In addition, factors such as testosterone (Vandershuerren et al., 2003; Venken et al., 2008), cortisol (Di Somma et al., 2002; Weinstein et al., 1998) and growth hormone/insulin-like growth factor-1 (Bex & Bouillon, 2003; Mukherjee et al., 2004) levels, which are responsive to metabolic and nutritional signals, all appear to

play a role in bone regulation, both directly and indirectly through an impact on muscular forces and loads (Solomon & Bouloux, 2006).

Research examining typical nutritional intake in jockeys has suggested that the typical energy intake reported may not be sufficient to maintain the lifestyle of a physically active professional jockey (Dolan et al., 2009; Dolan et al., 2008; Leydon & Wall, 2002, Labadarios, 1988). It was hypothesised therefore that an apparent energy deficit may have implications for bone mass in this group (Dolan et al., 2010; Warrington et al., 2009). The aim of this study therefore was to test the hypothesis that disruptions to a number of reproductive and metabolic hormones may have influenced the regulation of bone mass in a group of jockeys.

5.3. Methods

Aims and Objectives:

The aim of this study was to examine a number of reproductive and metabolic endocrine factors in a group of jockeys and to identify whether any of these factors influence bone regulation in this group.

Objective One:

To evaluate bone mass in a group of jockeys and age, gender and BMI matched controls.

Objective Two:

To evaluate bone turnover in a group of jockeys and age, gender and BMI matched controls.

Objective Three:

To examine the status of a number of essential reproductive and metabolic hormones associated with bone metabolism in a group of jockeys and age, gender and BMI matched controls.

Hypothesis:

That endocrine levels of a number of reproductive and metabolic hormones may be disrupted in the jockey group and that this may impact on bone mass and regulation.

5.3.1. Research Design Overview

Twenty jockeys and 20 age, gender and BMI matched recreationally physically active controls volunteered to participate in this study. Each participant provided a fasted early morning blood sample and second morning void urine sample for analysis of key biochemical markers of bone turnover and a number of endocrine factors associated with metabolism. BMD, BMC and BA of the total body, lumbar spine (L2 – 4) and femoral neck were assessed by DXA scanning.

5.3.2. Participants

The experimental group consisted of 20 full-time professional jockeys who were recruited via mass mailing to all registered jockeys in Ireland. The control group were recruited via mass emailing to students and staff in Dublin City University. Ethical approval for this study was granted by the Dublin City University Research Ethics Committee. All subjects provided written informed consent and medical history prior to participation in this study. Any participant with a medical condition known to affect either bone health or metabolic function was excluded from this study.

5.3.3. Blood and Urine Collection and Storage

Early morning blood and urine samples were taken in a fasted state. Blood samples were acquired via single venous puncture into the antecubital vein into a vacutainer coated with silica gel to allow for serum separation. Samples were allowed to stand for 30 minutes at room temperature before being placed on ice until return to the laboratory. Samples were then centrifuged at 3000 rpm for 15 minutes (ALC multispeed refrigerated centrifuge PK121R, Medical Supply Co Ltd, Ireland). Serum was separated and stored at -80°C until analysed. Urine samples were taken from the second morning void and placed on ice immediately until transferred to the -80°C freezer.



Illustration 5.1. Blood Sampling

5.3.4. Biochemical Analysis and Assays

Albumin, calcium, alkaline phosphatase, creatinine, inorganic phosphorous and magnesium were analysed using a kinetic color test, photometric color test or photometric UV test on the OLYMPUS analyser (Beckman Coulter, Ireland). Serum 25 (OH) D levels were measured using a commercially available competitive radioimmunoassay kit (Diasorin, Stillwater, USA). Reagents used in conjunction with the ISE module of OLYMPUS analysers were used for the quantitative determination of the electrolytes: sodium (Na^+), potassium (K^+) and chloride (Cl^-) (Beckman Coulter, Ireland). Cortisol, free thyroxine (free T₄), follicle stimulating hormone (FSH), lutenising hormone (LH) and thyroid stimulating hormone (TSH) were analysed using a paramagnetic particle chemiluminescent immunoassay on the Access Immunoassay system (Beckman Coulter, Ireland). Serum total procollagen type 1 amino terminal propeptide (P1NP) was analysed by immunoassay on an ELECSYS & Cobas E analyzer (Roche Diagnostics, Ireland). Testosterone was measured by liquid phase radioimmunoassay (Spectria, Orion Diagnostica, Finland). Sex hormone binding globulin (SHBG) was measured by solid phase two site chemiluminescent immunometric assay (Immulite 2000, Siemens, USA). Insulin-like growth factor-1 (IGF-1) was measured using a commercially available ELISA kit (R&D Systems Inc, Abingdon, UK). Cross linked N-telopeptides of type 1 collagen (NTx) was measured in the urine using an Osteomark EIA kit on an Etimax analyser (Claymon Biomnis Laboratories, Dublin, Ireland). Assay values were corrected for urinary dilution by urinary creatinine analysis. Creatinine concentration was measured by a kinetic Jaffe method. Urinary NTx/creatinine was expressed in nanomoles of bone collagen equivalents/liter per millimole creatine/liter.

5.3.4.1. Uncoupling Index (UI)

An “uncoupling index” between the two markers of bone turnover (serum P1NP and urinary NTX for bone formation and resorption respectively) was calculated so to allow assessment of the mean state of resorption/formation as previously described (Proteau et al., 2006). Z scores for each variable were calculated by subtracting the mean from each individual score and dividing by the standard deviation of all scores. This created a data set with a mean of 0 and a standard deviation of 1. The Z score of resorption was subtracted from the Z score of formation and a negative result was

taken to indicate a bone turnover balance in favour of resorption, while a positive result suggested a bone formative state.

5.3.4.2. Calculation of Free and Bioavailable Testosterone

Free (FT) and bioavailable testosterone (BAT) were calculated using the results for testosterone, SHBG and albumin and according to the methods described by Vermeulen et al., (1999):

$$BAT = (T * 100) / SHBG$$
$$FT = (T - (N * FT)) / Kt (SHBG - T + (N * FT))$$

Where Kt refers to the association constant of SHBG for testosterone, i.e. $1 * 10^9 \text{ L mol}^{-1}$; $N = KaCa + 1$, Ka is the association constant of albumin for testosterone, i.e. $3.6 * 10^4 \text{ L Mol}^{-1}$ while Ca is the albumin constant. This can be assumed as 43 g L^{-1} but this assumption was unnecessary as albumin levels were directly measured in this study. This calculation has been suggested as providing a reliable index of FT (Morley et al., 2002; Vermeulen et al., 1999).

5.3.5. Bone Mass and Body Composition

Bone mass was determined by dual energy x-ray absorptiometry (DXA) scanning using the GE lunar prodigy advance (GE Medical Systems, UK). Scans were performed to provide a measure of bone mass of the total body, lumbar spine (vertebrae L2-4) and femoral neck as previously described in study 2 (see chapter 4). Bone mineral apparent density (BMAD) and height and lean mass adjusted BMC were calculated as previously described (see chapter 4).

5.3.6. Statistical Analysis

All data were analysed using SPSS for Windows, version 17. Normality of data distribution was determined using the Shapiro Wilks test. Differences between the groups were identified using an independent samples t-test or Mann Whitney U test depending on data distribution. Any variable identified as being significantly different

between the groups was entered into a bivariate correlation analysis and Pearson's product moment was used to determine the strength of the relationship between variables. General linear modelling using univariate analysis was used to identify significant covariates of bone mass. Total body, lumbar spine and femoral neck bone mineral density (BMD), bone mineral concentration (BMC) and bone area (BA) were entered as independent variables. Group was included as the fixed factor and any variable identified as being significantly associated with these factors by bivariate correlation analysis was included as a covariate. Stepwise linear regression was then used to formulate the best model for estimation of each of the bone variables with the results of the univariate analysis included as dependent variables. All correlation and predictive analysis was performed following log transformation of all variables.

5.4. Results

5.4.1. Descriptive Data

All descriptive data and anthropometric characteristics of the participants are presented in Table 5.1. A number of significant differences were identified between the groups. These included: height, body mass, fat and lean mass. Groups had comparable levels of body mass and lean mass when assessed relative to height (BMI, and LMI).

Table 5.1: Anthropometric and Body Composition Information:

	Jockeys (n=20)	Controls (n=20)
Age (yr)	25.9 ± 3.26	23.9 ± 3.36
Height (m)	1.7 ± 0.07	1.78 ± 0.06**
Weight (kg)	61.1 ± 5.4	69.5 ± 6.7**
BMI (kg·m⁻²)	21.36 ± 1.8	21.99 ± 1.62
Lean Mass (kg)	52.53 ± 5.2	57.48 ± 6.01**
LMI (kg·m⁻²)	18.33 ± 1.46	18.2 ± 1.51
Fat Mass (kg)	6.84 ± 3.63	9.28 ± 2.97*
FMI (kg·m⁻²)	2.41 ± 1.37	2.94 ± 0.9*
% Body Fat	11.4 ± 5.6	13.9 ± 4*

*Data presented as mean ± SD, *p < 0.05; **p < 0.01*

5.4.2. Bone Mass Information

All indices of bone mass are presented in Table 5.2. Significant differences were shown between the groups for all absolute variables. When BMC was adjusted for height (g·m⁻³) no significant differences were identified between the groups. Control participants showed significantly greater bone area than the jockey group. This finding was reversed however when BA was assessed relative to m² of height, indicating wider bones relative to height in the jockey group. Calculation of bone mineral apparent volumetric density (BMAD) showed that the jockey group had significantly lower lumbar spine (L2 – 4), but not femoral neck BMAD than the control group. Significant

differences were also shown between the groups in relation to total body and lumbar spine BMC expressed relative to lean mass (g kg^{-1}).

Table 5.2: Bone Mass Information

	Jockeys (n=20)	Controls (n=20)
Total BMD (g cm^{-2})	1.134 ± 0.05	$1.27 \pm 0.06^{**}$
Total BMC (g)	2638 ± 285	$3121 \pm 346^{**}$
BMC – head (g)	2183 ± 256	$2645 \pm 314^{**}$
Total BA (cm^2)	2320 ± 176	$2458 \pm 201^*$
BA – head (cm^2)	2091 ± 166	$2227 \pm 193^*$
BMC/ht³ (g cm^{-3})	543 ± 36	556 ± 44
BA/ht² ($\text{cm}^2 \text{m}^{-2}$)	809 ± 31	$778 \pm 39^{**}$
L2 – 4 BMD (g cm^{-2})	1.11 ± 0.08	$1.28 \pm 0.12^{**}$
L2 – 4 BMC (g)	49.68 ± 7.57	$61.74 \pm 9.93^{**}$
L2 – 4 BA (cm^2)	44.58 ± 5.14	$48.05 \pm 4.76^*$
L2 – 4 BMAD (g cm^{-3})	0.137 ± 0.01	$0.149 \pm 0.02^{**}$
FN BMD (g cm^{-2})	1.06 ± 0.098	$1.15 \pm 0.13^*$
FN BMC (g)	5.55 ± 0.69	$6.25 \pm 0.72^{**}$
FN BA (cm^2)	5.22 ± 0.35	$5.45 \pm 0.3^*$
FN BMAD (g cm^{-3})	0.39 ± 0.04	0.405 ± 0.04
TB BMC/LM (g kg^{-1})	50.3 ± 3.6	$54.3 \pm 3^{**}$
L2 – 4 BMC/LM (g kg^{-1})	0.95 ± 0.13	$1.08 \pm 0.14^{**}$
FN BMC/LM (g kg^{-1})	0.105 ± 0.01	0.109 ± 0.01

*Data presented as mean \pm SD, * $p < 0.05$; ** $p < 0.01$*

5.4.3. Bone Turnover:

Markers of bone turnover are presented in Table 5.3. Bone resorption was shown to be elevated in the jockey group as evidenced by significantly increased urinary NTx and NTx/creatinine. In addition calculation of an uncoupling index (UI) between markers of bone formation and resorption showed that the value of the UI was significantly

greater in the control group when considered relative to the jockey group. The negative value of the UI in the jockey group indicated a bone metabolic balance in favour of bone resorption while the reverse appeared to be true of the control group.

Table 5.3: Bone Turnover

	Jockeys (n=20)	Controls (n=20)
Urinary Creatinine (mmol·L⁻¹)	18.89 ± 6.88	17.65 ± 8.57
Urinary NTx (nmol·L⁻¹)	1465 ± 778	959 ± 454*
Urinary NTX/Creatinine (nmol·mmol⁻¹)	76.94 ± 29.52	55.9 ± 13.9**
Serum P1NP (ng·ml⁻¹)	88.62 ± 46.69	88.77 ± 31.42
Uncoupling Index (NTX)	-0.35 ± 0.88	0.37 ± 0.96*
Uncoupling Index (NTX·Creatinine⁻¹)	-0.34 ± 0.81	0.41 ± 0.97*
Serum Creatinine (μmol·L⁻¹)	76.24 ± 10.78	81.04 ± 10.32
Alkaline Phosphatase (U·L⁻¹)	84.36 ± 26.33	77.07 ± 23.23

*Data presented as mean ± SD, * $p < 0.05$; ** $p < 0.01$*

5.4.4. Micronutrient and Electrolyte Information

No differences were shown in serum concentrations for any of the analysed micronutrients as presented in Table 5.4. Mean serum 25(OH) D levels were below the recommended threshold of 50nmol·l⁻¹ in the jockey group (Mosekilde, 2005).

Table 5.4: Micronutrient and Electrolyte Information

	Jockeys (n=20)	Controls (n=20)
Calcium (mmol·L⁻¹)	2.53 ± 0.12	2.46 ± 0.11
Corrected Calcium (mmol·L⁻¹)	2.39 ± 0.08	2.35 ± 0.08
PO4 (mmol·L⁻¹)	1.23 ± 0.19	1.26 ± 0.18
Magnesium (mmol·L⁻¹)	0.88 ± 0.05	0.87 ± 0.07
Sodium (mmol·L⁻¹)	141.5 ± 5.7	140.2 ± 4.5
Potassium (mmol·L⁻¹)	5.24 ± 1.25	4.35 ± 0.46
Chloride (mmol·L⁻¹)	104.2 ± 4.3	103 ± 3.3
Vitamin D/ 25(OH)D (nmol·L⁻¹)	43.9 ± 15.5	52.4 ± 20.3

*Data presented as mean ± SD, * $p < 0.05$; ** $p < 0.01$*

5.4.5. Endocrine Information

Hormonal measures are presented in Table 5.5. No differences were shown between the groups in relation to testosterone. SHBG was significantly elevated in the jockey group ($p = 0.000$) and the percentage of free and bioavailable testosterone were significantly decreased in this group ($p < 0.01$). A trend toward significantly lower IGF-1 levels was identified in the jockey group, although this finding did not reach statistical significance ($p = 0.07$).

Table 5.5: Endocrine Data

	Jockeys (n=20)	Controls (n=20)
Testosterone (nmol·L⁻¹)	26.48 ± 5.19	24.03 ± 5.27
SHBG (nmol·L⁻¹)	41.21 ± 9.77	28.24 ± 7.98**
Free Testosterone (nmol·L⁻¹)	0.512 ± 0.114	0.571 ± 0.141
Bioavailable Testosterone (nmol·L⁻¹)	12.87 ± 2.99	14.36 ± 3.44
Free Testosterone (%)	1.94 ± 0.27	2.35 ± 0.32**
Bioavailable Testosterone (%)	48.89 ± 7.38	59.18 ± 6.74**
FT4 (pmol·L⁻¹)	10.89 ± 2.08	10.81 ± 1.85
TSH (mIU·L⁻¹)	1.97 ± 0.79	2.02 ± 0.72
LH (mIU·L⁻¹)	5.69 ± 2.96	4.99 ± 2.99
FSH (mIU·L⁻¹)	4.99 ± 2.67	4.37 ± 2.5
Albumin (g·L⁻¹)	46.2 ± 2.87	45.18 ± 1.95
IGF-1 (ng·ml⁻¹)	15.03 ± 5.36	18.69 ± 7.01~
Cortisol (nmol·L⁻¹)	471.15 ± 143.87	484.25 ± 101.04
Cortisol:Testosterone Ratio	0.284 ± 1.54	-0.284 ± 1.29

*Data presented as mean ± SD, * $p < 0.05$; ** $p < 0.01$;*

5.4.6. Correlation Analysis

Bivariate correlational analysis showing significant associations with the more relevant bone variables are presented in Table 5.6. In addition lutenising hormone (LH), although not significantly different between the groups showed a significantly negative association ($p < 0.05$) with L2 – 4 BMD, L2 – 4 BMC and FN BMC with r values of -0.488, -0.380 and -0.326 respectively. No relationship was shown between SHBG and IGF-1 in this study. SHBG was shown to be positively correlated with urinary NTx ($r = 0.341$, $p = 0.036$). IGF-1 was positively correlated with serum P1NP ($r = 0.347$, $p = 0.030$).

Table 5.6: Bivariate Correlations with Bone Mass

Dependent Variables	Associated Variables (p < 0.05)
Total Body BMD (g·cm⁻²)	Height (r = 0.687); lean mass (r = 0.653); fat mass (r = 0.462); SHBG (r = -0.499); % free testosterone (r = 0.509); % bioavailable testosterone (r = 0.483)
Total Body BMC (g)	Height (r = 0.860); lean mass (r = 0.848); fat mass (r = 0.394); triglycerides (r = 0.389); SHBG (r = -0.419); % free testosterone (r = 0.415); % bioavailable testosterone (r = 0.441)
Total Body BA (cm²)	Height (r = 0.842); lean mass (r = 0.850)
L2 – 4 BMD (g·cm⁻²)	Height (r = 0.570); lean mass (r = 0.515); fat mass (r = 0.382); SHBG (r = -0.499); % free testosterone (0.509); % bioavailable testosterone (r = 0.483)
L2 – 4 BMC (g)	Height (r = 0.796); lean mass (r = 0.636); SHBG (r = -0.363); % free testosterone (r = 0.360); % bioavailable testosterone (r = 0.353)
L2 – 4 BA (cm²)	Height (r = 0.767); lean mass (r = 0.556)
Femoral Neck BMD (g·cm⁻²)	Age (r = -0.461); height (r = 0.421); lean mass (r = 0.475); IGF-1 (r = 0.517)
Femoral Neck BMC (g)	Age (r = -0.435); height (r = 0.699); lean mass (r = 0.720); % free testosterone (r = 0.325); % bioavailable testosterone (r = 0.353); IGF-1 (r = 0.424)
Femoral Neck BA (cm²)	Height (r = 0.761); lean mass (r = 0.706)

General linear modelling using a univariate analysis revealed those variables which could be considered to be significant covariates of the bone mass variables and are presented in Table 5.7 (p < 0.05).

Table 5.7: Significant Covariates of Bone Mass:

	Predictors (p < 0.05)
TB BMD (g·cm⁻²)	Group; lean mass; SHBG
TB BMC (g)	Height; lean mass; fat mass; SHBG (p = 0.054)
TB BA (cm²)	Height; lean mass; group*lean mass
L2 – 4 BMD (g·cm⁻²)	Lean mass; SHBG
L2 – 4 BMC (g)	Group; height
L2 – 4 BA (cm²)	Height
FN BMD (g·cm⁻²)	Lean mass; IGF-1
FN BMC (g)	Height; lean mass; IGF-1
FN BA (cm²)	Height; lean mass

Predictive equations were generated using the results of a stepwise linear regression model. R² values and power equations calculated were:

Total Body Bone Mineral Density (g·cm⁻²):

$$\text{Total Body BMD} = -0.709 - (\text{Jockey}^{0.058}) + (\text{LBM}^{0.285}) - (\text{SHBG}^{0.064})$$

(R² = 0.791)

Total Body Bone Mineral Content (g):

$$\text{Total Body BMC} = 4.451 + (\text{Height}^{1.455}) + (\text{LBM}^{0.637}) + (\text{FM}^{0.078})$$

(R² = 0.903)

Total Body Bone Area (cm²):

$$\text{Total BA} = 5.594 + (\text{LBM}^{0.388}) + (\text{Height}^{1.172}) - (\text{LBMControl}^{0.008})$$

(R² = 0.867)

L2 – 4 Bone Mineral Density (g·cm⁻²):

$$\text{L2 – 4 BMD} = -1.077 + (\text{LBM}^{0.430}) - \text{SHBG}^{0.135}$$

$$(R^2 = 0.449)$$

L2 – 4 Bone Mineral Content (g):

$$L2 - 4 BMC = 2.106 + (Height^{3.451})$$

$$(R^2 = 0.634)$$

L2 – 4 Bone Area (cm²):

$$L2 - 4 BA = 2.717 + (Height^{2.024})$$

$$(R^2 = 0.589)$$

Femoral Neck Bone Mineral Density (g cm⁻²):

$$FN BMD = -1.576 + (IGF-I^{0.111}) + (LBM^{0.341})$$

$$(R^2 = 0.382)$$

Femoral Neck Bone Mineral Content (g):

$$FN BMC = -0.867 + (LBM^{0.423}) + (Height^{1.287}) + (IGF-I^{0.085})$$

$$(R^2 = 0.647)$$

Femoral Neck Bone Area (cm²):

$$FN BA = 0.462 + (height^{0.790}) + (LBM^{0.194})$$

$$(R^2 = 0.636)$$

5.5. Discussion

Results from this study appear to support previous findings, both from this thesis (chapter 4) and the published literature (Warrington et al., 2009; Leydon & Wall, 2002) of low bone mass in jockeys. While the differences observed between the groups may in part be accounted for by variations in height and lean mass, it appears that endocrine adjustments within the jockey group may also be involved in bone mass regulation within this group. It is thought that these adjustments may have occurred in response to the chronically weight restricted lifestyle apparently followed by this group (Dolan et al., 2010).

All participants in this study were matched for age, gender, BMI and LMI suggesting a relative homogeneity between the two groups, although the control group was significantly taller and heavier and had a greater absolute amount of lean mass than the jockeys. Despite this, differences in bone mass were shown to be present between the groups for the majority of variables measured (see Table 5.2). Adjustments for differences in body size and body composition were made as described in study two (see chapter four). Similar to study two, results of the bone mass analysis suggest that a degree of the variation in bone mass may be related to differences in height and lean mass between the groups. Additional factors appear to be present however which may have influenced the mean bone mass observed for each group.

Results from the biochemical analysis of bone turnover lend support to the hypothesis that osteogenic function is somewhat disrupted in jockeys (see Table 5.3). Bone remodelling is a dynamic process and measurement of specific markers of bone turnover allows analysis of the state of the bone modelling process. Biochemical markers of bone turnover have been suggested as being useful in the prediction of the rate of bone loss (Garnero et al., 2009; Proteau et al., 2006) and fracture risk (Garnero et al., 2000; Romeo & Ybarra, 2007) and it has been suggested that they may provide additional influence and value to the predictive value of bone mass assessment (Dogan & Pasaci, 2002). Results from the present study suggest that bone resorption is elevated in the jockey group, as is evident by the significantly higher levels of urinary NTx and NTx/creatinine reported. In contrast formation rates, based on the levels of serum P1NP, appear to be similar between the jockey group and the controls.

Calculation of an uncoupling index using these markers, lends support to this contention in that it appears to suggest that the jockey group were in a mean resorptive state with the opposite appearing to hold true of the control group. That the control group appeared to be in a mean bone formative state would suggest that peak bone mass has not yet been achieved in this group. Given that there was no significant difference in the ages of the two groups, this makes the observation that the jockey group were in a mean bone resorptive state of greater concern. Development of osteoporosis appears to be largely determined by the level of peak bone mass achieved (Mora et al., 2000), which occurs largely in the first two decades of life (Bachrach et al., 2007) and the subsequent rate of bone loss (Hansen et al., 1991). Results from the present study, indicating low bone mass, accompanied by an increased rate of bone loss may enhance susceptibility to osteopenia and osteoporosis in this group. Elevated urinary NTx has previously been reported in a group of jockeys (Waldron Lynch et al., 2009). This study also reported elevated serum P1NP in comparison to a control group. Participants in the study by Waldron Lynch et al., (2009) consisted of those eleven jockeys which displayed the lowest BMD values from a larger group of 27 however, suggesting a substantial selection bias toward those with low initial BMD. Participants in the current study were selected from a random sample of volunteers and so may be considered to better reflect the Irish jockey population as a whole.

Serum analysis of a number of key micronutrients and electrolytes revealed no difference between the groups (see Table 5.4). Adequate intake of key micronutrients has long been cited as a key determinant of optimum bone strength and quality with calcium and vitamin D suggested as being the most relevant to consider (Miller et al., 2001; Mosekilde, 2005). Previous research on a group of Irish jockeys suggested a low calcium intake (Dolan et al., 2010) and it was suggested that this may have had a mediatory effect in the low bone mass levels identified in this group (Warrington et al., 2009). The serum calcium levels reported for the jockeys were similar to those observed in the control group in the present study. Serum calcium levels must be maintained within strict physiological limits however and bone tissue represents an available reservoir of calcium should intake ever be insufficient to maintain serum levels (Peacock, 2010). Maintenance of serum calcium levels may however have an impact on bones structural integrity and further research may be required so to assess the relationship between calcium intake and bone health in this specific population.

Although widely known as a micronutrient, vitamin D is in fact a hormone and has an important function in the promotion of bone health, largely through the stimulation of calcium and phosphate absorption from the small intestine and reduction of urinary calcium and phosphate excretion (Saladin, 1999b). Serum 25(OH) D levels have been suggested as being the most clinically relevant indicator of nutritional status (Mosekilde, 2005). Mean jockey serum 25(OH)D levels were below the recommended threshold of $50 \text{ nmol}\cdot\text{L}^{-1}$ (Mosekilde, 2005) which may have an impact on calcium absorption, bone mass and bone turnover (Iuliano Burns et al., 2009). Serum 25(OH) D levels were not shown to be significantly different between the groups however and so it is unlikely that this is a significant contributory factor to the differences in bone mass reported here.

Endocrine levels of a number of key reproductive and metabolic hormones were examined in this study (see Table 5.5). Perhaps the most relevant finding from this was that SHBG was significantly elevated in the jockey group, although testosterone levels were similar to those observed for the control group. Testosterone has been suggested as being the most biologically relevant of the male androgens (Venken et al., 2008), which are known as the male sex hormones, and are one of the primary anabolic agents within the body (Solomon & Bouloux, 2006). SHBG is a plasma glycoprotein whose primary biological action is to bind many of the androgens and estrogens, thereby disabling and regulating their action (Kahn et al., 2002). Higher concentrations of SHBG are associated with a decrease in free testosterone availability. SHBG levels appear to increase with age and have been suggested as having a role to play in the muscle and bone loss which is associated with aging (Khosla et al., 1998). This effect is said to be mediated through gonadotropic action (Feldman et al., 2002). Elevated SHBG levels as indicated in the present study suggest a disruption to the reproductive hormonal system. Disruptions to reproductive function have long been cited as a potential consequence of energy deficiency (De Souza & Williams, 2004) and it has been proposed that this may occur in an attempt to conserve energy so to allow maintenance of more immediate and essential processes (Wade et al., 1996).

IGF-1 levels appeared to be reduced in the jockey group in this study. IGF-1 is a pleiotropic growth factor which appears to exert an anabolic effect on virtually all of the tissues of the body (Rajaram et al., 1997). It appears that many of the actions of

IGF-1 are mediated through the release and action of growth hormone, and it has been suggested that serum IGF-1 may be a good marker of integrated GH secretion and function (Aimaretti et al., 2005; Holt et al., 2001). It seems that the IGF system is extremely sensitive to metabolic alterations and changes within it may be essential to the processes that link nutrition and growth (Frystyck et al., 1999). Differences in IGF-1 levels identified in this study did not reach statistical significance, but a trend toward significance between the groups was identified ($p = 0.07$). Given that current evidence suggests that IGF-1 levels may be reduced in times of energy imbalance (Counts et al., 1992; Haspolat et al., 2007; Misra et al., 2003b), it is thought that this finding may still be clinically relevant, despite the lack of statistical significance. Previous research appears to indicate that the actions of SHBG and IGF-1 are related to one another. Pfeilschifter et al., (1996) reported an inverse relationship between SHBG and IGF-1, IGF-2 and IGFBP3. Despite this finding no such relationship was shown in this study.

It was thought likely that the endocrine adaptations identified within this study may have been a regulatory factor in the low bone mass reported in the jockey group. Bivariate correlational analysis, general linear modelling using univariate analysis and a stepwise linear regression were used in this study so to predict those factors which may have a significant and independent relationship with bone mass. Pearsons bivariate correlation analysis was used to identify significant associations between bone mass and those metabolic factors which were identified as being significantly different between the groups. Univariate analysis was then used to identify significant covariates and the results of this were used to generate prediction equations for each of BMD, BMC and BA of the total body, lumbar spine and femoral neck. In agreement with results presented earlier in this thesis (see chapter four), height and lean mass consistently emerged as independent significant predictors of the different elements of bone mass. In addition, bone area of the jockey group appeared to be enhanced in comparison to the control group once the effects of lean mass and height were accounted for. This finding may suggest an increase in periosteal apposition in response to mechanical loading, but insufficient mineral accrual as a result of an assumed energy imbalance although further research may be required so to support and refute this hypothesis.

Both SHBG and IGF-1 emerged as independent significant predictors of bone mineral density and concentration. The relationship between both these factors and bone has been previously documented (Khosla et al., 1998; Slemenda et al., 1996). SHBG may affect the regulation of bone mass (Slemenda et al., 1996), by influencing the amount of bioavailable testosterone present (Ongyphadkhanalakul et al., 1995) and has been suggested as being associated with an increased risk of osteoporotic fractures (Khosla et al., 1998). Interestingly however, the elevated SHBG levels in this study did not translate into significantly reduced free or bioavailable testosterone levels (see Table 5.5). SHBG has been described as the principle regulator of both testosterone and estrogen activity (Slemenda et al., 1996). Testosterone is capable of converting to 17β estradiol by the P430 aromatase enzyme and so is capable of exerting an effect through the estrogen receptors α or β . Bioavailable estrogen levels have been suggested as having a primary influence on bone in males as well as females (Khosla et al., 1998), although there is no doubt that testosterone also plays a role (Vandershuerren et al., 2003). It is possible therefore that elevated SHBG levels may impact on bone mass through a regulatory effect on either testosterone or estrogen. Further research may be required so to more fully examine the effect of SHBG on bone mass and health in this population. That SHBG was elevated does however suggest some degree of disruption to usual reproductive and gonadal function in the jockey group.

In support of this finding was evidence that LH showed a significant and negative association with lumbar spine (L2 – 4) BMD and BMC and femoral neck BMC. Increases in SHBG are mediated through the action of the pituitary gonadotropins FSH and LH (Feldman et al., 2002), which act primarily as regulatory agents in a number of anabolic and reproductive processes. That LH was not significantly different between the groups, nor was it related to SHBG or total, free or bioavailable testosterone suggests that it may not be the most relevant factor to consider in this issue. However evidence of significantly negative associations between this hormone and bone mass at a number of sites lends support to the suggestion that disruptions to reproductive hormonal function may influence bone mass in jockeys. Previous research examining jockeys reported no evidence of hypogonadism and “normal” serum levels of SHBG (Waldron Lynch et al., 2009). That study did not however compare serum endocrine markers to a control group and results from the present study suggest that elevated

SHBG levels may have had an influence on bone mass, even though mean levels were within recommended physiological ranges.

IGF-1 was shown to be an independent and significant predictor of femoral neck bone mineral density and concentration, supporting previous research which suggests a clear association between IGF-1 and bone mass (Jurimae & Jurimae, 2006; Nindl et al., 2008; Rajaram et al., 1997). IGF-1 appears to influence the regulation of both bone formation and resorption. Furthermore, it has been suggested that IGF-1 may aid in the mediation of the complex coupling process of bone remodelling (Rubin et al., 2002c) as the presence of IGF-1 during osteoclastic activity may allow formation to follow, thereby coupling the two (Ueland, 2004). It appears that IGF-1's action on bone is mediated through the action of its binding protein IGFBP5 (Campbell & Andress, 1997), which may also have an independent action of its own on both osteoblast (Campbell & Andress, 1997) and osteoclast (Kanatani et al., 2000) activity.

Further evidence of the apparent catabolic role of SHBG and anabolic role of IGF-1 was provided by the demonstration of a significantly positive relationship between SHBG and the bone resorption marker urinary NTx and between IGF-1 and the bone formation marker P1NP.

5.6. Limitations

There are a number of limitations associated with this study which may have affected interpretation of results. Sample size was small which may have affected statistical power in this study. Results are based on single serum samples, taken in the morning after an overnight fast. Most hormones are released in an intermittent pulsatile fashion in quantities which are sufficient to meet anticipated needs, the rhythmic design of which is intended to enable the body to function as efficiently as possible. Frequent sampling so to allow examination of the pulsatile release of these hormones may provide a more in depth analysis of possible adjustments to endocrine function in jockeys. In addition examination of a broader range of associated factors may allow a more complete analysis to be made. Inferences have been made within this study as to the nutritional habits of the jockey group which are based on previous data (Dolan et al., 2010; Labadarios, 1988; Leydon & Wall, 2002) and the assumed energy imbalance habitually experienced by this group. Analysis of typical energy intake and expenditure in this group may have lent support to these assumptions.

5.7. Conclusion:

Low bone mass was reported in the jockey group in this study, along with an apparent bone turnover state in favour of resorption. Adaptations to a number of reproductive and anabolic endocrine agents, in particular SHBG and IGF-1 were shown to be related to bone mass in this group and may be a contributory factor in this finding. It is thought that disturbances to reproductive and anabolic processes may occur in an attempt to conserve energy within this group and suggest a potentially catabolic state in these jockeys. Further research may be required so to more fully elucidate the effects of a chronically weight restricted lifestyle on homeostatic and osteogenic function in this group.

Chapter Six

Study Four: The Effects of Six months Whole Body Vibration Therapy on Bone Mass and Turnover in a Group of Jockeys



6.1. Abstract

The low bone mass and high rate of bone turnover reported within previous studies in this thesis demonstrate the importance of identifying appropriate intervention osteogenic intervention strategies for use in this population. **Aim:** The aim of this study was to examine the impact of six months whole body vibration therapy on bone mass and turnover in a group of jockeys. **Methods:** Twenty three jockeys took part in this study consisting of a vibration training group (VTG) ($n = 12$; 29 ± 7 yrs; 1.68 ± 0.08 m; 60.1 ± 5.2 kg) and a control group ($n=11$; 26 ± 4 yrs; 1.71 ± 0.06 m; 63.1 ± 5.4 kg). Whole body vibration (WBV) platforms, which oscillated at 0.3 g and 30 Hz were provided to the VTG for a period of six months. All participants were provided with calcium supplements, supplying 800 mg day^{-1} . Prior to the commencement and following completion of the 6 month intervention bone mass was assessed by DXA scanning and bone turnover was determined from measurement of serum P1NP and urinary NTx levels. **Results:** No aspect of body composition, bone mass or turnover changed in the VTG group following the six month training intervention. In contrast the control group displayed a significant decrease in the bone formation marker serum P1NP (94.7 ± 10.4 Vs $66.6 \pm 2.33 \text{ nmol l}^{-1}$ for pre and post assessment respectively, $p < 0.05$) and an increase in L2 – 4 BMC (54.5 ± 10.4 Vs $56.4 \pm 11.02 \text{ g}$ for pre and post assessment respectively $p < 0.05$). **Conclusion:** Results from this study do not support the use of whole body vibration as an osteogenic intervention in this group. The effects of calcium supplementation require further research. Poor compliance and a high attrition rate may have affected interpretation of results and suggest that WBV may not be a practical or appropriate intervention for use in this group.

6.2. Introduction

Recent evidence (Warrington et al., 2009) and results from studies two and three of this thesis (see chapters 4 and 5) appear to suggest that jockeys have significantly lower bone mass than that reported for healthy males of a similar age. Low bone mass is associated with an increase in fracture susceptibility (Cummings et al., 2002; Lewiecki et al., 2008) and it is suggested that the finding of low bone mass and apparently increased bone turnover in this group may have particular implications for jockeys given the high risk nature of the sport and frequent occurrence of fractures and falls (Hitchens et al., 2009; Waller et al., 2000). It appears that low bone mass in this group may be a consequence of the chronically weight restrictive lifestyle apparently followed by many of these athletes (Dolan et al., 2010; Warrington et al., 2009). As such, the appropriateness of current weight standards and the methods used to achieve them should be reviewed as a matter of priority. In the interim however appropriate osteogenic interventions may be required so to aid in the enhancement of bone mass in jockeys currently involved in this sport.

Whole body vibration (WBV) therapy has been cited as the most effective non pharmacological intervention available today for protection against osteoporosis (Rubin et al., 2006). Low level mechanical stimulation transmitted via whole body vibration has been suggested as being anabolic to, or protective of bone in both animal (Christiansen & Silva, 2006; Garman et al., 2007; Maddalozzo et al., 2008; Rubinacci et al., 2008; Sehmisch et al., 2009; Xie et al., 2006) and human studies (Gilsanz et al., 2006; Rubin et al., 2004; Vershueren et al., 2004; Ward et al., 2004; Xiang Yan et al., 2008). This appears to occur as a result of a microscopic strain applied to the bone tissue, causing a responsive increase in bone strength.

It appears that the accumulative effect of a low magnitude strain, applied at a very high frequency, as is the case with the WBV therapy protocol used in this study may allow safe augmentation of the skeleton at strain levels far below those required to cause damage to bone (Rubin et al., 2006). WBV transmitted through the plantar surface of the foot (Rubin et al., 2003) appears to increase intramedullary pressure, causing fluid flow and stimulation of the osteocytes (Judex et al., 2006; Turner et al., 1994). WBV

may therefore mimic the action of matrix deformation without applying the strain magnitude required to cause actual deformation (Qin et al., 2003).

In addition to its apparent osteogenic effect, recent evidence is available which suggests that vibration therapy may also have a role to play in the development of lean muscle mass in human studies (Gilsanz et al., 2006), and in the inhibition of adipogenesis and fat acquisition in the animal model (Madalozzo et al., 2008; Rubin et al., 2007). This appeared to be achieved through a preferential differentiation of mesenchymal stem cells toward a connective tissue lineage. Given that whole body vibration therapy has been suggested as having a beneficial impact on all parameters of body composition it may be considered to represent a more holistic therapy than those which focus on bone alone. WBV may be particularly suited to jockeys therefore, given the need to enhance bone quality while maintaining a low body mass.

Research supporting the osteogenic benefits of WBV in humans may be considered quite preliminary, although the available literature appears encouraging. The majority of studies to date however appear to focus on participants with low initial bone mass (Gilsanz et al., 2006), or those who are in some way physically impaired, be it through age (Rubin et al., 2004; Xiang-Yan et al., 2008) or disability (Ward et al., 2004). In contrast, one study examining the effects of WBV in a group of young healthy adults showed no enhancement to any aspect of bone mass, quality or turnover (Torvinen et al., 2003). Results from these studies appear to suggest that the osteogenic potential of WBV may be more suited to rehabilitative purposes, rather than as an anabolic intervention. No research is available however detailing the effect of WBV on osteogenic function in an otherwise healthy, highly physically active weight category athletic group who appear to have low bone mass. The aim of this study therefore was to examine the effect of a six month WBV intervention on bone mass and turnover in a group of professional jockeys.

6.3. Methods

Aims and Objectives:

The aim of this study was to examine the impact of six months whole body vibration therapy on bone mass and turnover in a group of professional jockeys.

Objective One:

To assess the impact of six months of whole body vibration therapy at 30 Hz and 0.3g on bone mass and body composition in a group of professional jockeys

Objective Two:

To assess the impact of six months of whole body vibration therapy at 30 Hz and 0.3g on bone turnover in a group of professional jockeys

Hypothesis:

That WBV therapy will have a positive osteogenic effect on bone mass and/or turnover in this group.

6.3.1. Research Design Overview

Thirty-two professional jockeys volunteered to participate in this study. A random cross over design intervention study was designed whereby participants were randomly assigned to one of three groups, with bone mass measured at three time points (see figure 6.1). However due to a lack of compliance and a high attrition rate the original study design was rendered unsuitable. Participants who completed two bone mass or turnover assessments (baseline and retriial) were divided into a control (n = 12) and experimental group (VTG, n = 11) and data was analysed as a simple pre and post intervention analysis.

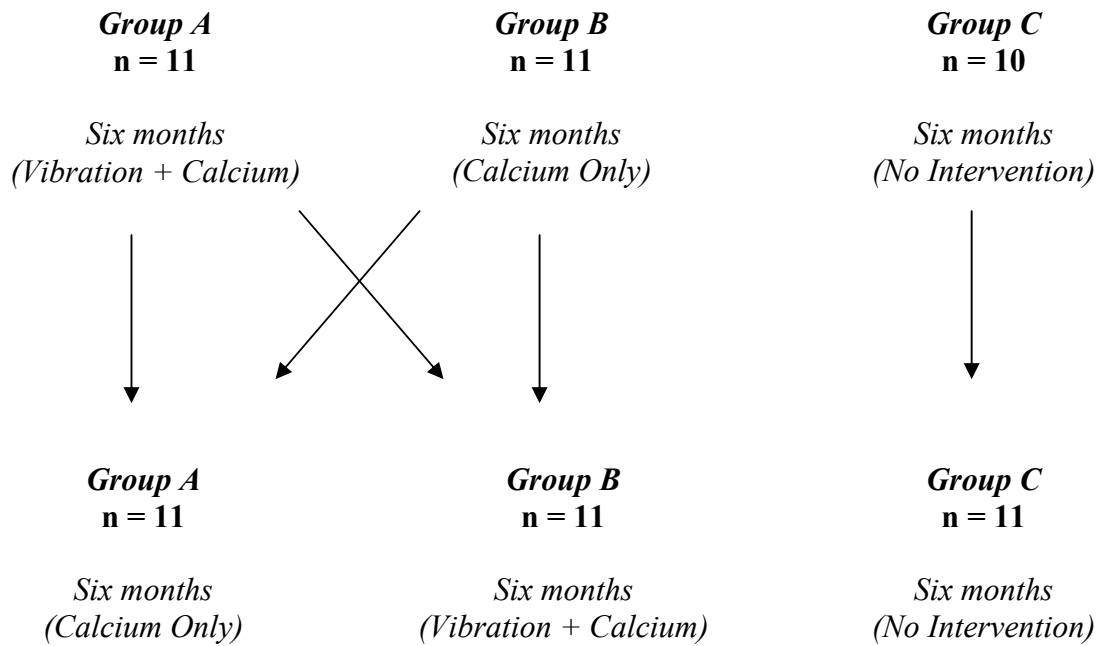


Figure 6.1: Original Study Design

6.3.2. Participants

Participants were recruited via mass mailing to all registered male jockeys on the Turf Club data base (n =204). Thirty-two jockeys volunteered to participate in the initial study. Ethical approval for this study was granted by the Dublin City University Research Ethics Committee. All subjects provided written informed consent and medical history prior to participation in this study. Any participant who had a medical condition or was undergoing any form of treatment which may impact on bone health was excluded from the study.

6.3.3. Determination of Bone Mass and Body Composition

Bone mass was determined by dual energy x-ray absorptiometry (DXA) scanning using the GE lunar prodigy advance (GE Medical Systems, UK). Scans were performed to provide a measure of bone mass of the total body, lumbar spine (vertebrae L2 - 4) and femoral neck as previously described in study two (see chapter four). Least significant biological change for BMD was calculated according to the methods reported by Bonnick et al., (2001), using the formula:

$$LSC = Z' (Pr) \sqrt{\frac{1}{n_1} + \frac{1}{n_2}}$$

where Z' reflects the level of statistical confidence, Pr the precision value and n_1 and n_2 the number of baseline and repeated measures. At a 95% confidence level the least significant change (LSC) at the femoral neck and lumbar spine was calculated as 6.87 and 4.07% respectively. Total body precision values were unavailable for this particular scanner and so the LSC for this measure could not be calculated.

6.3.4. Bone Turnover

Bone formation and resorption were assessed through examination of urinary NTX/creatinine and serum P1NP respectively. Serum samples were collected in the early morning after an overnight fast. Urine samples were taken from the fasted second morning void as described in chapter five. An uncoupling index between the two indices was calculated as previously described (see chapter five).

6.3.5. Vibratory Device

A whole body vibratory stimulus was provided using a commercially available platform, the Juvent 1000 vibration platform (Medivibes, USA). This platform has been designed to induce a vertical sinusoidal acceleration and includes sensors which enable the platform to self adjust to the specific requirements of the individual. The top peak of the platform accelerated at 0.3g where g denotes the gravitational constant of the earth ($9.81 \text{ m}\cdot\text{s}^{-1}$). This vibratory device employs the use of a linear actuator comprised of a magnet and voice coil providing a pure acoustical signal creating only vertical movement. Vibration frequency, defined as the repetition rate of the oscillatory cycle, was 30 Hz (30 cycles per second). The amplitude and frequency of this device is within International Organization for Standardization (ISO) safety guidelines and this particular platform has been certified as being safe to use for exposures up to four hours per day.

Individual vibration platforms were provided to each participant for their personal use for a period of six months. Participants were instructed to stand on the platform in a relaxed upright position for 20 minutes per day as per manufacturer instructions. Each vibratory device contains a built in electronic monitoring system which recorded daily duration of use and through which intervention compliance was determined. Compliance was encouraged through regular telephone contact. Appropriate intervention time length may be calculated as

$$\textit{Time Interval} = \textit{LSC} / (\textit{Expected Rate of Change/Year})$$

The literature regarding vibration therapy in humans may be considered quite preliminary however and does not appear to have been studied in a specific population such as jockeys before. The expected rate of change was therefore unavailable and the appropriate intervention length based on LSC could not be calculated. Previous human studies have however indicated a significant intervention effect after six months of treatment and so this was considered an appropriate and practical intervention length.



Illustration 6.1: Juvent 1000

6.3.6. Calcium Supplementation

To account for the possible confounding influence of calcium intake all participants were provided with supplementation in the form of “Aquacal Natures Calcium” (lithotamnium calcareum) (Marigot Ltd, Ireland) as previously described (Gilsanz et al., 2006). Participants were instructed to take two capsules with food in the morning and evening amounting to 800 mg·day⁻¹.

6.3.7. Statistical Analysis

Intervention affect was measured through both an Intention to Treat (ITT) analysis, and per protocol (PP) as previously described (Gilsanz et al., 2006). The ITT intervention employed the use of statistical analysis using SPSS version 17.0 to assess absolute and percentage change differences between the control and experimental group. Normality of distribution was tested using the Shapiro Wilks test. Within group differences were identified using paired sample t-tests or Wilcoxon signed ranks test, depending on the normality of data distribution. Between group differences were assessed using independent sample t-tests or the Mann Whitney U test. Any significant changes were then assessed in accordance with possible confounding variables using a repeated measures ANCOVA, with age, height and lean mass entered as covariates. The PP analysis was applied in order to identify a dose: response relationship between intervention compliance and efficacy. Participants were divided into quartiles, to allow assessment of those who fell within the lowest 25% of compliance, and to assess these versus those who fell within 25 – 50%, 50 – 75% and 75 – 100% compliance.

6.4. Results

6.4.1. Participants

Of the original 32 participants who volunteered to take part in this study, 23 completed the study (VTG, n = 11; Control n = 12), representing an attrition rate of 28%. Not all of the 23 participants who completed the study provided all retrieval data. 19 participants returned for repeat DXA scans (VTG, n = 9; Control, n = 10) and 19 provided a repeat blood sample (VTG n = 10; Control n = 9).

6.4.2. Baseline Data

Baseline anthropometric and bone characteristics of both groups are presented in Table 6.1. No differences were shown between the groups for any parameter.

Table 6.1: Baseline Anthropometric and Bone Characteristics

	VTG (n=11)	Control (n=12)
Age (yr)	29 ± 7	26 ± 4
Height (m)	1.68 ± 0.08	1.71 ± 0.06
Body Mass (m)	60.1 ± 5.2	63.1 ± 5.4
BMI (kg·m ⁻²)	21.3 ± 1.55	21.49 ± 1.67
Lean Mass (kg)	51.8 ± 4.6	54.8 ± 3.9
Fat Mass (kg)	6.4 ± 2.4	6.7 ± 2.8
Total Body BMD (g·cm ⁻²)	1.142 ± 0.059	1.17 ± 0.078
L2 – 4 BMD (g·cm ⁻²)	1.135 ± 0.069	1.160 ± 0.158
FN BMD (g·cm ⁻²)	1.053 ± 0.093	1.109 ± 0.122

Data presented as mean ± SD

6.4.3. Compliance

Participants used the vibration platform for an average of 686 ± 899 minutes throughout the six month period. This varied widely from 93 – 2762 minutes and was achieved through 57 ± 50 treatment bouts (range 14 – 172), with an average of 9 ± 4

minutes per treatment. In terms of minutes per month participants recorded an average of 114 ± 150 minutes. Figure 6.2 demonstrates minutes of compliance per month for each individual participant. Quartile ranges are illustrated by the vertical lines. One participant reached each of the latter three quartiles, while the remaining eight participants fell within the first quartile of compliance, i.e. used the platform for less than 150 minutes month⁻¹.

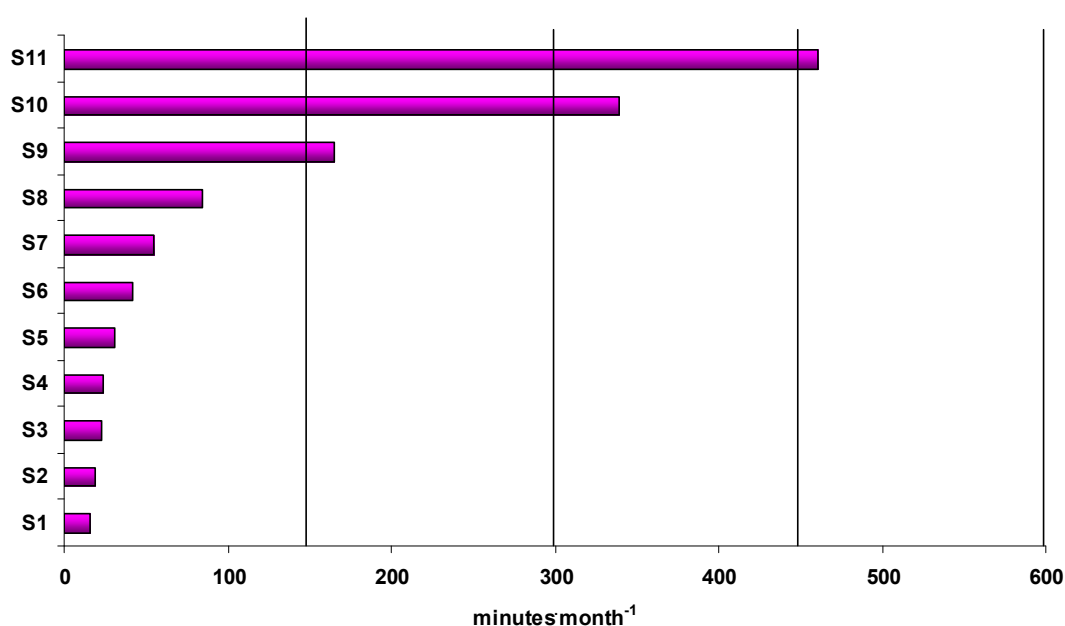


Figure 6.2: Participant compliance in minutes spent on platform per month.

6.4.4. Six Month Follow Up Testing

6.4.4.1. Anthropometric Characteristics

Anthropometric characteristics of those who underwent a repeat DXA are presented in Table 6.2. Throughout the experimental period the VTG demonstrated no significant change for any measured variable. In contrast the control group showed a decrease in body fat levels as evidenced by a significant decrease in fat mass, fat mass index and percent body fat. No differences were shown between the groups for any variable at any time.

Table 6.2: Anthropometric Characteristics – Pre and Post Intervention

	VTG (n=9)		Control (n=10)	
	Baseline	Retrial	Baseline	Retrial
Height (m)	1.69 ± 0.09	1.69 0.09	1.72 0.06	1.72 0.06
Weight (kg)	60.4 ± 5.6	61.1 ± 5.98	63.8 ± 5.5	63 ± 5.5
BMI (kg·m⁻²)	21.25 ± 1.72	21.49 ± 1.78	21.57 ± 1.57	21.28 ± 1.47
LBM (kg)	51.9 ± 4.8	52 ± 5.5	54.7 ± 4	54.8 ± 4.3
LMI (kg·m⁻²)	18.25 ± 1.35	18.26 ± 1.61	18.49 ± 1.04	18.530 ± 1.04
FM (kg)	6.5 ± 2.5	6.7 ± 2.9	7.5 ± 2.5	6 ± 1.9*
FMI (kg·m⁻²)	2.3 ± 0.91	2.37 ± 1.04	2.52 ± 0.86	2.03 ± 0.64*
Body Fat (%)	11.1 ± 3.9	11.4 ± 4.6	10.9 ± 3.6	9.7 ± 2.7*

*Data presented as mean ± standard deviation, * $p < 0.05$ from baseline within groups*

6.4.4.2. Bone Mass Characteristics: Intention to Treat Analysis

All bone mass information is presented in table 6.3. Total Body BMC and bone area are presented both including and excluding the head information. No significant differences were found between the groups for any variable, nor were any differences reported within the experimental group between trials. In contrast the control group showed a significant increase in L2 – 4 bone mineral content, representing a 2% increase in lumbar spine BMC. This finding was rendered insignificant when analysed with a repeated measures ANOVA with age, height and lean mass included as covariates although a trend toward significance remained ($p = 0.060$).

Table 6.3: Bone Mass Characteristics – Intention to Treat Analysis

	VTG		Control	
	n = 9		n = 10	
	Baseline	Retrial	Baseline	Retrial
TBBMD (g·cm⁻²)	1.145 ± 0.065	1.150 ± 0.061	1.184 ± 0.078	1.188 ± 0.088
TBBMC (g)	2618 ± 341	2619 ± 336	2836 ± 424	2837 ± 428
TB BMC (g)				
(minus head)	2175 ± 313	2170 ± 307	2353 ± 393	2351 ± 389
TBBA (cm²)	2279 ± 200	2271 ± 191	2386 ± 205	2377 ± 183
TBBA (cm²)				
(minus head)	2050 ± 191	2046 ± 180	2152 ± 201	2140 ± 179
Spine BMD (g·cm⁻²)	1.133 ± 0.074	1.119 ± 0.089	1.194 ± 0.152	1.195 ± 0.150
Spine BMC (g)	50.48 ± 8.11	50.75 ± 8.35	54.55 ± 10.4	56.4 ± 11.02*
Spine BA (cm²)	44.73 ± 4.7	45.29 ± 4.37	45.5 ± 4.15	47.03 ± 3.86
FN BMD (g·cm⁻²)	1.062 ± 0.102	1.062 ± 0.106	1.097 ± 0.129	1.090 ± 0.129
FN BMC (g)	5.57 ± 0.82	5.57 ± 0.85	5.85 ± 0.93	5.76 ± 0.93
FN BA (cm²)	5.23 ± 0.39	5.22 ± 0.45	5.33 ± 0.42	5.30 ± 0.37

*Data presented as mean ± SD, * p < 0.05 from baseline within groups.*

Absolute and percent bone mass changes are presented in Table 6.4. Percent change was calculated as the increase/decrease in the bone variable from baseline to retrial. No significant differences were shown between trials for any variable. No mean percent change for any variable reached the calculated least significant change (LSC) as indicated in the methodology section.

Table 6.4: Bone Mass - Absolute and Percentage Change

	Absolute Change		Percent Change	
	n = 9		n = 10	
	VTG	Control	VTG	Control
TBBMD (g·cm⁻²)	0.004 ± 0.01	0.004 ± 0.018	0.4 ± 1.14	0.37 ± 1.4
TBBMC (g)	1.67 ± 41.62	0.9 ± 57.23	0.1 ± 1.5	0.04 ± 2
TB BMC (g)	-4.56 ± 34	-2 ± 52.77	-0.17 ± 1.45	-0.45 ± 2.24
(minus head)				
TBBA (cm²)	-7.4 ± 33.55	-9.6 ± 41.45	-0.29 ± 1.5	-0.33 ± 0.17
TBBA (cm²)	-4.56 ± 31.26	-11.9 ± 43.5	-0.17 ± 1.58	-0.47 ± 1.92
(minus head)				
Spine BMD (g·cm⁻²)	-0.014 ± 0.04	0.001 ± 0.29	-1.23 ± 3.5	0.14 ± 2.62
Spine BMC (g)	0.27 ± 1.69	1.24 ± 2.03	0.54 ± 3.5	2.06 ± 3.23
Spine BA (cm²)	0.56 ± 0.86	0.89 ± 1.95	1.36 ± 2.16	1.93 ± 4.14
FN BMD (g·cm⁻²)	-0.002 ± 0.16	-0.007 ± 0.11	-0.046 ± 1.55	-0.60 ± 1.03
FN BMC (g)	-0.004 ± 0.082	-0.09 ± 0.17	-0.157 ± 1.487	-1.583 ± 3.36
FN BA (cm²)	-0.013 ± 0.12	-0.026 ± 0.096	-0.3 ± 2.19	0.42 ± 1.79

*Data presented as mean ± SD, * p < 0.05 from baseline within groups*

6.4.4.3. Bone Turnover

Bone turnover markers (urinary NTx/creatinine and serum P1NP) and an uncoupling index (UI) between the markers of bone formation and resorption are presented in Table 6.5. No significant differences between trials were identified in the experimental group. One participants bone resorption results were excluded from the analysis as they represented a significant outlier for this variable (Z score of urinary NTx difference between trials = 3.1, p < 0.01) (Field, 2005) and so an uncoupling index could not be calculated for this individual. Serum P1NP showed a significant decrease between trials in the control group and a trend toward significance was identified for control within group urinary NTx levels (p = 0.057) but not NTX corrected for creatinine. No differences were identified for the uncoupling index either between or within groups.

Table 6.5: Indices of Bone Turnover

	VTG (n = 10)		Control (n = 9)	
	Baseline	Retrial	Baseline	Retrial
NTx (mmol l⁻¹)	1184 ± 591	1215 ± 574	1665 ± 638	1332 ± 468
NTx/creatinine (nmol mmol⁻¹)	66 ± 21	65 ± 26	77 ± 20	70 ± 23
P1NP (ng ml⁻¹)	89.9 ± 57.8	72 ± 33.5	94.7 ± 37.5	66.6 ± 23*
UI NTX	-0.05 ± 0.53	-0.4 ± 0.8	0.0 ± 0.5	0.0 ± 1.2
UI NTX/creatinine	-0.02 ± 0.4	-0.45 ± 0.65	0.0 ± 1.0	0.0 ± 1.0

*Data presented as mean ± SD, * p < 0.05 from baseline within groups*

6.4.4.5. Individual Subject Data: Per Protocol Analysis

Figures 6.3 – 6.5 represent individual BMD percent change values for all VTG subjects. The first bar represents mean control percent change ± SD for each variable. Subject values are presented in ascending order according to compliance in minutes of vibration treatment per month, i.e. subject 1 (S1) represents that participant with lowest compliance, while subject 9 (S9) represents that participant with the highest compliance. Individual total body; L2 – 4 and femoral neck BMC and BA change values are presented in Appendix E.

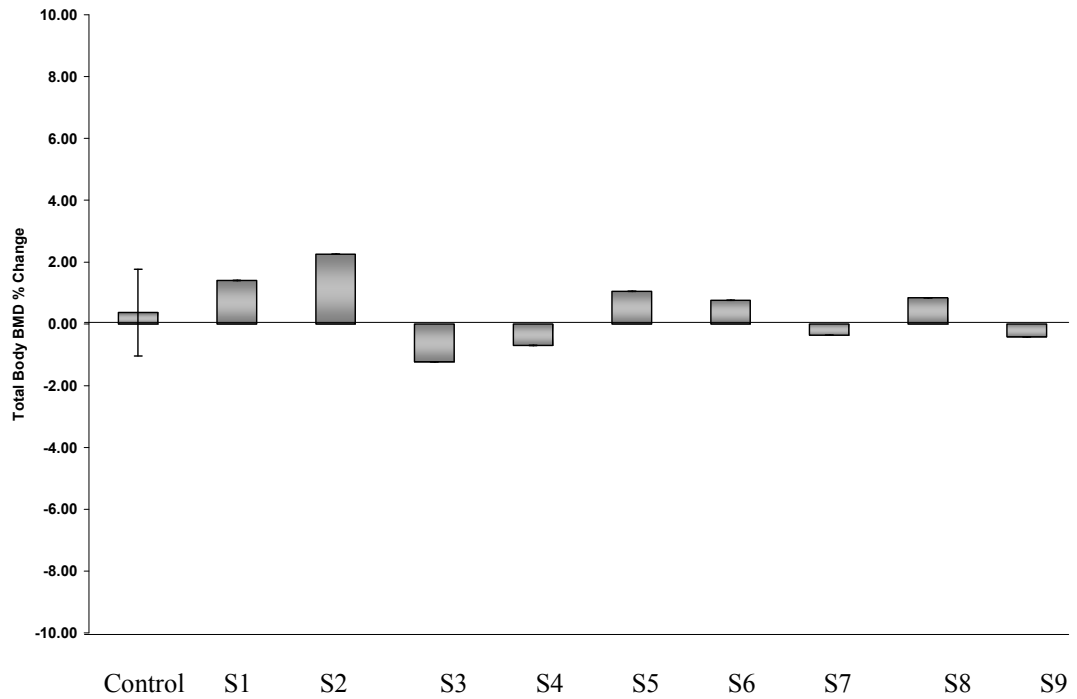


Figure 6.3: Total Body BMD Individual % Change

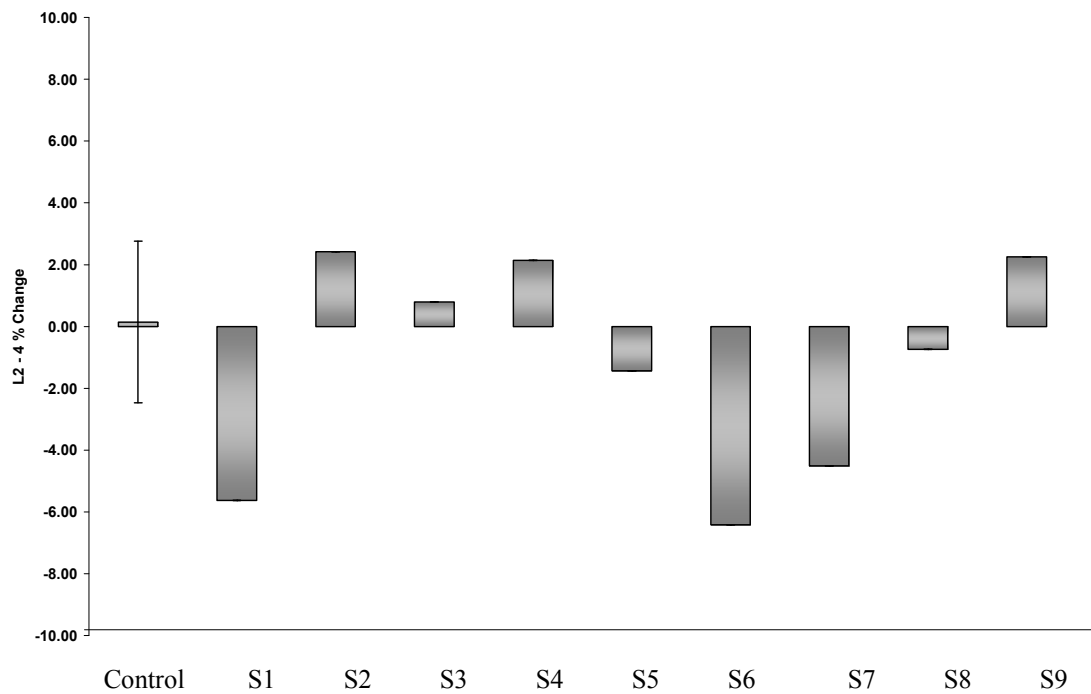


Figure 6.4: L2 – 4 BMD Individual % Change

Examination of individual data revealed that three participants exceeded the calculated least significant change (LSC = 4%) for lumbar spine BMD in a negative direction (VTG n = 2 (S1 and 6); Control n = 1), showing a significant decrease in bone density in these participants. Both of the VTG participants (S1 & 6) fell within the lowest quartile for intervention compliance, displaying 16 and 55 minutes per month respectively.

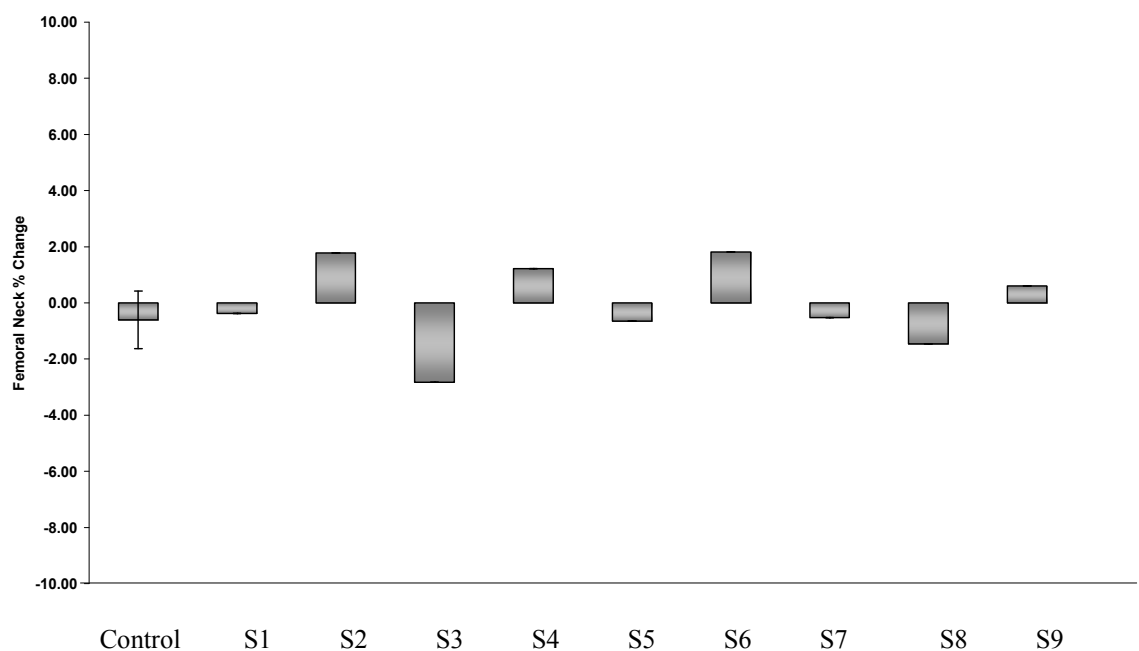


Figure 6.5: Femoral Neck BMD Individual % Change

No subject exceeded the LSC for femoral neck BMD (7%) in either a positive or negative direction.

6.5. Discussion

Results from this study do not support the use of WBV therapy as an osteogenic intervention for this group of jockeys. Bone mass and turnover was shown to be unaffected after six months in the WBV group. Poor compliance and a high attrition rate may have affected interpretation of data and masked potential intervention effects. Examination of individual response relative to compliance in the per protocol analysis did not however reveal any trends towards change in bone mass in those showing greatest adherence to study protocol.

An integral factor regarding the effectiveness of any therapeutic intervention is subject compliance to that intervention. Over the course of this intervention there was a high attrition rate with 29 % (n=8) of the initial volunteers dropping out prior to study completion. Of those remaining nine subjects who completed the vibration training intervention six fell within the lowest compliance quartile, while one participant fell within each of the 2nd, 3rd and 4th quartile for compliance. This was observed despite regular telephone contact and review, intended to promote compliance. The literature surrounding WBV suggests that intervention effect is strongly dependent on compliance (Gilsanz et al., 2006; Rubin et al., 2004). Recent evidence appears to indicate however that the 20 minutes/day⁻¹ recommended by manufacturers may be unnecessary as it appears that biological response to WBV is triggered and not accumulated (Gilsanz et al., 2006). Furthermore, the authors suggested that as little as two minute's exposure per day may be sufficient to invoke an osteogenic response. It is possible that the recommended 20 minutes per day may have been too much for a busy professional athlete such as a jockey to fit into already demanding daily schedule and a greater response may have been achieved if participants were instructed to use the platform for a shorter amount of time. The intensive protocol used here may have proved too daunting for study participants and may actually have discouraged optimum adherence. The poor compliance and high attrition rate may represent the most relevant finding of this study as it appears to indicate that this form of intervention may not be suitable for this group.

While poor compliance is likely to have been a contributory factor to the lack of change reported in this study, examination of individual cases did not appear to show

any increased trends towards change when data were considered in accordance with compliance (see figures 6.3 – 6.5). Much of the research regarding the osteogenic potential of WBV in human subjects has focused on those whose physical capabilities were impaired in some way, be it through age (Rubin et al., 2006; Xiang Yan et al., 2008) or disability (Ward et al., 2004), although an osteogenic benefit has been suggested as having occurred in a group of young women with low BMD (Gilsanz et al., 2006). In contrast, WBV has previously been shown to have no effect on bone mass, quality or turnover in a group of young healthy males (Torvinen et al., 2003). It is possible that a greater stimulus may be required in this specific population comprising young athletic males who despite having apparently low bone mass (Warrington et al., 2009) are otherwise highly physically active (Dolan et al., 2008). Provision of a greater mechanical signal, be it through an increase in either magnitude or frequency, may provide a stronger osteogenic stimulus, so invoking a greater response. Alternatively the use of WBV while incorporating resistance exercise as has previously been shown to augment bone mass during bed rest achieved immobilization (Ambrecht et al., 2009) may provide a greater benefit to these athletes.

Results from this research project appear to indicate that the low bone mass in this group may have occurred as a result of chronic energy deficiency which may have induced catabolism within the body. Dietary and nutritional interventions may therefore provide a greater osteogenic benefit to this group. Despite evidence of low gravitational forces associated with horse-racing (Cullen et al., 2009) the highly physically active lifestyle of jockeys should provide sufficient mechanical stimulation to maintain bone, and so it is likely that nutritional and hormonal factors may be the more relevant limiting factors to consider.

In contrast to the VTG, the control group appeared to show a 2% increase in spinal BMC throughout the experimental period. The significance of this finding disappeared however when the confounding influences of age, height and lean mass were considered, although a trend toward significance remained. The calcium supplementation provided to this group may have had a role to play in this finding. Calcium may provide an osteogenic benefit in one of two ways. Calcium is the major mineral contained within bone and supplementation may ensure that sufficient substrate is available for the process of bone remodelling. In addition, diets low in fruit

and vegetable intake, as has been suggested in this group (Dolan et al., 2010) may represent a threat to bone as alkaline salts may be leached from the bone in order to preserve blood pH neutrality (Sebastian et al., 1994). Calcium supplementation may provide alkaline neutralization and so benefit bone in this way. Changes in lumbar spine BMC did not however reach the calculated least significant change (4%) (Bonnick et al., 2001). In addition, the same supplement was provided to both the experimental and control group and any true intervention effect should have affected both groups. Further research may be required so to more fully evaluate the effects of calcium supplementation on bone health in this population.

Somewhat paradoxical to the finding of apparently increased spinal BMC was evidence that serum levels of P1NP appeared to significantly decrease in the control group throughout the experimental period, demonstrating an apparent reduction in bone formation. If the trend toward significance in the uncorrected urinary NTX levels is taken to be relevant this may be indicative of decreased bone turnover and a possible preservation of bone mass. These findings in the control group were unexpected however and the somewhat tenuous explanations proposed here require further investigation.

6.6. Limitations

A number of limitations are present in this study which may have influenced study findings and interpretation of results. The high attrition rate and poor compliance in some ways may be viewed as a result on their own in relation to intervention suitability within this population of jockeys; however they do render interpretation of physiological effects difficult and prone to error. Nutritional and physical activity habits have been identified as the main modifiable lifestyle factors involved in bone regulation (Bass et al., 2005). These factors were not measured at baseline and retriial however and so it is unknown whether changes in these factors may have had a role to play in study findings. Both experimental and control groups were supplemented with calcium as suggested in similar studies (Gilsanz et al., 2006) and so a true control group against which to measure results was absent in this study. As previously discussed, DXA scanning is incapable of fully accounting for changes in body size and volumetric bone density (Prentice et al., 1994). It is possible that this method of assessment may not be sensitive enough to identify subtle changes in bone mass and quality and WBV effects have previously been shown to exist when analysed by QCT but not DXA (Gilsanz et al., 2006).

6.7. Conclusions

In conclusion results of this study do not support the efficacy of WBV as an osteogenic device for this group of jockeys. Poor compliance rendered study findings difficult to interpret however and contributed to the inconclusive results shown. An apparent increase in L2 – 4 BMC in the control group may have occurred as a result of the calcium supplement supplied and further research may be required so to assess the effect of this. Low bone mass in jockeys remains a major challenge to the racing industry and examination of alternative osteogenic interventions may be required. The more fundamental issue to consider however may be the appropriateness of current weight standards and the methods typically used to achieve them. It appears likely that it is these issues which may have impacted on bone mass in jockeys. Primary prevention may therefore be the more appropriate intervention to consider.

Chapter Seven

Summary, Conclusions and Recommendations for Further Research



7.1. Summary

The aim of this study was to examine the effects of a weight restrictive lifestyle on a numbers of parameters of physiological; osteogenic; metabolic and cognitive function in jockeys. This was achieved through the implementation of four separate, though related experiments as part of the overall study design. Results from this research suggest that many aspects of physiological, osteogenic and metabolic function are indeed affected in jockeys in comparison to healthy age and BMI matched male controls as well as other weight category athletes. It appears likely that the main findings reported which included increased physiological strain and reduced aerobic exercise capacity when “at weight”; low bone mass; a high rate of bone turnover and disruptions to a number of endocrine factors involved in the processes of growth and reproduction may have occurred as a result of the physiological stress imposed on the body through the repeated pressure of attaining the stipulated weights and remaining at weight throughout the protracted racing season.

Study One examined the acute impact of a four % reduction in body mass within 48 hours on physiological and cognitive function. Participants were instructed to reduce their body mass “using what means you usually would for racing” and protocol for this study was based on the results of a self report questionnaire. This study was designed in order to reflect the actual demands of making weight for racing. Results showed a clear impairment to physiological function, as assessed through performance on an incremental cycle ergometer test to volitional fatigue. This was demonstrated by a significant decrease in peak power output achieved, and an increase in the level of submaximal cardiovascular strain experienced throughout the test. No such detriments to cognitive function were observed, as assessed through tests of motor response, decision making, executive function and working memory. Assessment of urine specific gravity, and a food diary maintained throughout the weight loss period suggest that a hypo-hydrated and energetic state may have contributed to the acute detriments to physiological function observed within this study.

A unique challenge facing jockeys is the length and intensity of the current racing season, which provides little respite from the rigours of making weight. Study one showed clearly negative acute effects of “making weight”. Habitual experience of this

acute physiological stressor however was thought likely to have an impact on more chronic aspects of physiological and metabolic function. Studies three and four tested this hypothesis, through an examination of bone mass and osteogenic function in jockeys. Study two examined bone mass in a group of flat and national hunt jockeys in comparison with another group of weight category athletes (elite amateur boxers) and age, gender and BMI matched controls. All data were analysed in accordance with relevant aspects of body composition in an attempt to control for their potential confounding influences. The results clearly demonstrated that bone mass was reduced in both jockey groups at a number of sites, namely total body BMD; total body BMC (minus the head); L2 – 4 BMD and femoral neck BMD and BMC. Normalisation of data for lean mass and stature partly accounted for some of the variance in bone mass reported, but it was clear that additional factors were present which appeared to reduce bone mass in the flat jockey group and enhance it in the boxer group. It is thought that gravitational, nutritional and hormonal factors may in part account for the variation in bone mass demonstrated here.

Results from study two clearly indicated that bone mass was reduced in jockeys. It seemed likely that the apparent energy deficit habitually experienced by jockeys may have been involved in this finding. In order to more fully explore this theory a number of endocrine factors related to reproduction and metabolism were examined in relation to bone mass and turnover in a group of jockeys and age and BMI matched male controls. Results showed a clear disruption to aspects of endocrine function. In particular, the rate of bone turnover appeared to be elevated in the jockey group, as evidenced by significantly higher levels of NTx in the urine. Sex hormone binding globulin, the principal regulator of the male and female sex hormones was higher in the jockey group, and the growth factor IGF-1 showed a trend toward being significantly lower in the jockey group ($p = 0.07$). Examination of these variables in relation to bone mass identified SHBG and IGF-1 as significant and independent predictors of bone mass in this group. It was thought that disturbances to reproductive and anabolic processes within the jockey group may have occurred in an attempt to conserve available energy for more immediate and essential processes and to indicate a potentially catabolic state within the jockeys studied.

Low bone mass is associated with an increase in fracture susceptibility and it is proposed that low bone mass, and an apparently increased rate of bone loss may have particular implications for this group. In this context, the appropriateness of current weight standards and the methods used to achieve them must be reviewed as a matter of priority. In the interim however, appropriate osteogenic interventions may be required so to aid in the enhancement of bone mass of jockeys currently involved in this sport. Consequently, study four examined the effect of a six month whole body vibration therapy intervention on bone mass and turnover in a group of jockeys. Results did not support the efficacy of this intervention as a potential osteogenic stimulus in this population. No differences in any aspect of bone mass or turnover were identified within the vibration therapy group. Poor compliance and a high attrition rate rendered study findings difficult to interpret however and may have contributed to the inconclusive results demonstrated here.

The primary aim of this study was to assess whether acute and chronic aspects of physiological, metabolic, osteogenic and cognitive function were affected by the chronically weight restricted lifestyle chronically followed by these athletes. Results suggest that both acute and chronic aspects of physiological, metabolic and osteogenic function are indeed altered in this group. Acute impairments to physiological function as evident following a four % reduction of body mass within 48 hours may have had a role to play in the alterations to metabolic and osteogenic function identified in studies two and three. In addition, it appears that disruptions to endocrine function, as evidenced by alterations to circulating levels of many key reproductive and metabolic hormones, may have had a role to play in the reduced bone mass which appears to be commonplace within this group of athletes. Further research may be required so to analyse these findings and their implications in greater depth.

7.2. Recommendations for Future Research

Results from this research display clear disruptions to aspects of physiological, metabolic and osteogenic function in jockeys and it is likely that this occurred in response to habitual weight cycling and repeated bouts of acute weight loss. Further research is required however so to more fully explore this area and aid in the development of sport specific recommendations and strategies designed to combat this problem. In particular examination of the specific physiological and biomechanical demands of racing may allow sport specific recommendations regarding appropriate nutritional and training strategies be made. In relation to the specific studies conducted within this research project, a number of questions remained unanswered and may warrant further scientific attention.

Although a clear impairment to physiological function was demonstrated in study one, as assessed through performance on a maximal incremental cycle ergometer test to volitional fatigue further research may be required so to assess whether similar detriments occur to more sport specific performance variables. These include racing specific tests on a racehorse simulator; tests of anaerobic function; assessment of balance and coordination and a more in depth psychological analysis, to include assessment of emotive as well as cognitive factors. Identification of the threshold of body mass loss at which performance decrements occur, as well as examination of different weight loss practices may aid in the development of more appropriate weight management strategies for this group. Blood analysis of appropriate endocrine factors when in a weight reduced state may shed further light on mechanisms of enhanced physiological strain and may also contribute to elucidation of other results reported within this study, including low bone mass and disrupted reproductive and anabolic function.

Studies two and three clearly demonstrated reduced bone mass in the jockey group and an apparently increased rate of bone turnover. This appeared to occur at least in part through disruptions to reproductive and anabolic processes. Further analysis, including maintenance of physical activity and nutritional records, information regarding pubertal onset and examination of a broader range of endocrine factors, including the IGF binding proteins, corticosteroid binding globulin and factors involved in the regulation

of energy balance such as leptin and adiponectin may provide additional information regarding endocrine disturbances in this group and the effect which this may have on osteogenic function in this group. Examination of the pulsatile release of key hormones via frequent sampling over a 24 hour period would be useful so to more fully examine potential endocrine disturbances in this group.

Although the WBV intervention employed in this research project was not shown to be a suitable osteogenic intervention in this group, findings of disrupted osteogenic function from studies two and three remain and as such identification of alternative interventions may be required. An alternative protocol of WBV may be useful. A shorter duration of treatment bout may encourage compliance, while an enhanced strain, be it through an increase in magnitude or frequency may elicit a greater osteogenic adaptive response. Nutritional interventions may also be of benefit in this group.

Results from this thesis clearly demonstrate that the challenge of “making weight” and remaining at weight throughout the protracted season may have many negative connotations for jockeys. A more flexible and scientifically validated handicapping system may be required in order to reduce the strain placed upon these athletes. For example individualised minimum weight standards, determined through analysis of a number of parameters may be useful, although there are numerous methodological and logistical difficulties associated with this approach. Further research to more fully evaluate the challenges associated with making weight in horse-racing, and related effects on physiological, endocrine, osteogenic and psychological function may enable the development of more appropriate legislation and should be undertaken as a matter of priority.

7.3. Study Implications and Conclusion

Making weight and remaining at weight throughout the prolonged racing season represents a major challenge to the jockeys who compete in this sport. The original aim of this study was to examine the impact of acute and chronic effects of weight restriction and weight regulation practices typically adopted by jockeys on physiological, metabolic, osteogenic and cognitive function. Results from this thesis suggest that both acute and chronic aspects of physiological, osteogenic and metabolic function are negatively affected in this group. Such findings may be considered to represent a major health and safety concern to the racing industry. Horse-racing is a high risk activity, with a frequent occurrence of falls and high injury rate. Findings from this study, including reduced bone mass; high levels of dehydration and impaired physiological function may intensify the short term risks associated with participation in this sport. In addition the finding of low bone mass, a high rate of bone turnover and apparently altered metabolic function may have potentially severe long term consequences for this unique population. Scientific evidence strongly supports a move to a more flexible, regularly revised weight handicapping system and ongoing education about healthy eating, physical fitness and the risks associated with chronic energy restriction and making weight. International collaboration and the phased implementation of such strategies are necessary to ensure that the international competitive nature of the horse-racing industry can accommodate these changes in the interest of jockeys long-term health and wellbeing.



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Appendices

Appendix A:
Informed Consent Forms

Appendix B:
Medical History Form

Appendix C:
Making Weight Questionnaire

Appendix D:
Study One: Supplementary Results

Appendix E:
Study Four: Supplementary Results

Appendix A



DUBLIN CITY UNIVERSITY
Informed Consent Form

Title:

The Effects of making weight on physiological and cognitive function in professional horse-racing jockeys

Investigators:

Dr. Adrian McGoldrick
Dr. Giles Warrington,
Eimear Dolan

Introduction to the Study:

The purpose of this study is to examine the effect which making weight may have on the physical, and cognitive capacities of top level Irish Jockeys.

Participant Requirements:

You will perform an exercise test on a bike, to assess your aerobic fitness levels before and after making a weight. For the test the exercise intensity will begin at a low level and will be advanced in stages depending on your fitness level. We may stop the test at any time because of signs of fatigue or changes in your heart rate, or blood pressure, or symptoms you may experience. It is important for you to realise that you may stop when you wish because of feelings of fatigue or any discomfort. During the test you will wear a facemask, through which the air inhaled and exhaled is analysed. You will also complete a computer based cognitive function test, and provide blood and urine samples. You will then be given 2 days to reduce your weight by 4% before returning to RACE to complete all tests.

Participant Responsibilities:

Information you possess about your health status or previous experiences of heart-related symptoms (such as shortness of breath with low-level activity, pain, pressure, tightness, heaviness in the chest, neck, jaw, back and/or arms) with physical effort may affect the safety of your exercise test. Your prompt reporting of these and any other unusual feelings with effort during the exercise test itself is of great importance. You are responsible for fully disclosing your medical history, as well as symptoms that may occur during the test. You are also expected to report all medication (including non-prescription) taken recently and, in particular, those taken today, to the testing staff.

Potential risks to participants from involvement in the Research Study:

You may experience some muscle soreness in my legs or nausea following the maximal exercise test. Exercise testing carries with it a very small risk of abnormal heart rhythms, heart attack, or death. The pre-test likelihood of these risks in young, healthy males with no known history of heart disease is very low.

Benefits (direct or indirect) to participants from involvement in the Research Study:

The results obtained from the exercise test may assist in the evaluation of your physical conditioning and provide feedback for future training. They may also be used in the diagnosis of illness or evaluate the effect of medications. You will receive a full report detailing your personal results on both pre and post tests.

Participant – please complete the following (Circle Yes or No for each question)

Do you understand the purpose and requirements of this study?

Yes/No

Have you had an opportunity to ask questions and discuss this study?

Yes/No

Have you received satisfactory answers to all your questions?

Yes/No

Confirmation that involvement in the Research Study is voluntary

If I do agree to take part in this study I may withdraw at any point. There will be no penalty if I withdraw before I have completed all stages of the study, however once I have completed the study I will not be able to remove my personal information or results from the database or study reports.

Advice as to arrangements to be made to protect confidentiality of data, including that confidentiality of information provided is subject to legal limitations

My identity and other personal information will not be revealed, published or used in further studies. I will be assigned an ID number under which all personal information will be stored in a secure file and saved in a password protected file in a computer at DCU. Only listed investigators and the project medical coordinator will have access to the data.

Confidentiality is insured, but I must be aware that confidentiality of information provided can only be protected within the limitations of the law - i.e., it is possible for data to be subject to subpoena, freedom of information claim or mandated reporting by some professions.

VII. Signature:

I have read and understood the information in this form. My questions and concerns have been answered by the researchers, and I have a copy of this consent form. Therefore, I consent to take part in this research project

Participants Signature: _____

Name in Block Capitals: _____

Witness: _____

Date: _____



DUBLIN CITY UNIVERSITY
Informed Consent Form

Title:

“The effects of vibration therapy at improving bone health in Irish jockeys”

Investigators:

Dr. Giles Warrington,
Dr. Adrian McGoldrick,
Ms. Eimear Dolan

Introduction to the Study:

The purpose of this study is to examine the effects of vibration therapy at improving the bone health of Irish jockeys. Bone health will be examined both before and after a vibration therapy intervention, and any changes in bone health will be used to assess the effectiveness of the vibration therapy intervention. This research is being supported by an unrestricted grant from the Irish Turf Club to Dr. Giles Warrington at Dublin City University

Participant Requirements:

1. I will report to the Exwell Medical Centre in the DCU sports grounds where I will receive a free DEXA scan which will examine the strength of my bones.
2. I will provide a blood and urine sample to allow examination of biochemical markers of bone turnover. These samples must be provided in a fasted state in the morning. A time will be arranged for this which is convenient for myself and the investigators.
3. If selected I will be provided with a vibration platform for use in my own home for a period of 6 months. It is thought that this platform will work best if combined with calcium supplementation. I will be provided with these supplements for the duration of the study.

Potential risks to participants from involvement in the Research Study:

I understand that DEXA Scanning involves exposure to a small amount of x-ray radiation, however this is a routine assessment. There is a possibility that I may feel a slight pain when the needle is inserted into my arm for the blood sample, and I may develop a bruise where the blood sample is obtained. The pain and bruising is usually mild and a medically trained person will obtain my blood. The amount of blood drawn is not harmful, however if I have a history of anaemia I should inform the investigator.

Benefits (direct or indirect) to participants from involvement in the Research Study:

I will receive a free DEXA scan which will allow me to better understand my bone health. I will receive valuable advice and assistance as to how to increase my bone strength. If chosen to undergo the intervention I will receive a free vibration platform and calcium supplements to aid in the development of my bone strength. I will also receive a full blood profile.

Participant – please complete the following (Circle Yes or No for each question)

Do you understand all the information provided here?

Yes/No

Have you had an opportunity to ask questions and discuss this study?

Yes/No

Have you received satisfactory answers to all your questions?

Yes/No

Confirmation that involvement in the Research Study is voluntary

I understand that I am taking part in this study and may withdraw at any point should I wish. There will be no penalty if I withdraw before I have completed all stages of the study; however once I have completed the study I will not be able to remove my results from the database or study reports.

Advice as to arrangements to be made to protect confidentiality of data, including that confidentiality of information provided is subject to legal limitations

My identity and other personal information will not be revealed, published or used in further studies. I will be assigned an ID number under which all personal information will be stored in a secure file and saved in a password protected file in a computer at DCU. Listed investigators only will have access to the data.

Confidentiality is insured, but I must be aware that confidentiality of information provided can only be protected within the limitations of the law - i.e., it is possible for data to be subject to subpoena, freedom of information claim or mandated reporting by some professions.

VII. Signature:

I have read and understood the information in this form. My questions and concerns have been answered by the researchers, and I have a copy of this consent form. Therefore, I consent to take part in this research project

Participants Signature:

Name in Block Capitals:

Witness:



DUBLIN CITY UNIVERSITY
Informed Consent Form

Title:

The Relationship Between Bone Mass, Body Composition and a Number of Metabolic Markers in Jockeys and Age and BMI Matched Controls

Investigators:

Ms. Eimear Dolan
Dr. Giles Warrington,
Dr. Adrian McGoldrick,

Introduction to the Study:

The purpose of this study is to examine differences between racing jockeys and controls regarding bone mass, body composition and a number of energy regulating and metabolic hormones.

Participant Requirements:

4. I will report to the Santry Sports Surgery Clinic in the Northwood Business Park where I will receive a free DEXA scan which will examine the strength of my bones.
5. I will provide a blood and urine sample to allow examination of biochemical markers of bone turnover. These samples must be provided in a fasted state in the morning. A time will be arranged for this which is convenient for myself and the investigators.

Potential risks to participants from involvement in the Research Study:

I understand that DEXA Scanning involves exposure to a small amount of x-ray radiation, however this is a routine assessment. There is a possibility that I may feel a slight pain when the needle is inserted into my arm for the blood sample, and I may develop a bruise where the blood sample is obtained. The pain and bruising is usually mild and a medically trained person will obtain my blood. The amount of blood drawn is not harmful, however if I have a history of anaemia I should inform the investigator.

Benefits (direct or indirect) to participants from involvement in the Research Study:

I will receive a free DEXA scan which will allow me to better understand my bone health. I will receive valuable advice and assistance as to how to increase my bone strength.

Participant – please complete the following (Circle Yes or No for each question)

Do you understand all the information provided here?
Yes/No

Have you had an opportunity to ask questions and discuss this study?

Yes/No

Have you received satisfactory answers to all your questions?

Yes/No

Confirmation that involvement in the Research Study is voluntary

I understand that I am taking part in this study and may withdraw at any point should I wish. There will be no penalty if I withdraw before I have completed all stages of the study; however once I have completed the study I will not be able to remove my results from the database or study reports.

Advice as to arrangements to be made to protect confidentiality of data, including that confidentiality of information provided is subject to legal limitations

My identity and other personal information will not be revealed, published or used in further studies. I will be assigned an ID number under which all personal information will be stored in a secure file and saved in a password protected file in a computer at DCU. Listed investigators only will have access to the data.

Confidentiality is insured, but I must be aware that confidentiality of information provided can only be protected within the limitations of the law - i.e., it is possible for data to be subject to subpoena, freedom of information claim or mandated reporting by some professions.

VII. Signature:

I have read and understood the information in this form. My questions and concerns have been answered by the researchers, and I have a copy of this consent form. Therefore, I consent to take part in this research project

Participants Signature:

Name in Block Capitals:

Witness:

Date:

Appendix B



The Effects of Making Weight on Physiological and Cognitive Function in Horse-Racing Jockeys

DATE

CONTACT DETAILS)

Last name: _____

First name: _____

Date of birth: _____

Age _____

Address: _____

Mobile _____ Work: _____
Home: _____

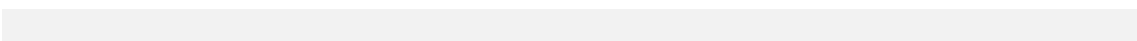
Email Address: _____

Next of kin

Name _____

Contact tel: _____

Relationship to you _____



MEDICAL HISTORY (PHYSICIAN ADMINISTERED)

1. Do you suffer from any of the following (tick box)?		Yes	I
a)	High blood pressure (hypertension)	<input type="checkbox"/>	
b)	Angina i.e. chest pain, neck pain, jaw pain, arm pain or undue breathless on exertion (such as walking fast or walking up a hill)	<input type="checkbox"/>	
c)	Heart disease of any sort e.g. heart attack blocked blood vessels to the heart abnormal heart rhythm	<input type="checkbox"/>	
d)	Peripheral vascular disease e.g. intermittent claudication (calf pain on walking) stroke	<input type="checkbox"/>	
e)	Elevated blood cholesterol or triglycerides	<input type="checkbox"/>	
f)	Diabetes	<input type="checkbox"/>	

2. Have you ever had any of the following (tick box)?		Yes	No
a)	A heart attack	<input type="checkbox"/>	<input type="checkbox"/>
b)	Heart surgery	<input type="checkbox"/>	<input type="checkbox"/>
c)	An angiogram	<input type="checkbox"/>	<input type="checkbox"/>
d)	Insertion of a stent	<input type="checkbox"/>	<input type="checkbox"/>
e)	Treatment of an irregular heart beat	<input type="checkbox"/>	<input type="checkbox"/>
f)	A blackout (loss of consciousness)	<input type="checkbox"/>	<input type="checkbox"/>

**3. Please list any other medical conditions you suffer from
at present or have suffered from in the past**

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

4. List any medications which you are now taking

5. Your family history

Do any of your first degree relatives (parents, brothers, sisters) suffer from any of the following (tick box if yes)?

	Yes	No
a) heart disease	<input type="checkbox"/>	<input type="checkbox"/>
b) high blood pressure	<input type="checkbox"/>	<input type="checkbox"/>
c) diabetes	<input type="checkbox"/>	<input type="checkbox"/>

Has any first degree relative of yours died from heart disease? Yes ☐
No ☐

6. Alcohol / Cigarettes

Do you consume alcohol regularly? Yes ☐ No ☐

If yes, how many units per week? _____

Do you smoke? Yes ☐ No ☐

If yes, how many cigarettes a day? _____

Signature: _____

Date: _____

Appendix C

Making Weight Questionnaire

This short questionnaire will take no more than 5 minutes to complete

All responses will remain strictly anonymous and confidential. Replies will be used for research purposes only, and will be analysed as group average data and not individually.

-
- **What is your typical non-racing weight that you would feel most comfortable at?**

-
- **What is your typical riding weight, i.e. the typical weight which you need to be at in order to comply with racing restrictions?**

-
- **What is the largest amount of weight which you have ever lost for a race?**

-
- **How much time are you typically given to make a weight?**

-
- **What is the least amount of notice which you have ever been given to make a weight?**
-

Thank you for taking the time to fill in this questionnaire

Eimear Dolan, Dr Adrian McGoldrick, Dr Giles Warrington

Appendix D

⇒ Study One: Jockey and Control Submaximal Data

Subject Absolute Submaximal Physiological Values

Watts	Baseline				Retrial			
	60	90	120	150	60	90	120	150
% Max Wattage	29 ± 4	43 ± 6	57 ± 8	71 ± 10	33 ± 4**	49 ± 7**	66 ± 9**	82 ± 12**
VO ₂ (L·min ⁻¹)	1.4 ± 0.6	1.69 ± 0.5	1.94 ± 0.36	2.11 ± 0.13	1.71 ± 0.7	1.95 ± 0.63	2.22 ± 0.6	2.35 ± 0.4
VO ₂ (ml·kg ⁻¹ ·min ⁻¹)	24.6 ± 9.6	28.8 ± 9	32.43 ± 7.3	35.1 ± 5.02	30 ± 11.7*	34.3 ± 11*	39.18 ± 10.14*	41.27 ± 8.89*
VCO ₂	1.45 ± 0.68	1.71 ± 0.58	1.97 ± 0.38	2.14 ± 0.32	1.64 ± 0.75	1.98 ± 0.705	2.28 ± 0.62	2.5 ± 0.43
RER	0.97 ± 0.1	0.99 ± 0.05	1.03 ± 0.1	1.02 ± 0.09	0.95 ± 0.1	1 ± 0.06	1.02 ± 0.04	1.02 ± 0.04
HR	115 ± 25	132 ± 25	147 ± 19	160 ± 15	131 ± 30	150 ± 28 ⁻¹	165 ± 24*	176 ± 12*
Lactate	2.2 ± 1.5	2.7 ± 1.5	3.7 ± 1.2	5.5 ± 1.8	2.1 ± 1.9	4.6 ± 4	6 ± 3.7 ⁻²	7.44 ± 3.2 ⁻³
RPE	9 ± 3	11 ± 3	13 ± 3	15 ± 3	11 ± 4	13 ± 3	15 ± 2	18 ± 2 ⁻⁴

**p < 0.05 between groups; **p < 0.01 between groups; *p < 0.05 between trials; **p < 0.01 between trial; ~ trend toward within group difference; ~1 p = 0.069; ~2 p = 0.063; ~3p = 0.060; ~4 p = 0.070.*

Relative Submaximal Values

% Max Workload	Baseline				Retrial			
	20	40	60	80	20	40	60	80
VO ₂ (L·min ⁻¹)	1.45 ± 0.5	1.77 ± 0.5	2.10 ± 0.27	2.52 ± 0.33	1.77 ± 0.6*	2.13 ± 0.6*	2.37 ± 0.39*	2.69 ± 0.29
VO ₂ (ml·kg ⁻¹ ·min ⁻¹)	24.8 ± 9	30.2 ± 8	35.8 ± 4.74	44 ± 4.8	31.2 ± 10*	37.5 ± 9.7**	42 ± 7.2*	49.9 ± 4.4~
VCO ₂	1.5 ± 0.7	1.78 ± 0.44	2.18 ± 0.2	2.79 ± 0.48	1.75 ± 0.7	2.17 ± 0.6*	2.52 ± 0.39*	2.9 ± 0.27
R	0.99 ± 0.08	1.02 ± 0.06	1.07 ± 0.11	1.13 ± 0.13	0.97 ± 0.09	1.02 ± 0.58	1.07 ± 0.1	1.11 ± 0.09

**p < 0.05 between groups; **p < 0.01 between groups; *p < 0.05 between trials; **p < 0.01 between trial; ~ trend toward within group difference (p = 0.079)*

⇒ Study One: Control Submaximal Data

Table 6: Control Submaximal Values (at different workloads)

Workload (watts)	Baseline				Retrial			
	60	90	120	150	60	90	120	150
% Max Workload	25 ± 2	37 ± 3	49 ± 4	61 ± 4	25 ± 2	37 ± 3	49 ± 4	61 ± 5
VO ₂ (L·min ⁻¹)	1.16 ± 0.08	1.47 ± 0.09	1.78 ± 0.2	2.16 ± 0.12	1.2 ± 0.08	1.49 ± 0.09	1.87 ± 0.12	2.2 ± 0.12
VO ₂ (ml·kg ⁻¹ ·min ⁻¹)	17.6 ± 2.7	22.24 ± 3.5	27.08 ± 5.4	32.84 ± 5.5	18 ± 2.86	22.6 ± 3.5	28.38 ± 4.64	33.81 ± 5.6
VCO ₂	1.07 ± 0.12	1.4 ± 0.11	1.78 ± 0.14	2.25 ± 0.14	1.12 ± 0.08	1.44 ± 0.12	1.87 ± 0.15	2.3 ± 0.16*
RER	0.92 ± 0.09	0.97 ± 0.09	1.01 ± 0.1	1.05 ± 0.1	0.93 ± 0.09	1.01 ± 0.1	0.97 ± 0.1	1.05 ± 0.1

	0.09	0.09	0.08	0.1	0.09	0.09	0.09	
HR	105 ± 8	121 ±	140 ±	156 ±	102 ±	117 ±	138 ±	155 ± 12
		12.6	11	16	10	8.1	9.4	
Lactate	1.7 ±	2.34 ±	3.4 ±	5.03 ±	1.63 ±	2.4 ±	3.5 ±	5.24 ± 1.6
	0.4	0.5	0.6	0.7	0.37	0.68	1.02	
RPE	9 ± 2	11 ± 2	13 ± 2	15 ± 2	8 ± 1	10 ± 1	13 ± 1	15 ± 1

⇒ Study One: Corrected Cognitive Results

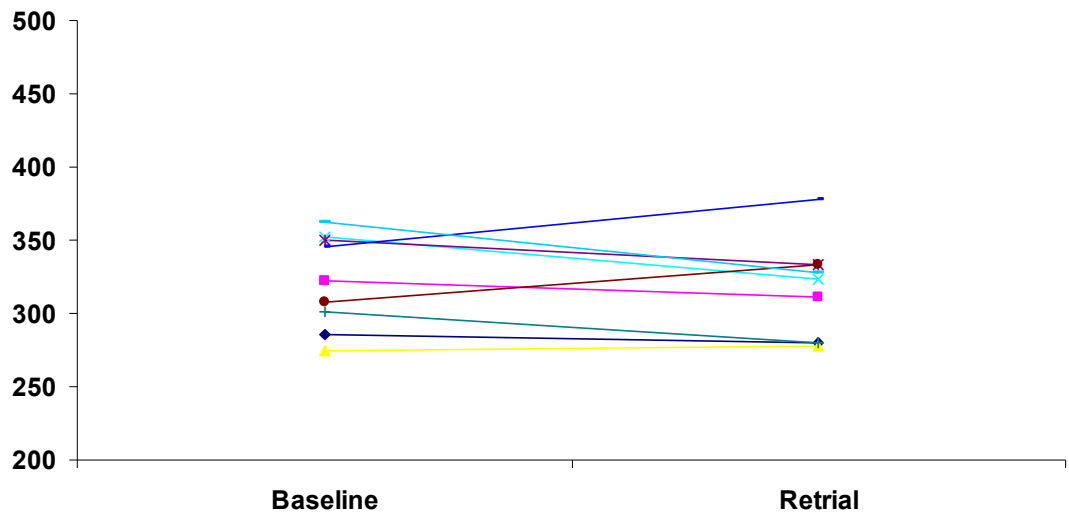
Mean Corrected Cognitive Scores

	Subjects		Controls	
	Baseline	Retrial	Baseline	Retrial
Simple Reaction Time	336.2 ± 25	342.7 ± 57.5	313.2 ± 28.4	304.6 ± 20.3
<i>SRT 1</i>	321.5 ± 21.4	320.4 ± 40.4	307.1 ± 31.9	308.3 ± 27.4
<i>SRT 2</i>	336.4 ± 28.6	333.6 ± 35.4	319.1 ± 25	302.6 ± 22.4 ^{†*}
Choice Reaction Time	466.7 ± 68.54	485.5 ± 118.4	432.6 ± 85.8	399.6 ± 82.5
CRT 1	396.2 ± 67.8	468.4 ± 66.3	386.6 ± 52.6	364.3 ± 72.7*
CRT 2	468.3 ± 56.6	435.8 ± 67.4	430.2 ± 119.1	411.7 ± 65.5
Stroop Baseline	883.8 ± 108.3	872.6 ± 73.1	806.2 ± 119.1	760.1 ± 104.7 ^{†*}
Stroop Interference	973.9 ± 160.3	996.5 ± 140.8	845.9 ± 113	811.6 ± 109.3 ^{†*}
Interference	90.1 ± 86	123.9 ± 119.1	39.6 ± 75	51.6 ± 49.5
RVIP	494.3 ± 119.2	531.6 ± 87.1	431.2 ± 99.4	474.9 ± 92

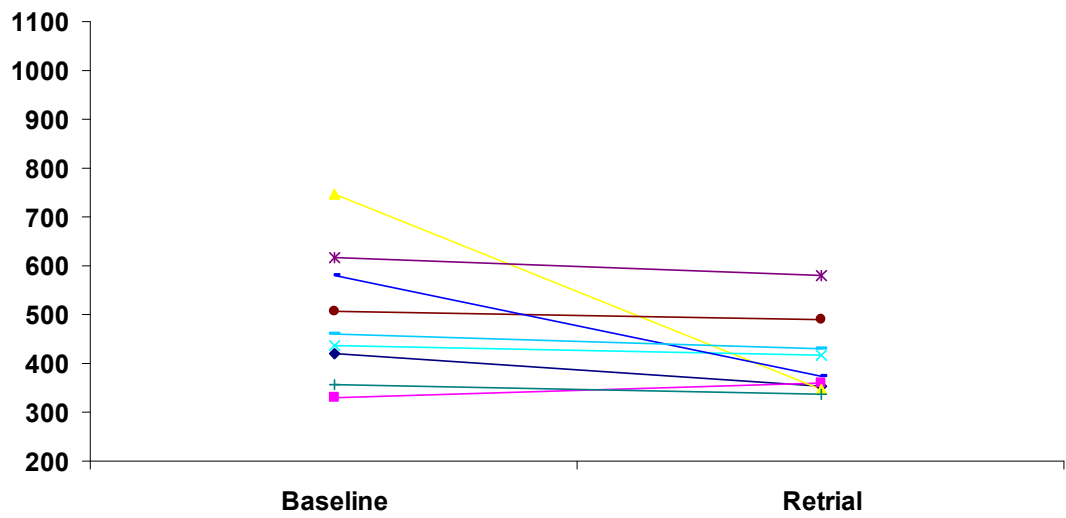
[†]*p* < 0.05 between groups, ^{††}*p* < 0.01 between groups, * *p* < 0.05 between trials, ^{††*}*p* < 0.01 between trials

⇒ Study One: Individual Cognitive Response Graphs (Controls)

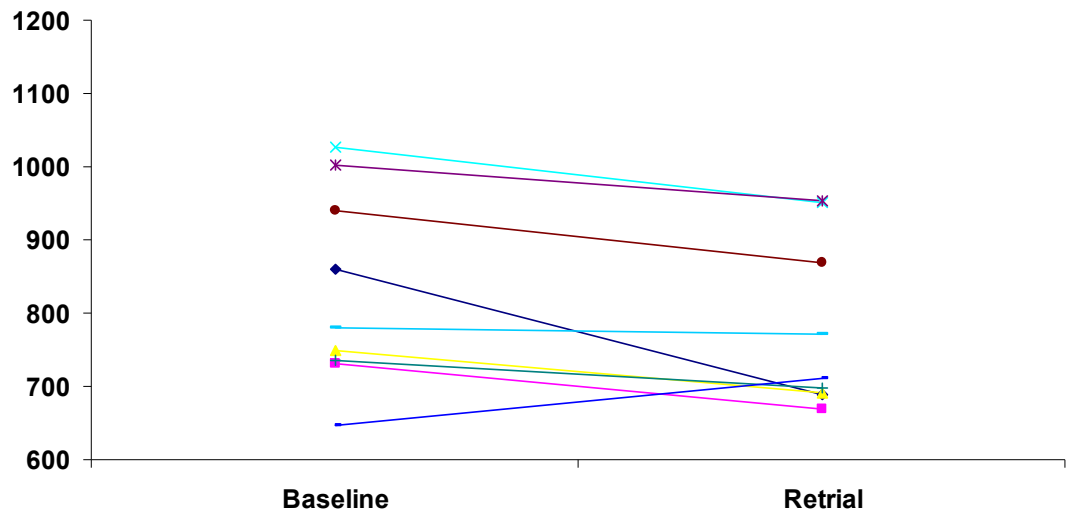
Control Simple Reaction Time



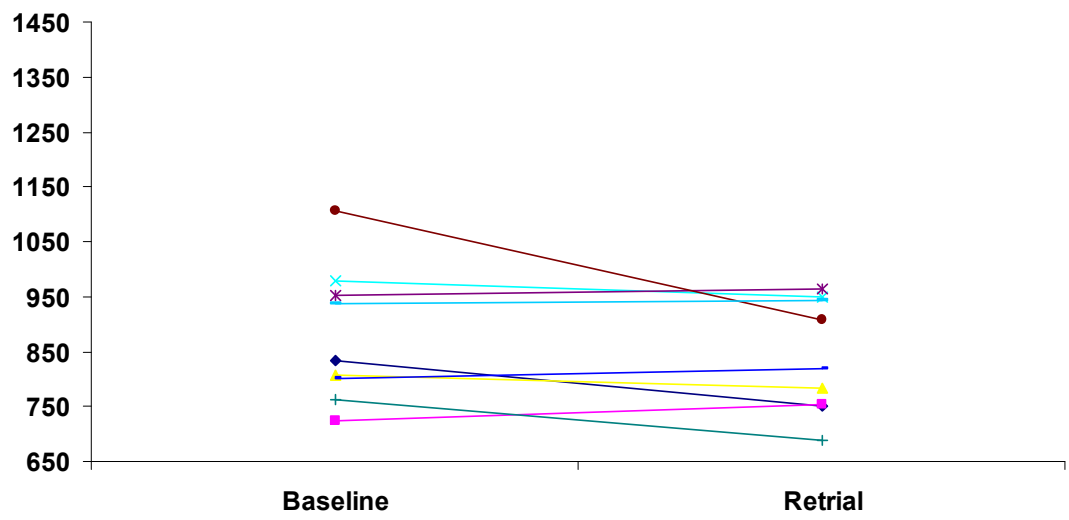
Control Choice Reaction Time



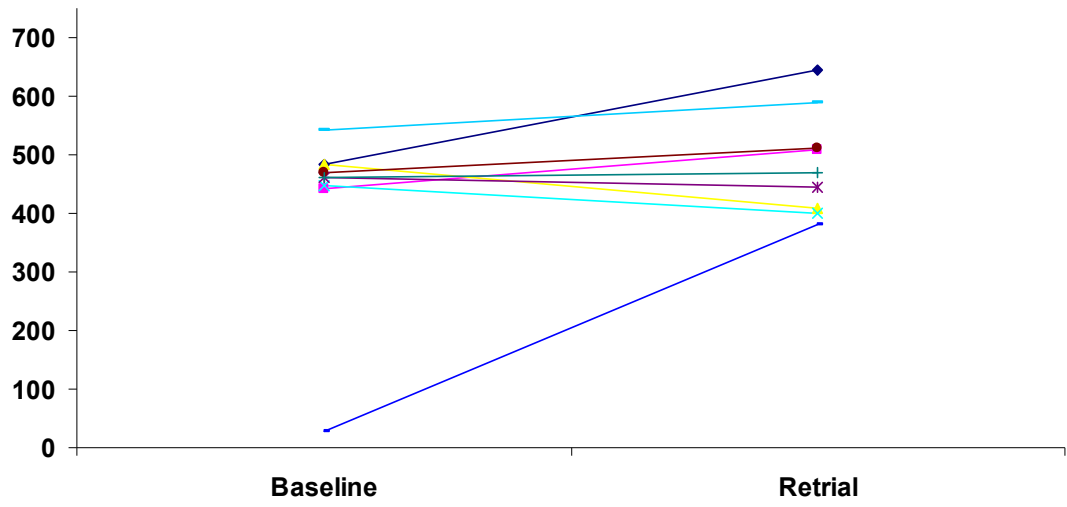
Control Stroop Baseline



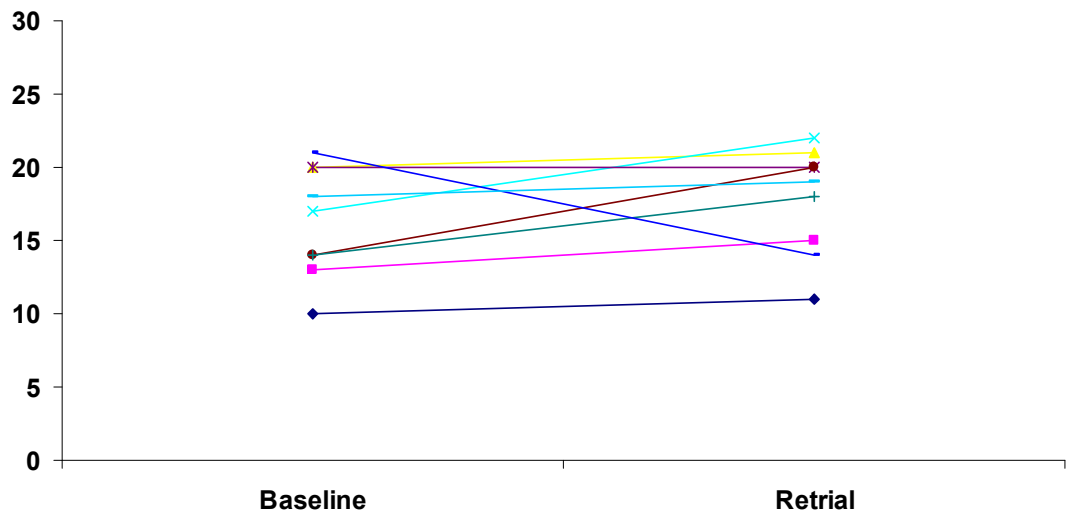
Control Stroop Interference



Control Rapid Visual Information Processing



Control RVIP Hits



⇒ Study One: Macro and Micronutrient Information

Table 3.5: Macronutrient Composition of Nutrient Intake

	Subjects			Controls
	Mean	Day One	Day Two	Mean
Total Kj	4007 ± 1492	6244 ± 2869	1771 ± 1316 [♣]	9154 ± 2471*
Total Kcal	955 ± 357	1488 ± 686	422 ± 315 [♣]	2178 ± 591*
CHO	141.9 ± 58.4	218.1 ± 121	65.6 ± 52.84 [♣]	263.9 ± 55.1*
% of Energy	45.2 ± 10.9	45.2 ± 10.9	40.2 ± 28.4	46.23 ± 7.7
Fat	36.07 ± 19	58 ± 32.3	14 ± 12.3 [♣]	91.1 ± 31.4*
% of Energy	26.6 ± 11.5	32.65 ± 8.2	20.59 ± 15.9 [♣]	36.8 ± 6
Protein	35.5 ± 9.6	55.9 ± 17.4	15 ± 10.4 [♣]	83.6 ± 28.29*
% of Energy	13.9 ± 7.2	17.09 ± 8.12	10.64 ± 8.1 [♣]	15.8 ± 3.7
g kg⁻¹	0.59 ± 0.16	0.95 ± 0.34	0.24 ± 0.17 [♣]	1.26 ± 0.52*

*Data presented as mean ± SD, * $p \leq 0.05$ between groups; ♣ $p \leq 0.05$ between days*

Table 3.6: Micronutrient Composition of Nutrient Intake

	Subjects	Controls	EAR	LTI
Potassium	1183 ± 442	2897 ± 959*	-	1600
Calcium	362 ± 163	689 ± 227*	615	430
Magnesium	96 ± 31	265 ± 78*	-	-
Phosphorus	525 ± 118	1214 ± 336*	400	300
Iron	4.06 ± 1.47	12.99 ± 3.97*	7.7	5.4***
Copper	0.39 ± 0.15	1.28 ± 0.49*	0.8	0.6
Zinc	3.6 ± 1.57	10.38 ± 3*	7.5	5
Chloride	1478 ± 627	4662 ± 1778*	-	-
Manganese	1.19 ± 0.49	2.32 ± 0.73*	-	-
Selenium	17 ± 7.3	43.6 ± 8.1*	40	20
Iodine	33.9 ± 34.7	92.2 ± 44.25*	100	70
Retinol	135 ± 112	266 ± 93*	-	-
Carotene	1296 ± 1996	2253 ± 1745	-	-

Vitamin D	0.26 ± 0.19	1.53 ± 0.69*	-	-
Vitamin E	4.66 ± 2.85	7.6 ± 3.9	-	-
Thiamin	0.649 ± 0.38	1.685 ± 0.85*	72	50
Riboflavin	0.457 ± 0.24	1.463 ± 0.55*	1.3	0.6
Niacin	11 ± 3.9	23.8 ± 10*	1.3	1.0***
Vitamin B6	1.14 ± 0.47	2.28 ± 0.79*	13**	-
Vitamin B12	1.179 ± 0.84	3.456 ± 0.93*	1.0	0.6
Folate	83.2 ± 33.5	212 ± 75*	230	160
Panhotenate	2.86 ± 0.16	4.39 ± 1.09*	-	-
Biotin	28.35 ± 27.7	23.69 ± 11.8	-	-
Vitamin C	31.57 ± 32.02	67.25 ± 26.65*	46	32
NSP	5.49 ± 2.13	15.95 ± 6.97*		

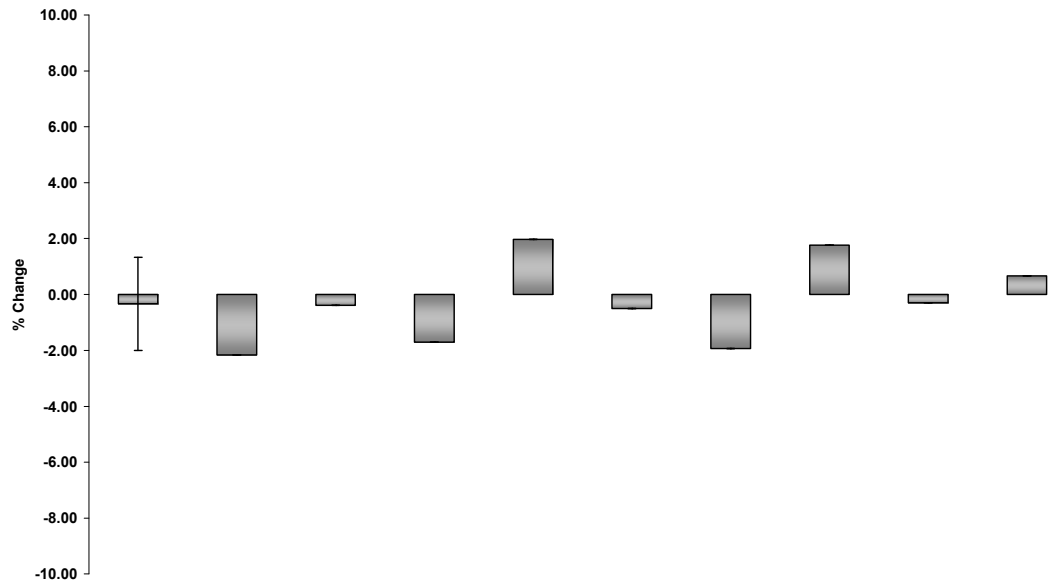
*Data presented as mean ± SD, * p < 0.05 between groups*

Appendix E

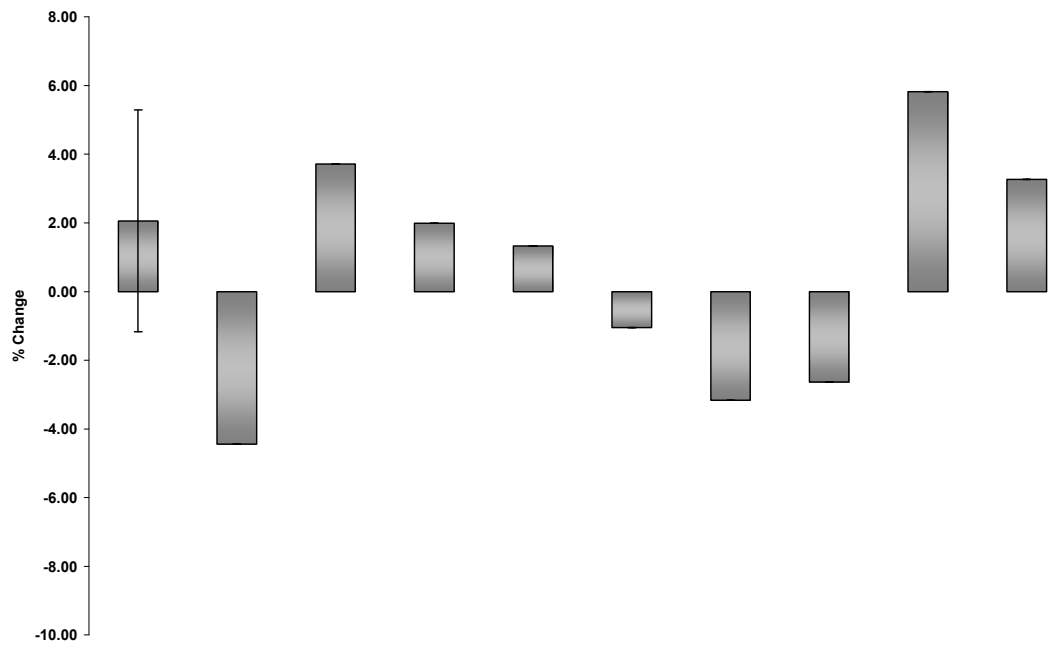
⇒ **Experiment Four: Total Body, L2 – 4 and Femoral Neck BMC and BA**



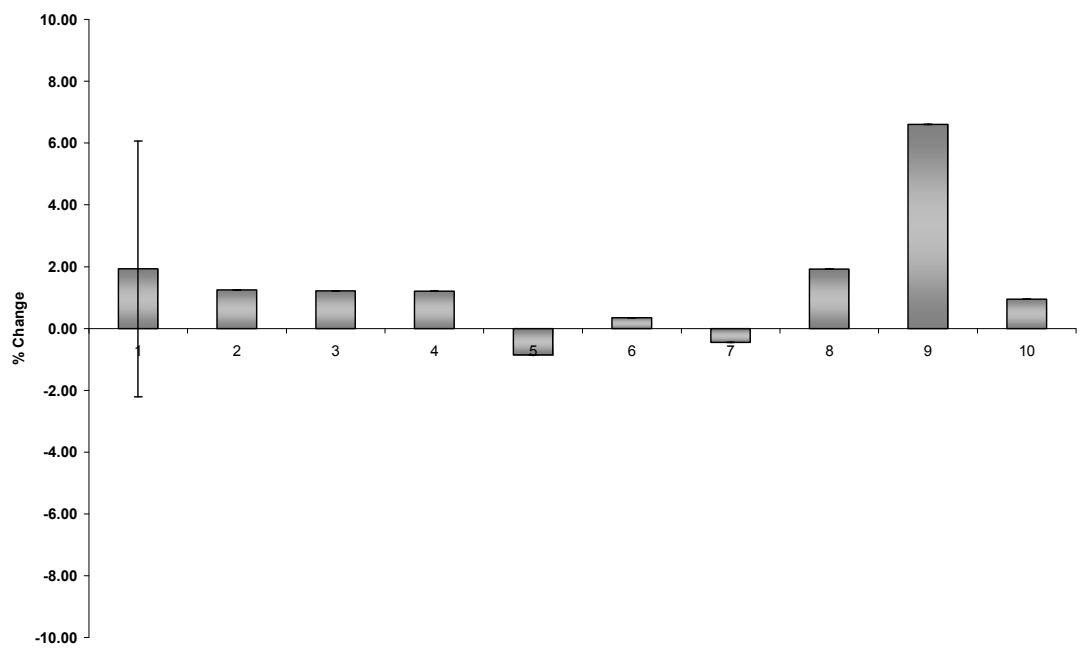
Total Body BMC



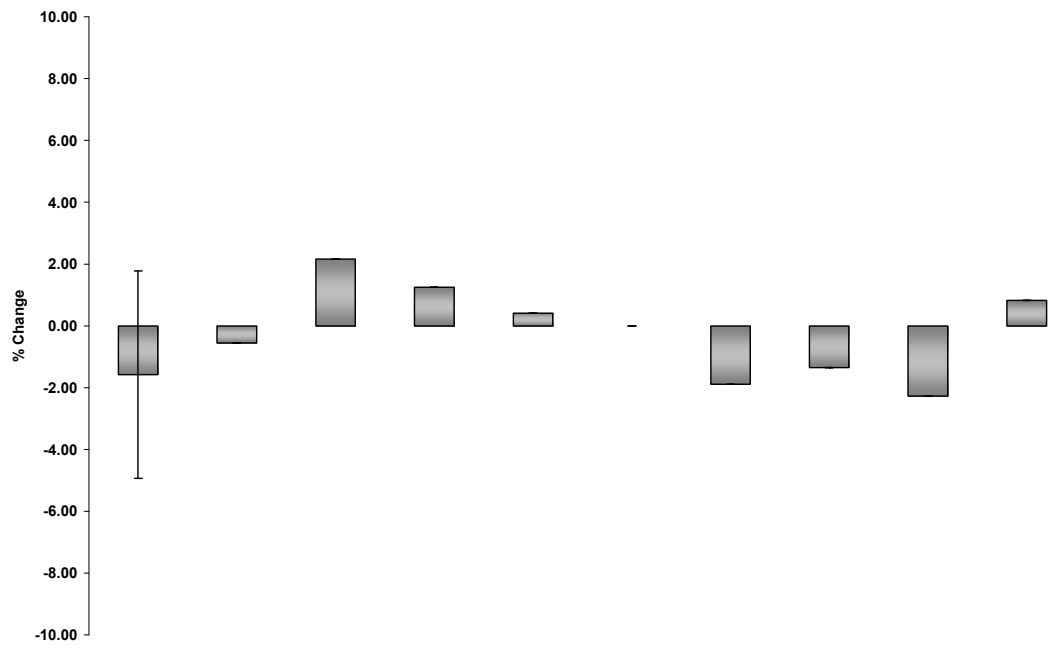
Total Body BA



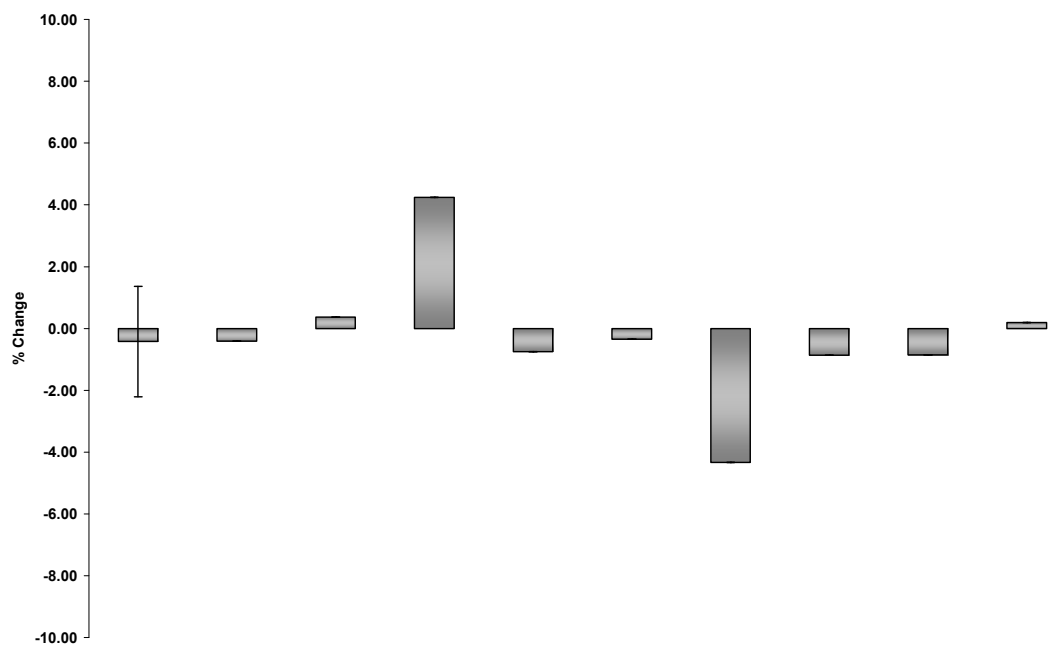
L2 – 4 BMC



L2 – 4 BA



Femoral Neck BMC



Femoral Neck BA