Musculoskeletal Health of Retired Rugby Players

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The musculoskeletal health of retired rugby players

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Abstract

Currently active rugby players demonstrate greater bone density, lean mass and strength compared to other athletes. However, the longevity of these characteristics and the long term health of retired rugby players is not well understood. There exists a high injury toll in rugby, including concussion and the impact of these injuries in retirement is less well known.

In later life, as part of the ageing process, there is a loss of bone density, lean mass and muscle strength. It is not known if the benefits of sports participation, specifically rugby, are preserved in later life and whether the superiority in bone density and strength is present in retirement, following cessation of sports participation.

This research was performed as a cross-sectional study of 138 male participants. Retired rugby players (n=87; 46.1 ±10.5 y; 100 ±15.1 kg; 1.80 ±0.08 m) were from amateur and elite rugby union and rugby league codes. The non-rugby group (n=51; 49.7 ±14.4 y; 86.7 ±14.5 kg; 1.77 ±0.06 m) consisted of retired non-contact athletes (n=30) or those that had never taken part in organised sport (n=21). Participants completed a validated general health questionnaire and were subsequently invited to clinical testing. Basic anthropometry was performed followed by DXA scans of the whole body, both hips and lumbar spine. Balance was assessed using a force plate. A handgrip dynamometer was used to assess grip strength. All participants completed a bone-specific physical activity questionnaire to record past and current levels of physical activity.

Former elite rugby players demonstrated higher bone density (hip neck) than amateur rugby players (1.150 $kgcm^{-2}$ vs. 1.060 $kgcm^{-2}$, p = 0.03) and non-contact athletes (1.150 $kgcm^{-2}$ vs. 1.032 $kgcm^{-2}$p = 0.01). The elite group also had greater lean mass (71.6 kg vs. 65.2 kg, p=0.002) and strength (54.3 kg vs. 48.8 kg, p=0.038) compared to the amateur group and non-contact group. However, following

adjustment of bone density scores for weight and age, there were no differences between groups. Furthermore, muscle quality was similar across all groups. No difference in balance performance were seen between groups.

The greater bone density, lean mass and strength of retired elite rugby players may represent a genetic influence providing an inherent advantage for rugby participation or the result of previous participation in a physical, contact sport such as rugby. However, the absence of a relationship between past physical activity and bone density suggests that current physical activity exerts more influence on current musculoskeletal health status. This highlights the importance of continued physical activity in retirement in order to offset the age-related decline in bone density, lean mass and strength.

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Abbreviations

BMD – Bone Mineral Density

BESS – Balance Error Scoring System

BMU – Bone Multicellular Unit

cBPAQ – current Bone Specific Physical Activity Questionnaire

pBPAQ – past Bone Specific Physical Activity Questionnaire

DXA – Dual Energy X-ray Absorptiometry

LOS- Limits of Stability

mCTSIB- modified Clinical Test of Sensory Interaction in Balance

SET – Stability Evaluation Test

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Papers published from the research contained in this thesis.

Entwistle, I., Francis, P., Lees, M., Hume, P. and Hind, K. 2022. Lean Mass, Muscle Strength, and Muscle Quality in Retired Rugby Players: The UK Rugby Health Project. *International Journal of Sports Medicine*. **43**(11), pp.958–963.

Entwistle, I., Hume, P., Francis, P. and Hind, K. 2021. Vertebral Anomalies in Retired Rugby Players and the Impact on Bone Density Calculation of the Lumbar Spine. *Journal of Clinical Densitometry*. **24**(2), pp.200–205.

Additional papers

Hind, K., Konerth, N., **Entwistle, I.,** Hume, P., Theadom, A., Lewis, G., King, D., Goodbourn, T., Bottiglieri, M., Ferraces-Riegas, P., Ellison, A. and Chazot, P. 2022. Mental Health and Wellbeing of Retired Elite and Amateur Rugby Players and Non-contact Athletes and Associations with Sports-Related Concussion: The UK Rugby Health Project. *Sports Medicine*. **52**(6), pp.1419–1431.

Hind, K., Konerth, N., **Entwistle, I.,** Theadom, A., Lewis, G., King, D., Chazot, P. and Hume, P. 2020. Cumulative Sport-Related Injuries and Longer Term Impact in Retired Male Elite- and Amateur-Level Rugby Code Athletes and Non-contact Athletes: A Retrospective Study. *Sports Medicine*. **50**(11), pp.2051–2061.

Published Abstracts

Entwistle, I., Francis, P., Hume, P.A. and Hind, K. 2018. Bone mineral density in retired rugby players: initial findings from the UK Rugby Health project. *Journal of Clinical Densitometry*. **21**(4), p.1.

Entwistle, I., Francis, P., Hume, P.A. and Hind, K. 2018. Vertebral body anomalies on bone density scans in younger and older retired rugby players: the UK Rugby Health project. *Journal of Clinical Densitometry*. **21**(4), p.1.

Entwistle, I., Francis, P., Lees, M., Hume, P. and Hind, K. 2021. P716 Current but not past, bone-specific physical activity levels are associated with bone mineral density in retired rugby players. *Osteoporosis International*. **32**(Suppl 1), p.S345.

Entwistle, I., Francis, P., Lees, M., Hume, P. and Hind, K. 2021. P718 Retired Rugby players possess superior lean mass and strength but not muscle quality. *Osteoporosis International*. **32**(Suppl 1), p.S346.

Chapter 1.0 Introduction

Lean mass and strength accumulate from birth until adulthood but from around midlife, there is a progressive loss of lean mass (Dodds et al., 2014; Cruz-Jentoft et al., 2019). The maintenance of bone, muscle mass and strength is important in advancing years in order to prevent the onset of osteopenia, osteoporosis, or sarcopenia.

Osteoporosis is a disease state, characterised by altered bone architecture and reduced bone mass (Hernlund et al., 2013). Bone fracture is the clinical outcome of concern with an associated burden of pain, disability and death (Svedbom et al., 2013), presenting a national and global public health challenge. In the UK, in 2019, the cost of osteoporosis incident fractures was over 3 billion euros, with total costs exceeding 5 billion, and fracture costs estimated to be 2.4% of all healthcare spending (Kanis et al., 2021). Sarcopenia is the age associated loss of muscle mass and strength (Cruz-Jentoft et al., 2019) and is associated with several detrimental outcomes including increased risk of falls, morbidity and mortality (Mayhew et al., 2019).

Rugby is a popular contact sport with 8 million rugby union players (World Rugby, 2023). In England, 2020, there were 195,000 people playing rugby union and approximately 59,000 playing rugby league at least twice per month (Statista, 2023). Competitive rugby athletes develop a unique physique (Olds, 2001; Reale et al., 2020), participating in high levels of physical activity and game play. The demands of the game together with the contact nature of the sport, places emphasis on certain characteristics, such as mass and speed in order to generate momentum (Baker and Newton, 2008). Rugby players have demonstrated higher amounts of mass and lean mass (Hind et al., 2015) and muscle strength (Elloumi et al., 2009) compared to non-athletes. Partaking in intense, physical exercise in order to develop mass and strength, also promotes osteogenesis and development of bone mass (Kelley et al., 2000; Maillane-Vanegas et al., 2018; Kopiczko and Cieplińska, 2022). Currently active rugby players have higher bone density than other athletes (Hind et al., 2015).

Player health encompasses several aspects as noted by Griffin et al. (2021), these are cardiovascular, metabolic, musculoskeletal, neurological and immunological. The scope of this thesis is to investigate the musculoskeletal health of retired rugby players. In particular, bone density, muscle strength and muscle mass. Neurological health will be investigated by measures of balance and postural control.

While there have been studies addressing the health of retired rugby players (Davies et al., 2017; Hind et al., 2020; Hume et al., 2022), there have been non where musculoskeletal health has been the primary focus. In particular, bone density data in retired rugby players does not exist. Davies et al., (2017) demonstrated a significantly higher prevalence of reported osteoporosis. However, bone density was not measured, rather this was self-reported 'physician-diagnosed' osteoporosis and therefore, error is possible.

There is little understanding of the ability of the retired rugby player to actively participate in retired life. The high proportion of athletes with degenerative joint disease is concerning and the association with reduced mental and physical quality of life (Davies et al., 2017; Paget et al., 2020). The potential mental health issues and reliance on painkillers and alcohol (Hume et al., 2022) would be advantageous to avoid. The nature of the problem lies in the potential for long term detriment due to the issues such as osteoarthritis and lack of physical activity.

The high injury toll of a career in rugby has been established and the long term outcomes documented (Davies et al., 2017; Hind et al., 2020; Hume et al., 2022). Moreover, the capacity to continue involvement in physical activity could be impacted by the injuries sustained while playing. There is a high occurrence of degenerative disease in multiple joints seen in former rugby players (Davies et al., 2017; Davies et al., 2017; Hind et al., 2020). The ability to pursue weight bearing, bone developing activity may be diminished in this population.

The potential long-term consequences of a high injury toll, and difficulty in maintaining a regular physical activity may have an impact on the posture and balance performance of these retired

athletes. In particular, concussion has been the most reported injury by retired rugby players (Hind et al., 2020) and the immediate, negative effects on balance are well documented (Broglio and Puetz, 2008; Parker et al., 2008a). The balance performance following a career in rugby, compounded by the ageing process is not known.

The combined impact of reduced balance performance and resilience to the effects of falling place importance on strategies to combat these downfalls in retirement and whether the optimal period of activity is in youth and young adulthood or an emphasis on continued activity in retirement.

While other retired athletes have tested favourably in various measures of health and performance in retirement (Räty et al., 2002; Kettunen et al., 2010; Andreoli et al., 2012; Laine et al., 2016a), it is not known if these desirable characteristics in rugby players are maintained in retirement, following cessation of the sport.

The lack of studies in this area is a challenge and the potential reasons for this may be due to the reluctance of players to speak negatively about a significant part of their lives, on which they hold a positive opinion (Davies et al., 2016). A perceived loyalty to the sport from which they have gained much from participation. Support from governing bodies may also be challenging given the conceivable negative impact on current players and those yet to start the game (including their parents). The recent attention on the unknown long term effects of injuries such as concussion may make some uncomfortable.

The purpose of this project, therefore, was to investigate the musculoskeletal health status of retired rugby players compared with retired non-contact athletes and develop a comprehensive understanding of the retired rugby player. The importance of research such as this is to elucidate further the health status of these retired athletes in order to provide appropriate support for the maintenance of good health. The increasing injury rates in rugby potentially contributing to the higher prevalence of osteoarthritis (Davies et al., 2017). Furthermore, this is also associated with significantly

poorer mental health (Davies et al., 2017; Paget et al., 2020). This may make a contribution to poorer balance, potentially increasing the risk of falls and subsequent fracture.

Moreover, high incidence of concussion has also been associated with adverse mental health (Hind et al., 2022) poorer mental health could be further compounded by hazardous alcohol consumption, where higher levels have been demonstrated in rugby players (Hume et al., 2022). Therefore, addressing these issues of physical health such as the multifaceted musculoskeletal health could impact the mental health and future quality of life of this retired athlete group.

1.1 Aims and Objectives

The research focussed on the comparative analysis of retired male rugby union and rugby league players, versus age-equivalent retired non-contact athletes with the following aims.

Aims

1. Identify a method for assessing lumbar spine bone density in men under 50 years and determine the impact of vertebral anomalies in the lumbar spine and the effect on bone density calculation.
2. Determine the spine and hip bone density in retried rugby players compared to age-matched, retired non-contact athletes.
3. Identify past and current levels of participation in osteogenic physical activity and determine the association with the bone density.
4. Compare muscle mass, strength, muscle quality and body fat across retired rugby players and retired non-contact athletes.
5. Assess balance performance between retired rugby players and non-contact athletes and associations with previous injury. Elucidate potential risk of falling and difficulties with tasks of daily living.

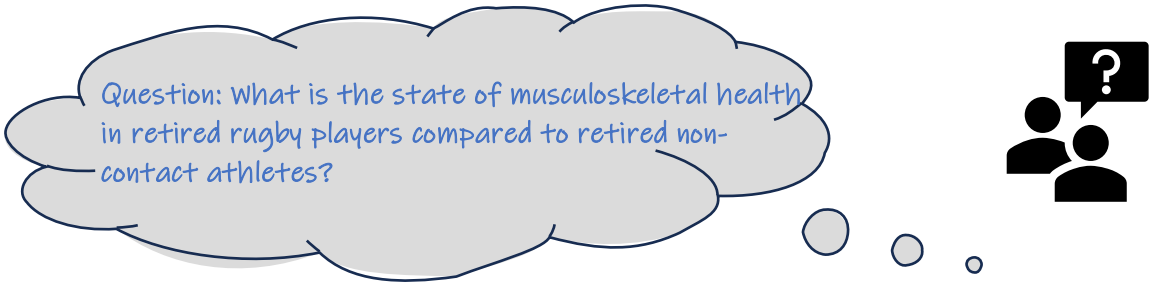
Objectives

1. Complete Dual X-ray Absorptiometry scans of the hips, spine and whole body for purposes of bone density assessment of the hip and spine and calculation of levels of lean and fat mass (Aims 1, 2 and 4).

2. Complete Bone-Specific Physical Activity Questionnaire (BPAQ) to determine the amount of osteogenic physical activity across the lifecourse (Aim 3).
3. Measure hand grip strength using handgrip dynamometer to determine strength and allow calculation of muscle quality (Aim 4).
4. Perform tests of posture and balance performance, namely the 'Limits of Stability (LOS)', 'Stability Evaluation Test (SET)' and the 'modified Clinical Test of Sensory Interaction on Balance (mCTSIB)' using Neurocom VSR force plate, to compare risk of falling and associations with previous injury (Aim 5).
5. Obtain career and injury history, using validated General Health Questionnaire (GHQ) (Aim 5).

The significance of this research to inform potential strategies to promote healthy ageing and mitigate the onset osteoporosis and the age-related loss of lean mass, to guide policy for the prevention of falls and the related sequalae, including fracture, and to gain insight into the optimum lifetime exercise strategy for maintaining musculoskeletal health.

Figure 1.1 Thesis Structure



Literature Review

Ageing and Musculoskeletal Health

The rugby player and the retired athlete

Methods:

Clinical Testing

Basic Anthropometry

DXA scans for:

Whole body composition

Hip and spine bone mineral densitu

Hand grip strength testing

Balance and posture assessment

BPAQ Questionnaire

Chapter 4 Study 1

Vertebral anomalies in retired rugby players and the impact on bone density calculation at the lumbar spine

Chapter 5 Study 2

Bone mineral density in retired rugby players and associations with previous and current physical activity

Chapter 6 Study 3

Lean mass, muscle strength and muscle quality in retired rugby players

Chapter 7 Study 4

Balance and dynamic posturography in retired rugby players

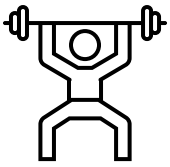
Discussion and Conclusion

Figure 1.2 Addressing the Aims and the PhD

Determine Spine and hip bone density,and the risk of osteoporotic fractures.

There is increased risk of fracture with lower bone density. Muscle mass provides tension to produce strain on the bone, producing an osteogenic effect.

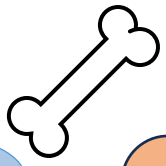
Measure the attributes of muscle mass, including quality and functionality.



Osteogenic physical activity such as rugby training, stimulates bone growth and increases in muscle tissue

Record levels of participation in osteogenic physical activity and determine the association with bone health.

Does participation in physical activity continue post retirement or do long term injuries prevent this?



Assess posturography and balance performance, including potential risk of falls.

If there is low bone density, there is less resilience to trauma such as falling. Optimal balance performance can reduce the risk of falling and therefore the risk of fracture.

Chapter 2.0 Literature Review

This chapter presents a comprehensive literature review that encompasses bone biology, risk factors for osteoporosis, the effects of exercise on bone and muscle health, and the musculoskeletal health of athletes and retired athletes. By critically analysing existing physiological theory and applied research and synthesizing key findings, this chapter aims to provide a solid critical interpretation of these interconnected areas of study.

Bone biology is a complex field that examines the structure, formation, and remodelling processes of bone tissue (Rosen, 2013). Understanding the intricate mechanisms governing bone metabolism and the factors influencing bone health is crucial for comprehending the development and prevention of bone-related disorders, including osteoporosis which is a key area of focus within this thesis. This literature review will critically explore current understanding of bone biology, including the cellular and molecular processes involved in bone formation and resorption, as well as factors influencing bone mineral density (BMD) and strength. This chapter will also further critically review existing literature on the various risk factors for osteoporosis, including age, sex hormones and lifestyle factors.

The effects of exercise on bone and muscle health have garnered considerable attention due to their potential implications for preventing osteoporosis and maintaining musculoskeletal well-being. This literature review will examine the impact of exercise on bone remodelling processes, BMD, and bone strength. Additionally, the interplay between exercise, muscle health, and bone health will be critically explored, as muscle contractions during exercise exert mechanical loading on bones, triggering adaptive responses. Athletes, both current and retired, represent a unique population with distinct musculoskeletal characteristics and concerns and there is a dearth of evidence pertaining to this group. This literature review will examine the musculoskeletal health of athletes in general, and specifically in rugby players, focusing on the potential benefits and risks associated with athletic training and competitive sports participation. The effects of intensive training regimens, repetitive

loading, and sport-specific impacts on bone density, muscle mass, and overall musculoskeletal health will be examined. Furthermore, the transition from an active athletic career to a sedentary lifestyle during retirement and its impact on musculoskeletal health will be discussed.

2.1 Bone Anatomy and Physiology

2.1.1 Bone composition

The composition and structure of healthy bone enables the skeleton to function as an integral part of the human body in terms of an anatomical scaffold and mineral store. An ability to respond effectively to changing environments and mechanical stimulus in order to withstand stresses and strains without fracture, is a key feature of the human bone. Bone is composed predominantly of the extracellular matrix (ECM) and mineral. The ECM is largely Type I collagen, and this provides the building blocks of the matrix and the location for the mineral deposition. The mineral is hydroxyapatite and this delivers strength to the collagen and confers mechanical resistance (Boskey and Robey, 2019). There are three cells located within the bone; osteoblasts, osteoclasts and osteocytes. The osteoblasts are the bone forming cells (Bradley et al., 2019). The osteoclasts are responsible for bone resorption (Takayanagi, 2019). The osteocytes are thought to function as a network of sensory cells embedded in the bone tissue (Bonewald, 2019).

At a macroscopic level, the long bones are constructed of a hollow medullary cavity surrounded by the compact, cortical bone. The spongy, trabecular bone is situated at the epiphyseal ends of long bones and also the majority of the shorter, flat and irregular bones (Tortora and Derrickson, 2006). This organisation provides a strong and relatively light skeletal arrangement. The cortex is dense and is the predominant skeletal mass, providing a surface for tendinous attachment (Office of the Surgeon General (US), 2004).

2.1.2 Bone growth, modelling and remodelling

Bone mass in adulthood varies with those at either end of the spectrum differing by approximately 50% (Seeman, 1998; Seeman, 2019). However, the rate of bone loss is much less severe and suggests that peak bone mass is important in the inevitable decline of mass in advancing years (Hui et al., 1990). The evidence suggests that traits developed by the first 2 years track into adulthood, with such traits as height, bone size and strength predicted by crown heel length at >6 months (Wang et al., 2010). Whilst there is a genetic path controlling bone growth (Murray and Huxley, 1925), external influences have a regulatory influence on the eventual morphology (Lanyon, 1992). For example, racquet sport players have demonstrated differences in bone size of the playing arm versus the non-playing arm (Kontulainen et al., 2003). It is this environmental impact on bone growth that is difficult to define. The ability of the individual to respond to stimulus appears to be more sensitive before adulthood is reached. Less periosteal apposition takes place once full, longitudinal growth is achieved (Seeman, 2008a).

During growth, bone is constructed and shaped by modelling and remodelling. Modelling is the process by which bone is constructed by the osteoblasts without any previous resorption taking place. Although modelling is generally considered to be bone forming, it can be resorptive in nature, when the bone is removed without subsequent bone deposition. This is demonstrated with endosteal resorption during marrow excavation during growth(Seeman, 2008b; Isales and Seeman, 2019). Remodelling is the reconstruction of bone following the resorption of bone by the osteoclasts at the same site (Bliziotes et al., 2006; Seeman, 2008b). This requires the co-ordinated action of the osteoclast and the osteoblast, which form the basic multicellular unit (BMU). The primary aim of this arrangement during growth is to maximise strength, while minimising weight (Seeman, 2008a). This can be achieved with the long, tubular bones through the hollow centre of the medullary cavity. This cavity can be widened and thus increasing the cross-sectional area of the bone, without the presence of more total mass. This is achieved through strategic removal of bone from the inner, endosteal

surface by the osteoclasts and a corresponding deposition of bone on the outer, periosteal surface (Zebaze et al., 2007). This process can continue until full longitudinal growth has been achieved in the late teens, early 20s (Baxter-Jones et al., 2011).

The modelling and remodelling processes take place throughout life, though modelling has consistently been shown to be more dominant during youth for the purpose of achieving peak strength and mass (Seeman, 2008b). Remodelling functions to maintain the bone quality and strength and is the dominant process in adulthood. The resorption and replacement with new bone is pivotal for the maintenance of bone health. Fatigued, damaged bone is removed and replaced with new bone (Parfitt, 2002) and the importance of this has been highlighted when the use of antiresorptive agents resulted in atypical fracture (Mashiba et al., 2000; Odvina et al., 2005). The reduction in strength is often due to the reduction in bone formation, resulting in a negative balance between resorption and formation(Rosen, 2013). Diseases of bone are often due to interference with the remodelling process.

Osteoblasts and osteoclasts achieve remodelling, in a healthy individual, with a harmonised process. Osteoclasts are of hematopoietic origin (Takahashi et al., 2008), from those precursor cells from which white blood cells originate (Takahashi et al., 2008) and are responsible for bone resorption. The resorptive stage of the remodelling process lasts for approximately 3 weeks whereby osteoclasts secrete enzymes to dissolve the mineral component and breaking down the bone matrix (Takayanagi, 2019). Following resorption there is a reversal period, during which the osteoblasts are thought to form from precursor cells. The bone formation stage then follows, this is the longest period, lasting for 3 months and new bone is laid down (Parfitt, 1994; Office of the Surgeon General (US), 2004). Primary mineralisation occurs at this point, but secondary mineralisation can take much longer (Akkus et al., 2003; Isales and Seeman, 2019). It is this newly mineralised bone where the deficit exists, given the discrepancy between the removed, old bone and the new, not yet fully mineralised bone (Office of the Surgeon General (US), 2004).

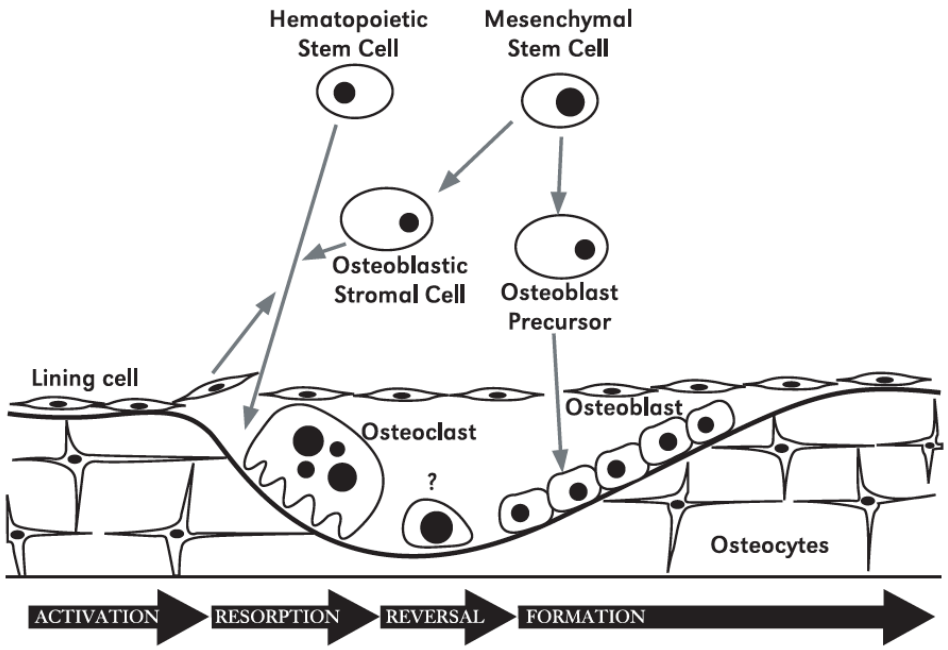


Figure 2.1: The BMU and the remodelling process (Office of the Surgeon General (US), 2004, p. 23)

2.1.3 The Osteocyte

The osteocyte is the most abundant of the bone cells, contributing 90-95% of all bone cells (Bonewald, 2019). Osteocytes are osteoblasts that have become buried in the bone matrix. These cells are characterised by long dendritic processes and are connected in a network to each other and cells on the bone surface (Manolagas, 2000). The developing evidence is that the osteocyte plays a key role in the response to mechanical stimulus (Bonewald, 2011) and that this controls the bone resorption and formation process (Bonewald, 2019), with evidence seen in animal models (Tanaka et al., 1995). It appears that apoptosis of the osteocytes stimulates action of the osteoclast (Bonewald, 2019)

This network of osteocytes enables cells to perform as mechano-sensors. The arrangement within the bone of the osteocytes connected to via extensive dendrites allow the passage of signals from various locations from deep within the bone and those lining the surface (Cowin et al., 1995; Klein-Nulend and Bonewald, 2008). Animal studies have demonstrated that the osteocyte is sensitive to mechanical stress (Skerry et al., 1989). Metabolic activity within the osteocytes promptly changing

following intermittent loading (Klein-Nulend and Bonewald, 2008). The initiation of action by the osteoclast and osteoblasts is regulated by strain, via the signalling of osteocytes, where mechanical stimulus creates a biochemical response (Bullock et al., 2019). The stimulation of the osteocytes by mechanical overloading instigates the recruitment of osteoblasts, whereas underloading prompts the resorption process by the osteoclasts (Smit and Burger, 2010). Furthermore, animal studies have shown that the removal of osteocytes via ablation provided protection from bone loss initiated by lack of use (Tatsumi et al., 2007).

The method by which osteocytes detect the stimulus and initiate the response is unclear, while it is known that osteocytes residing in the bone can directly detect strain, they are better situated to detect the fluid flow (You et al., 2000). Fluid moving from an area of higher pressure across the cell surface to an area of lower pressure generates shear stress when long bones are bent (Bullock et al., 2019). This stress is magnified at the cellular level compared to that experienced on the whole bone (Wang et al., 2008).

2.1.4 Mechanical loading and its osteogenic effect

The development of bone mass follows a predetermined, genetic pathway. This is seen with limb buds grown in vitro, developing into femurs (Murray and Huxley, 1925). However, the ability of the skeleton to respond to mechanical stimulus is a feature that allows functional adaptation, given the right stimulus. Wolff's law, in 1892 stated:

"Every change in the form and function of bone or of their function alone is followed by certain definite changes in their internal architecture, and equally definite alteration in their external conformation, in accordance with mathematical laws" (Wolff, 1986)

That is, the bone will alter its structure in response to or lack of mechanical stimulus and this premise has largely been accepted. This functional adaptation of the bone is necessary in order to preserve the orchestrated balance between the mass required, necessary for adequate bone strength, while preserving only enough mass for the bone weight to not be excessive. Frost later proposed his 'mechanostat' (Frost, 1987), where it was theorised that the response by the bone to mechanical loading is controlled by something akin to a thermostat. A loading strain threshold or set point whereby stimulus above this threshold will instigate the modelling process but a load stimulus below this set point would initiate bone removal via the remodelling process

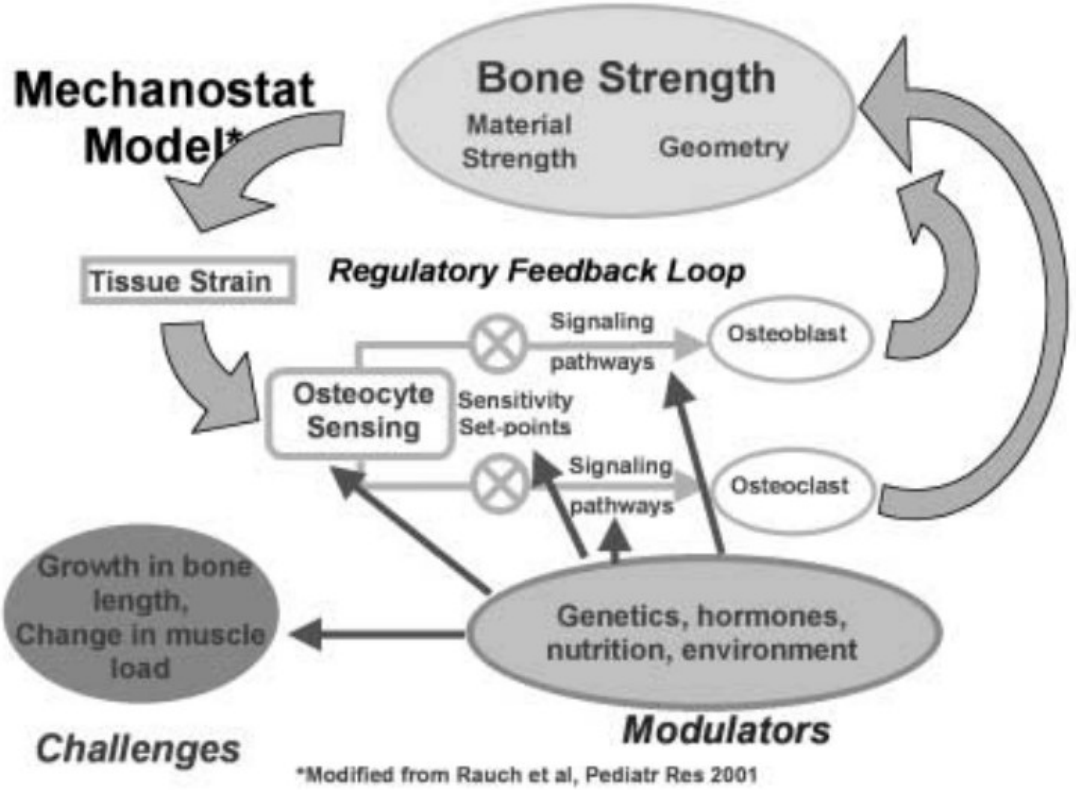


Figure 2.2 A functional model of bone development based on the mechanostat theory (Petit et al., 2005, p. 216)

The timing of the physical activity over the lifespan can be optimised to promote adaptation. Evidence suggests that the period prior to puberty rather than post puberty is more successful in generating a response from the developing bones. This has been demonstrated in the arms of racquet sport players, where larger side to side differences in bone mineral content were seen in those starting prior to puberty rather than after (Kannus et al., 1995; Kontulainen et al., 2003). Forwood and Burr (1993) concluded that exercise in childhood and adolescence is of greater importance than that performed in adulthood for achieving higher peak bone mass. Therefore, it is important to take advantage of this apparent window of opportunity, within which mechanical stimulation can promote bone building to achieve optimal peak bone mass, augmented by the already active modelling process. In relation to exercise and bone mass, it is not known how well preserved in adulthood the bone mass achieved in youth. Given a large intervening period and a number of cofounding factors, it is very difficult to determine. Cross sectional studies suggest that bone mineral density is not preserved into adulthood and fracture risk is not reduced, compared to controls (Karlsson et al., 2000).

2.2 Bone Pathophysiology

Osteoporosis is a disease state, characterised by altered bone architecture and reduced bone mass (Hernlund et al., 2013). Bone fracture is the clinical outcome of concern with an associated burden of pain, disability and death (Svedbom et al., 2013).

There is an inevitable loss of bone mass with ageing in both men and women. Following the cessation of longitudinal growth, the purpose of bone formation is repair and maintenance. It is an imbalance of the remodelling that occurs at around midlife (more aggressively in females at menopause) which promotes the structural deterioration of the skeleton (Seeman, 2018). This is a culmination of a number of age-related changes in the cellular components of the modelling and remodelling process that bring about the reduction of the bones ability to withstand force and the compromising of its structure (Zebaze and Seeman, 2010). The imbalance occurs due to a reduction

in bone formation coupled with increases in bone resorption, together with an increased rate of remodelling (Zebaze and Seeman, 2010). The rate of periosteal apposition seen during growth is now reduced (Isales and Seeman, 2019)

The increased remodelling rate reduces bone strength via different means. The new bone formed can take months or even years to fully mineralise and fully restore bone mass (Akkus et al., 2003). The newly mineralised bone is less resistant to the bending strain and more vulnerable to fracture (Boivin and Meunier, 2002). Trabecular bone has a greater surface area than cortical bone on which the osteoclasts can perform. The remodelling process produces resorption cavities in the trabecular bone, this creates thinning or complete perforation of the trabeculae (Zebaze et al, 2008; Van Der Linden et al, 2001). The complete loss of connectivity vastly reduces bone strength and is more detrimental than thinning (Van Der Linden et al., 2001). This is seen more dramatically in women as a consequence of less bone mass prior to the onset of rapid remodelling around menopause (Zebaze and Seeman, 2010). Furthermore, while there is a decrease in formation of bone by the BMU, there is not an increase in resorption, as seen in women, resulting in the loss of trabecular connectivity (Aaron et al., 1987).

Remodelling is also active on the cortical bone surface, although the surface area to volume ratio is greater in trabecular bone, the overall volume of cortical bone is superior. Over time, the quantity of cortical bone mass loss is greater than trabecular bone (Rosen, 2013). The remodelling takes place on the intracortical surface increasing the coalescence of pores, thus increasing the overall porosity of the cortical bone, reducing fracture resistance by the ability of the bone to limit crack propagation (Yeni et al., 1997).

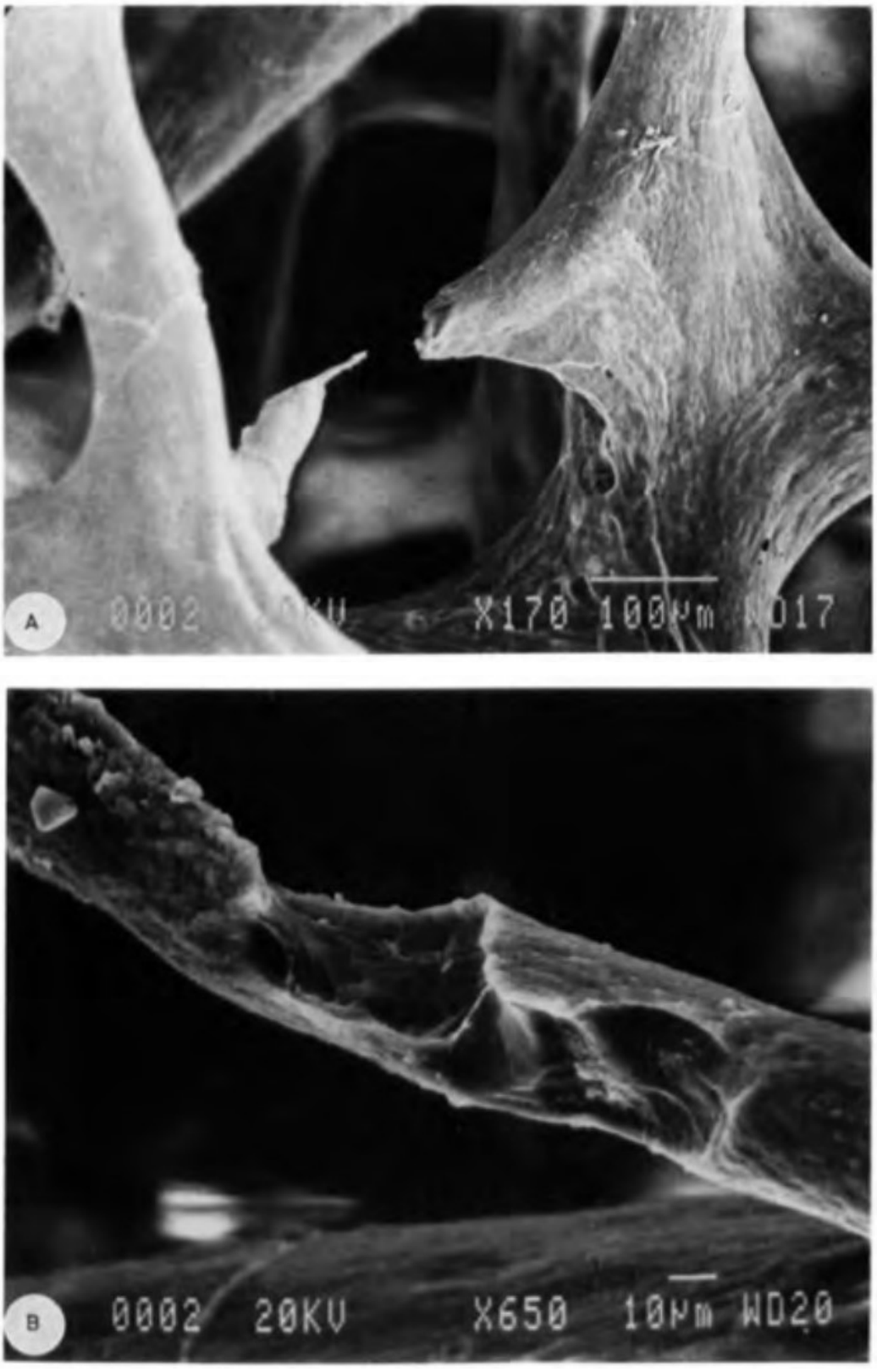


Figure 2.3 A. Perforate horizontal trabecula. B. Thin trabecula demonstrating osteoclastic resorption

(Mosekilde, 1990, pp. 29-30)

2.2.1 Sex differences in bone remodelling

The age-dependent attrition of BMD displays sex-specific temporal divergence due to differential endocrine transitions experienced by men and women. Both sexes experience an inevitable reduction in BMD as a part of the aging process; however, the temporal progression and intensity of this decline differ between sexes. In women, a significant acceleration in bone mass loss is observed peri-menopausally, a phase typically marked by cessation of ovarian function and subsequent reduction in oestrogen production, usually in the fifth decade of life. Oestrogen plays a pivotal role in the regulation of bone homeostasis; thus, its sharp decrease during menopause can trigger a phase of rapid osteoclastic activity leading to net bone loss. This rapid phase of bone depletion can persist for several years post-menopause, significantly increasing the risk of osteoporosis and fragility fractures in women.

Conversely, men do not undergo an equivalent life stage event that instigates an abrupt decrease in BMD. The decline of androgens, specifically testosterone, which substantially contributes to bone health in men, follows a more gradual trajectory compared to the rapid drop in oestrogen levels accompanying menopause. This steady decline commences in middle age and continues into later years, resulting in a more gradual and less acute decrease in BMD compared to women. Despite this more gradual reduction, older men remain at risk for developing osteoporosis, albeit typically at a later age than women. Moreover, lifestyle variables, including dietary habits, physical activity, and risk behaviours such as smoking or excessive alcohol consumption, can modulate the rate of bone loss in both sexes.

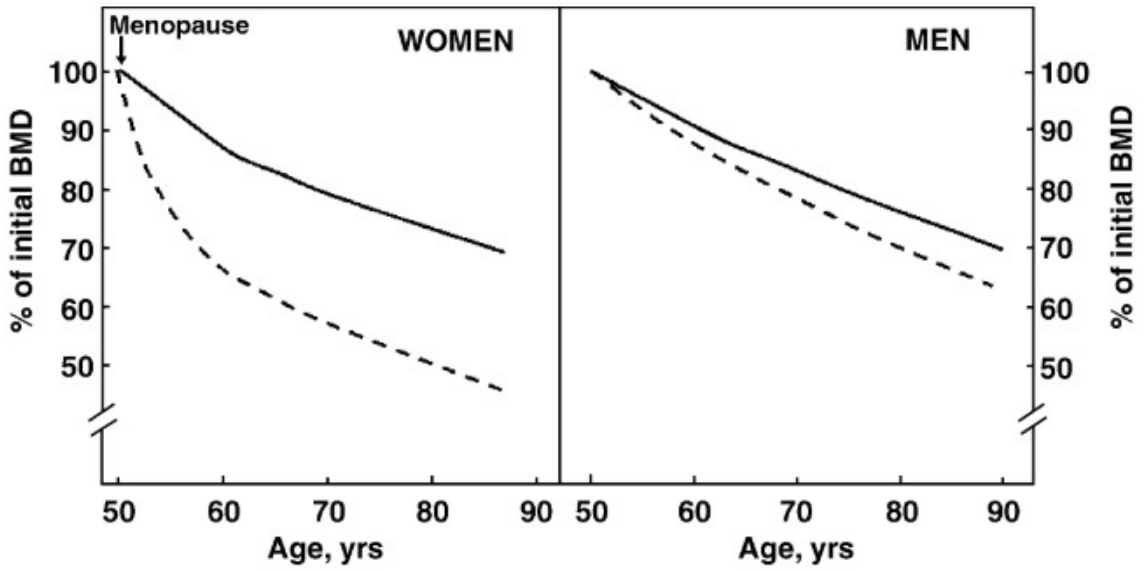


Figure 2.4 Age related bone loss in women and men, dashed lines represent trabecular bone and solid lines cortical bone (Khosla and Riggs, 2005, p. 1016)

2.2.2 Epidemiology and the Burden of Osteoporosis

"Osteoporosis is a skeletal disease characterised by low bone mass and microarchitectural deterioration of bone tissue, with consequent increase in bone fragility and susceptibility to fracture" (Peck, 1993).

Osteoporosis is a national and global public health challenge, with 9 million new osteoporotic fractures seen in one year (Johnell and Kanis, 2006). The Global Burden of Disease Study has shed light on the escalating issue of musculoskeletal disorders, indicating a 17.7% increase in disability-adjusted life years (DALYs) between 2005 and 2013 (Murray et al., 2015). More disconcertingly, lower back pain has been identified as the predominant cause of years lived with disability (Vos et al., 2015).

In the European context, 2010 data showed that approximately 22 million women and 5.5 million men were diagnosed with osteoporosis. The UK contributed significantly to this figure, accounting for 2.5 million women and 0.7 million men, respectively (Hernlund et al., 2013). Prevalence rates were 6.7% for men over 50 and 22.1% for women in the UK, figures that are similar to the rest

of Europe (Hernlund et al., 2013). The primary clinical concern arising from osteoporosis is fracture, particularly of the hip, vertebrae, and distal forearm, which are the most common sites of osteoporotic fractures (Warriner et al., 2011). In 2010, in the UK alone, 6,059 deaths were attributed to fractures, with almost half (46%) being due to hip fractures (Svedbom et al., 2013). These numbers are particularly concerning given the significant morbidity, mortality, and healthcare costs associated with such fractures. Over the course of a lifetime, the fracture incidence differs in men compared to women, with men demonstrating peaks in fracture rate in adolescence (Moon et al., 2016) and then again in old age (Curtis et al., 2016). This is shown in figure 2.5.

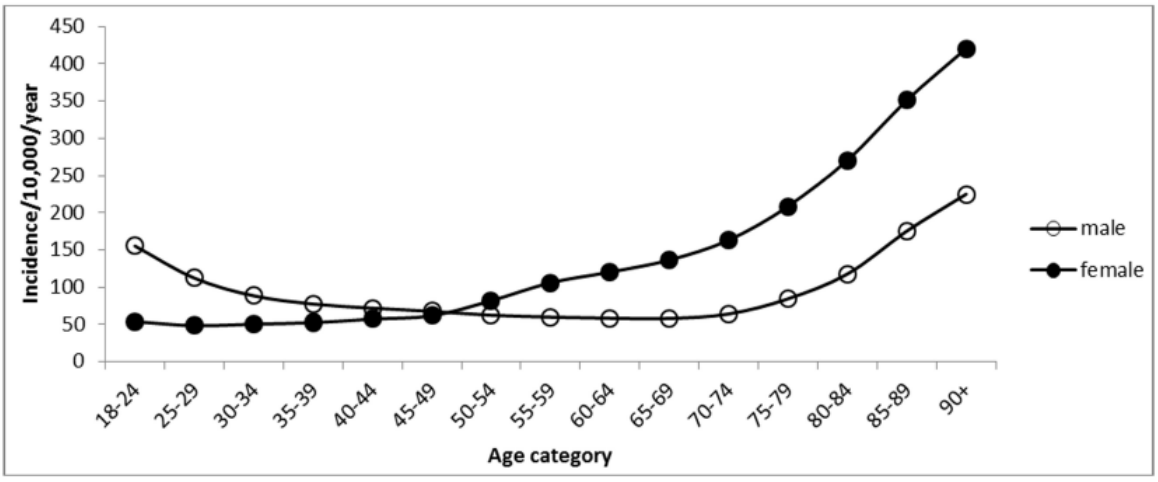


Figure 2.5 Age and sex specific fracture incidence at any site among adults 1988-2012 (Curtis et al., 2016, p. 14)

The rate of fracture increases in later life, with an earlier onset seen in women around 50 years, compared to men at around 70 years, as demonstrated in a UK study of GP records. This is shown in figure 2.6.

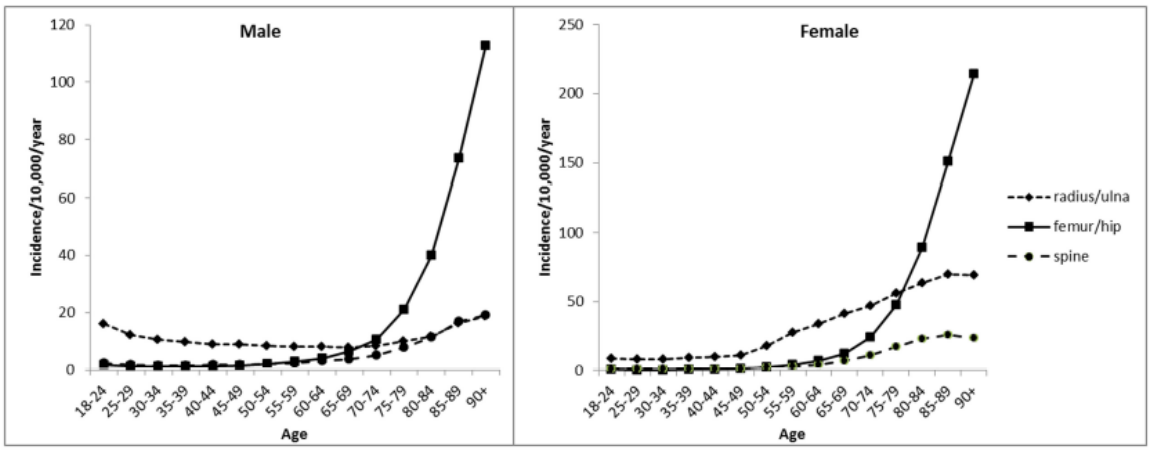


Figure 2.6 Age and sex specific incidence rates of fracture at the femur/hip, radius/ulna and spine 1988-2012 (Curtis et al., 2016, p. 15)**.**

The incidence of hip fracture, reported by a UK study, was at 11.3 and 32.1/10000 person years for men and women over 50 years respectively (Curtis et al., 2016). The incidence rapidly rises above 70 years in both sexes, with fracture rates in women greater than those in men. This ratio was also seen in a previous UK study reporting hip fracture incidence rates of 11.1/10000 person-years in men and 37.2/10000 person years in women (van Staa et al., 2001). The fracture-related mortality appears substantial following hip fracture. For example, a meta-analysis reported the relative hazard for mortality at various time intervals following hip fracture. In men, the relative hazard was at 8.0, 3.7 and 2.5 after 3 months, 1 year and 10 years respectively but in women, the relative hazard was 5.8, 2.9 and 2.3 at the same time-points. (Haentjens et al., 2010). This evidence points to a greater risk of fracture-related death for men, with that risk persisting for at least 10 years. A British population based retrospective cohort study demonstrated that the risk of death, 1 year post hip fracture had remained largely unchanged between 2000 and 2010, reporting a 3.5 fold risk in men and 2.4 fold risk in women (Klop et al., 2014).

Vertebral fracture is more challenging to accurately document. The major clinical consequences of vertebral fracture are back pain, loss of height and kyphosis (Curtis et al., 2018). Not all vertebral fractures are identified clinically or radiographically (Oleksik et al., 2000). The European Vertebral Osteoporosis Study (EVOS) determined the prevalence of vertebral deformity for men and women (aged 50-79 years) across Europe was 12% (O'Neill et al., 1996). The age standardised incidence of vertebral fracture (morphometrically defined) as reported by the European Prospective Osteoporosis Study (EPOS) was 5.7 and 10.7/1000 person-years for men and women respectively (aged 50-79 years) (Felsenberg et al., 2002). The incidence increased with age, with the highest incidence in the 75-79 years group in both men and women. This is supported by the findings from the EVOS data, with the highest prevalence in the 75-79 years group. The associated mortality from vertebral fracture is significant with a standardised mortality ratio (SMR) of 2.12 in men and 1.82 in women reported from a longitudinal study performed between 1989 and 2007 on those over 60 years (Bliuc et al., 2009). Furthermore, the SMR for vertebral fracture was greatest in men aged 60-74 years, for the five years post fracture at 4.19 (Bliuc et al., 2009).

Distal forearm/wrist fracture is subject to a much less severe incline in rates with advancing age, particularly so in men. The incidence was reported at 8.9 and 39.7/10000 person years in men and women ages over 50 years respectively (Curtis et al., 2016). Wrist fracture does not appear to influence mortality, with observed survival being largely similar to that expected in men and women (van Staa et al., 2001).

2.2.3 Risk Factors for Osteoporosis

2.2.3.1 Low bone mineral density (BMD)

The World Health Organisation (WHO) states the standard by which osteoporosis is diagnosed in postmenopausal women and men over 50 years is a femoral neck BMD of 2.5 standard deviations below the young female adult mean. (World Health Organisation, 2007b). Osteopenia or low bone mass is described as a BMD of between -1.0 and -2.5 standard deviations below the young adult mean (World Health Organisation, 1994; World Health Organisation, 2007; Kanis et al., 2008). The BMD of healthy women aged 30-40 years is normally distributed (see figure 2.7).

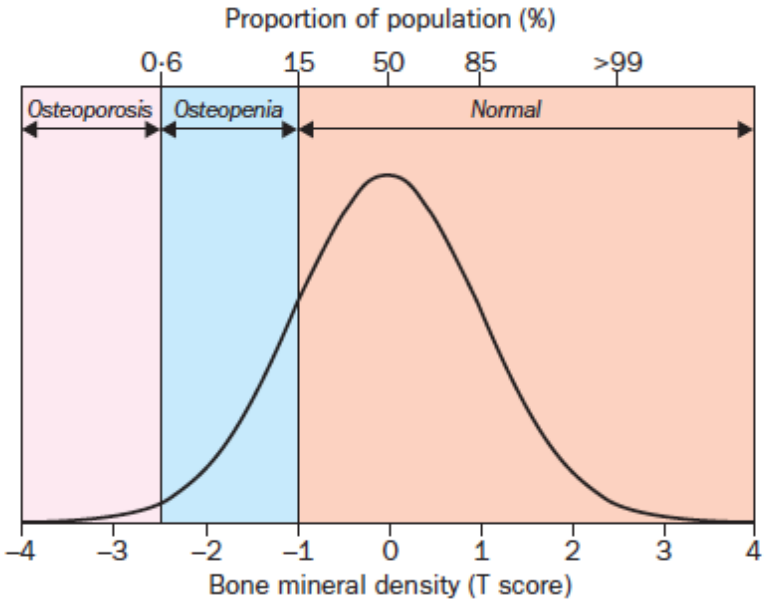


Figure 2.7 BMD distribution in in healthy women aged 30-40 years (Kanis, 2002, p. 1930)

The distribution gradually shifts towards the negative, with increasing age BMD reduces, as the proportion of those with osteoporosis increases exponentially (Kanis et al., 1994). As such, BMD is a strong indicator of fracture risk for men and women (Marshall et al., 1996; Schuit et al., 2004; Johnell et al., 2005), with relative risk ranging from 1.4-2.6 for a 1 SD decrease in BMD at a given site (Marshall et al., 1996). Furthermore, the relative risk increases for a specific site when the BMD measurement is at the same site, i.e. a higher fracture risk at the hip when the BMD measurement is taken of the hip (Marshall et al., 1996; Kanis, 2002). Data from the Rotterdam study reported very low incidence (men: 2.78%; women: 5.17%) of hip fracture in those with normal BMD (Schuit et al., 2004).

Bone density measurement as a method of fracture prediction has high specificity but low sensitivity, therefore fracture risk in the presence of osteoporosis is high but the but risk is not absent in the absence of osteoporosis (see figure 4) (Marshall et al., 1996; Kanis, 2002). The relationship of BMD and fracture has been shown to be similar to that seen with stroke risk and increasing blood pressure (Cooper and Aihie, 1994).

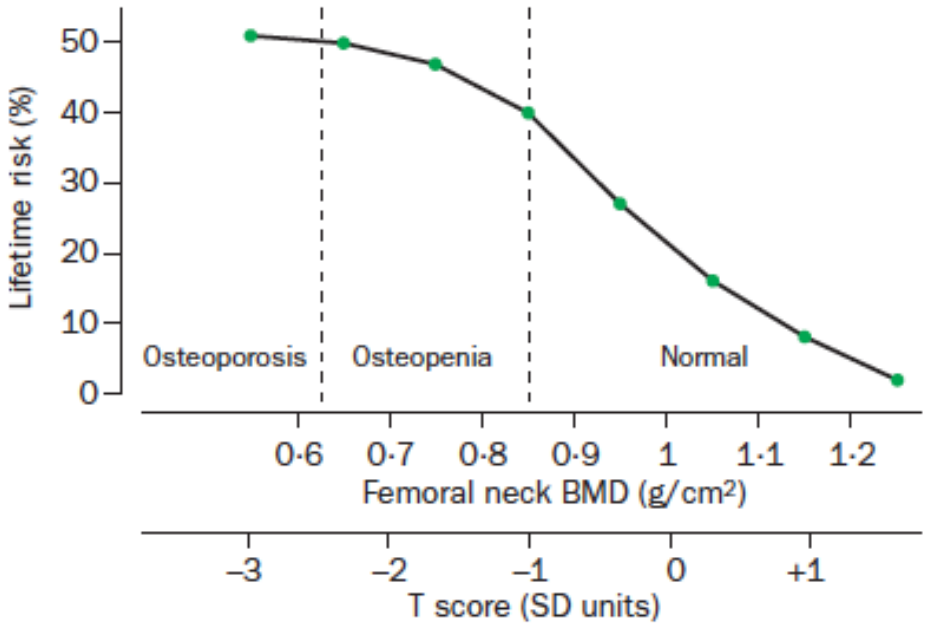


Figure 2.8 Remaining lifetime risk of hip fracture in women aged 50 years, according to bone mineral density (BMD) or T-score at the hip (Kanis, 2002, p. 1933)

2.2.3.2 Sex differences

The prevalence of osteoporosis is higher in women than men at 21.9% and 6.7% respectively in the UK population over aged 50 years (Hernlund et al., 2013). The incidence of the major osteoporotic fractures of the hip, vertebrae and forearm are all higher in women compared to men (over 50 years), when combined the estimated incidence of the fractures in women is more than double that of men

(155,264 vs 58,823) (Svedbom et al., 2013). The largest discrepancy in incidence of fracture at the forearm with estimated number of forearm fracture in at 59,007 in women and 9,913 in men (Svedbom et al., 2013). Data from (Curtis et al., 2016) also demonstrating a much higher incidence rate of any fracture in women over 50 years compared to men (155.4 vs. 71.8). However, The International Osteoporosis Foundation state that osteoporotic fractures in men account for 39% of all osteoporotic fractures (IOF, 2024). Although, the risk of fracture in men increases with age, the increase develops ten years later in men than in women, with a 60-year-old male having a 25% risk of fracture during his lifetime (IOF, 2024) and one in five men, over the age of 50, suffering from osteoporotic fracture (IOF, 2024).

2.2.3.3 Race/Ethnicity

There are ethnic differences in bone density and thus, fracture risk. Using data from NHANES III, a higher prevalence of osteoporosis and osteopenia, measured from femoral bone density, was present in non-Hispanic whites compared to non-Hispanic blacks in both men and women (Looker et al., 1997). Moreover, other studies have also reported, higher BMD in black men compared to white men at the radius, lumbar spine and femoral neck (Nelson et al., 1995). Population-level data on hip fracture incidence in California, showed that the highest rates were in white men, compared to black, Asian and Hispanic men, who recorded the lowest incidence (Silverman and Madison, 1988). In the same study, white women were also reported to have the highest incidence of hip fracture and Hispanic women the lowest (Silverman and Madison, 1988). In a review by Dhanwal and colleagues, the rates of hip fracture were highest in Caucasian and lowest in black ethic populations (Dhanwal et al., 2010). Furthermore, the highest fracture rates are reported in the Scandinavian countries and the lowest in Africa (Dhanwal et al., 2010; Cheng et al., 2011). This pattern is repeated in data from the UK with the lowest rates of fragility fractures in those over 50 years seen in black men and women and the highest fracture rates in white men and women (Curtis et al., 2016).

2.3 Hormones and Bone

2.3.1 Oestrogen and Androgens

The influence exerted by the sex hormones oestrogen and testosterone can be seen throughout the lifespan. Rapid bone growth at puberty in both males and females is attributed to the stimulatory effects of oestrogen and testosterone. The decline of oestrogen in women at menopause and the reduction of both testosterone and oestrogen concentrations in males in later life contributes to the development of osteoporosis (Manolagas and Almeida, 2019).

Oestrogens are largely secreted by the ovaries in women. In men, they are secreted by the testes (15%) but the predominant amount (85%) is produced by the aromatization of testosterone (Almeida et al., 2017). The main circulating androgen is testosterone and is produced by the testes (95%), and to a lesser extend the adrenal glands. In women the testosterone is produced by the ovaries, the adrenals and from the conversion of other sex steroids (Bellido and Hill Gallant, 2019). There are specific androgen receptors (AR) and oestrogen receptors (ERα and ERβ) found on the osteoblasts, osteoclasts, osteocytes and growth plate chondrocytes, indicating the direct influence on these key bone cells via the stimulation of their receptors (Vanderschueren et al., 2004; Baroncelli and Bertelloni, 2010). The sexual dimorphism during growth is believed to the different actions of the sex steroids on their different receptors, given the stimulatory effect of androgens and the inhibitory effect of oestrogens on bone growth (Bellido and Hill Gallant, 2019). In females, the oestrogen has a stimulatory effect on the endosteal surface via ERα and inhibitory effect on the periosteal surface via ERβ, thus creating cortical thickening with endosteal contraction (Bellido and Hill Gallant, 2019). In males, androgens stimulate the periosteal expansion via AR, resulting in cortical thickening with periosteal expansion (Bellido and Hill Gallant, 2019).

It is thought that both oestrogen and testosterone activate the GH/IGF – 1 axis and longitudinal growth (Bellido and Hill Gallant, 2019). In both sexes, the small concentrations in oestrogen at the start of puberty compared to the increased concentrations at the end of puberty exert a stimulatory or inhibitory effect, respectively on chondrocyte proliferation (Baroncelli and Bertelloni, 2010). The higher concentrations of oestrogen at the end of puberty leads to epiphyseal fusion and the cessation of longitudinal growth (Manolagas and Almeida, 2019). The higher concentrations of oestrogen in girls provides reasoning for the shorter growth period (Bellido and Hill Gallant, 2019), taller girls treated with oestrogens reduces growth velocity (Baroncelli and Bertelloni, 2010). Men with aromatase deficiency demonstrate a lack of epiphyseal fusion (Bellido and Hill Gallant, 2019).

The rapid decline in oestrogen in women following menopause has a direct effect on bone mass. Oestrogens influence the rate of bone turnover by restricting bone resorption via control of osteoclastogenesis and osteoclast apoptosis (Manolagas and Parfitt, 2013; Manolagas and Almeida, 2019). The initial rapid decline of estrogen coincides with a period of rapid bone loss followed by a more gradual loss (Manolagas and Almeida, 2019). The concentrations of circulating oestrogens are in the period following menopause, lower than that of men of the same age. In men the loss of testosterone is gradual, with oestrogen remaining largely constant, however concentrations of sex-hormone binding globulin (SHBG) increase resulting in a decline in the bioavailable concentrations of testosterone and oestrogen (Khosla et al., 1998).

2.3.2 Growth Hormone and Insulin-like Growth Factor

Growth hormone (GH) is produced and secreted by the anterior pituitary gland. The release of GH stimulates the release of Insulin-like Growth Factor – 1 (IGH-1) (Bellido and Hill Gallant, 2019). Growth hormone acts to influence bone growth via the GH/IGF-1 axis. GH is fundamental in longitudinal growth, with influence mediated by IGF-1 to increase bone formation by osteoblast proliferation and

differentiation (Giustina et al., 2008). It is thought that IGF-1 is inhibits cell apoptosis (Peruzzi et al., 1999). IGF-1 also acts to increase chondrocyte proliferation at the growth plate, moreover GH acts directly to stimulate prechondrocyte proliferation (Bellido and Hill Gallant, 2019).

A deficiency in GH in childhood presents with reduced height from lack of longitudinal growth and can affect bone mineral density (Bellido and Hill Gallant, 2019). Excessive secretion of GH is results in the disease state of Acromegaly. This is characterised by excess bone formation and bone turnover, resulting in increased BMD at cortical sites and reduced BMD at trabecular bone sites (Giustina et al., 2008).

2.3.3 Parathyroid Hormone (PTH)

Parathyroid hormone (PTH) is secreted by the parathyroid in response to low concentrations of calcium in the blood (Bellido and Hill Gallant, 2019). PTH has two opposing influences on bone biology whereby, it controls regulation of osteoclast differentiation by increasing the expression of receptor activator of nuclear factor κB ligand (RANKL) and promotes activation of osteoclasts (Nissenson, 2013). Chronically elevated concentrations of PTH increases bone remodelling rates with a resultant bone loss (Bellido and Hill Gallant, 2019). In contrast, PTH contributes to bone formation by increasing osteoblast number and delaying osteoblast apoptosis (Jilka, 2007). This promotes trabecular and cortical bone formation (Nissenson, 2013). Intermittent doses via daily injections of PTH has been shown to decrease the risk of fracture and increase bone density (Neer et al., 2001).

2.4 Muscle and Bone Interactions

It is difficult to discuss the physiology and pathophysiology of bone in isolation when it exists in concert with other organ systems in the human body. Chief of those is the musculoskeletal system. The

coordination and anatomical arrangement of muscle and bone together is such that the muscle exerts a mechanical and biochemical influence on the bone and in turn, the bone plays a part in releasing factors that affect muscle. The two systems are so closely related that those influencing factors that stimulate an anabolic or catabolic effect in bone will also exert a similar outcome in muscle. Bone and muscle growth tend to occur in similar fashion, stimulated by mechanical loading. Conversely, loss of bone and muscle tissue also occurs together across the lifespan. The trend of muscle mass accumulation and loss together with strength follows a similar trajectory to bone across the life course. The co-existence of low muscle mass together with low bone density poses a compounded risk to the individual with the increased risk of falling due to poor balance from reduced muscle mass, together with the weakened state of the bone to tolerate trauma without fracture (Girgis et al., 2014).

The attachment of muscle to bone provides the strain required to stimulate adaptation (see 'Mechanostat' work of Frost and Wolff, (Wolff, 1986; Frost, 1987)). The relationship between muscle and bone is seen as early as embryogenesis. In mouse models the muscle has exerts influence on the bone shape during development through contraction (Sharir et al., 2011). Moreover, when the muscle is reduced or non-existent (and therefore the muscle contraction is absent), retardation of bone formation is seen (Kahn et al., 2009; Nowlan et al., 2010). The accrual of lean muscle mass during adolescence tends to precede the accumulation of bone mass, potentially providing the stimulus for the increase in bone mineral density (Kirk et al., 2019; Girgis et al., 2014). Furthermore, higher volume of lean mass is associated with higher BMD and reduced fracture risk (Kaji, 2013). Both conditions, sarcopenia and osteoporosis tend to co-exist, or at least low levels of lean mass are seen in those with low bone mass and those with osteoporosis suffer from lower muscle mass (Yoshimura et al., 2017). (Girgis et al., 2014) proposes a possible theory to explain the controlling effect of muscle on bone, this being that the muscle initiates a chain of events when loaded, producing the biochemical factors that induce bone growth and modelling. When muscle loading is absent such as in zero gravity environments experienced by astronauts, bone loss is seen (Keyak et al., 2009).

The mechanostat theory explains part of the interaction between muscle and bone but the associations of bone strength parameters and muscle mass are not limited to those sites adjacent to the muscle. Appendicular muscle mass has been found to associate with cortical bone thickness at various sites including the lumbar spine and femoral neck (Lebrasseur et al., 2012). There are other examples of a relationship between muscle and bone, where muscle has aided in the healing of open fracture, performing better than skin and fascia alone in healing rate quality of repair in mouse models (Harry et al., 2008). Further to this, when muscle is compromised in compartments syndrome, this has a negative effect on bone union and healing time in tibia fractures (Reverte et al., 2011). (Girgis et al., 2014) suggest that muscle acts a 'second periosteum' assisting in bone repair.

2.4.1 Endocrine Cross-talk

Aside from the direct mechanical relationship between muscle and bone, there exists a less well understood relationship in endocrine cross-talk. Frost, proposed that Oestrogen played a regulatory role in the sensitivity of the mechanostat (Frost, 1999), supported by studies since demonstrating how Oestrogen acts to compound or mitigate the effect of strain (Riggs et al., 2002). Given that the apparent communication between muscle and bone is not restricted to the muscle and bone arranged adjacently, there is a systemic influence that exists. The muscle and bone function as endocrine organs (Kirk et al., 2019) and secrete a number of proteins that exert influence on other organs (Bonetto and Bonewald, 2019). These Osteokines and Myokines secreted by the bone and muscle respectively communicate in a bidirectional fashion. Muscle contraction can stimulate the release of such endocrine factors (Lombardi et al., 2019). There are a number of factors that notably act on muscle and bone, for example IGF-1, produced by bone, muscle also the liver appears to play a part in both muscle and bone function (Moriwaki et al., 2019). Circulating IGF-1, has an indirect effect on bone by stimulating muscle growth (Bonewald et al., 2013). Reduced levels of IGF-1 have been demonstrated in those with muscular atrophy as seen in disuse (Bonetto and Bonewald, 2019).

Myostatin, produced by the muscle, regulates muscle size by control of myoblast proliferation (Elkina et al., 2011). Increased mass is seen with low levels and conversely, atrophy seen in high levels (Bonetto and Bonewald, 2019). Upregulation of myostatin is seen in disease states such as cancer and HIV where muscle wasting occurs (Elkina et al., 2011). In bone, osteoclast recruitment is upregulated by myostatin (Bonetto and Bonewald, 2019) and where gene deficiency is seen, osteogenic activity is increased for improved bone repair and density (Kirk et al., 2019; Hamrick et al., 2007). Other myokines include Interleukin-6 (IL-6), which has a catabolic effect on muscle but an anabolic effect on bone IL-7, which impedes bone development and IL-15, which reduces bone resorption (Bonetto and Bonewald, 2019). Notable osteokines produced largely by bone tissue include Sclerostin, which down-regulates bone formation; Wnts , which initiates muscle formation and promotes bone formation (Bonetto and Bonewald, 2019). Osteocalcin has far reaching influence throughout the body, stimulating the secretion of insulin in the pancreas and increasing the sensitivity of insulin in adipose tissue, together with promoting testosterone production in the testes (Karsenty and Mera, 2018).

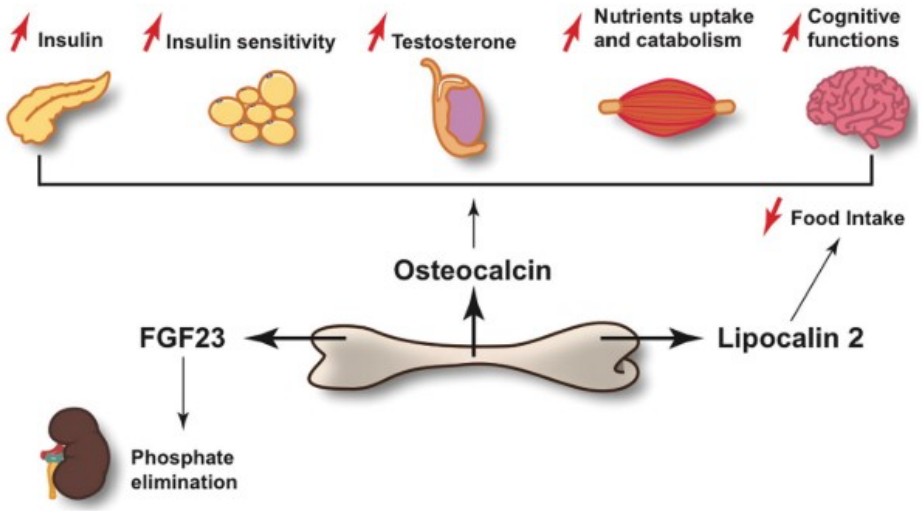


Figure 2.9 Representation of the physiological functions affected by osteocalcin (Karsenty and Mera, 2018, p. 44)

The complex interplay of numerous biochemical factors secreted by muscle and bone demonstrates the influence both locally and systemically throughout the body. This highlights the importance of not only the obvious mechanical relationship within the musculoskeletal system, but also the endocrine, paracrine and autocrine status of the muscle and bone as endocrine organs, not limited by their obvious anatomical role in locomotion.

2.5 Sarcopenia

Unlike osteoporosis, which has a clear definition and method of assessment, sarcopenia has never established a specific definition, only working definitions and the like. Initially the term was used to describe the loss of lean body mass, predominantly associated with aging (Rosenberg, 1997). A working definition was then developed in 2010 by the European working Group on Sarcopenia in Older People, which required the presence of low muscle mass and function to diagnose sarcopenia, where muscle function is strength and/or performance (Cruz-Jentoft et al., 2010). The updated version of this definition from the same group described sarcopenia as that of a skeletal muscle disorder associated with various undesirable outcomes, including fractures, falls and other frailty-related conditions (Cruz-Jentoft et al., 2019). This latest definition proposes that the focus be on muscle strength rather than mass, given it is a better predictor of adverse outcomes (Cruz-Jentoft et al., 2019).

Several measurements can be used to determine the presence of sarcopenia. A ratio between appendicular skeletal muscle mass (ASM) and height falling 2.5 standard deviations below the expected young adult norm can be used to determine the clinical evidence of sarcopenia (Bonetto and Bonewald, 2019), in a similar way to the identification of osteoporosis using bone density. The European Working Group has a number of cut-off points for the identification of sarcopenia, including Grip strength, chair stand, ASM, ASM/height^2 and gate speed (Cruz-Jentoft et al., 2019). The different

areas of assessment used to define sarcopenia can be problematic, especially as there is no clearly defined clinical endpoint such as fracture with osteoporosis. There are several potential adverse clinical outcomes including falls, fracture, immobility, disability and death (Edwards et al., 2015).

The mechanism of development occurs due to an imbalance between protein synthesis and degradation, mirroring that seen when the rate of bone loss overtakes that of bone production in osteopenia and osteoporosis. This has long been associated with ageing (Morley et al., 2001) but is now suspected to be initiated earlier in life (Sayer et al., 2008). Sayer and colleagues pointing out that while loss of lean mass is driving the decline, a critical factor is the attainment of peak mass and strength in early adult life. Again, mirroring the life cycle of bone accumulation and inevitable loss in later life, the inevitable loss of muscle mass could be offset by optimum lean mass (bone and muscle) in the developmental stages of growth. While the lean mass is important, there is a suggestion that muscle strength and power are more sensitive to the physiological decline in muscle quality and have been seen to decline at a faster rate than mass alone (Edwards et al., 2015). Of note, there is a positive association between birth weight and strength throughout the lifecourse (Dodds et al., 2012). Handgrip strength is an important indicator of functional capacity and low grip strength is associated with various undesirable outcomes including longer hospital stays, reduced quality of life and death (Cruz-Jentoft et al., 2019) and may provide a superior forecast of outcomes than muscle mass (Lauretani et al., 2003) and provide a surrogate for other, more complex measures of strength (Cruz-Jentoft et al., 2010)

2.5.1 Muscle Quality

Muscle quality as a term can be applied to describe the composition of the muscle (Cruz-Jentoft et al., 2019), but is also used to describe the ratio of strength to mass (Lynch et al., 1999). The muscle changes seen in sarcopenia are not just limited to mass and a decline in said mass but also a reduction in quality. Fatty infiltration of the muscle tissue takes place, increasing the fat mass (Fielding et al.,

2012). This can also mitigate the apparent loss of lean mass, if using a simple weight measurement as the loss of lean mass is countered by the increase in fat mass. Measurement of lean mass and fat mass could be discerned by employing such methods as DXA, moreover, utilising the same method of assessment as bone density assessment in those at risk of osteoporosis and sarcopenia. Muscle loss has further reaching effects given, muscles are known to produce myokines that have influence more broadly in the musculoskeletal system. These myokines (see muscle-bone crosstalk) can affect a number of outcomes including, volume of muscle mass, the response to exercise and insulin sensitivity (Demontis et al., 2013). Therefore, a reduction in muscle, is a reduction in myokine production (Frontera, 2019).

As there are a number of parameters used to determine the presence of sarcopenia, as opposed to a single value when diagnosing osteoporosis, determining prevalence can be more difficult. However, several studies have reported the prevalence of sarcopenia and although different measures using differing tools have been employed to measure the various parameters, these have been synthesised using meta-analysis. From a meta-analysis of 35 studies, using the European Working Group on Sarcopenia (EWGSOP), The International Working Group on Sarcopenia (IWGS) and the Asian Working Group for Sarcopenia (AWGS) definitions of Sarcopenia of low muscle mass and strength or physical performance in individuals over 60 years, prevalence has been reported at 10% in men and women (Shafiee et al., 2017). In a more recent systematic review, using a number of different definitions of sarcopenia including the EWGSOP definition, ALM/height and ALM/weight, the prevalence varied from 9.9% - 40.4% (Mayhew et al., 2019).

2.6 Osteosarcopenia

The anatomical and physiological relationship between bone and muscle make it impossible to separate the interactions of one without the other. The conditions of sarcopenia and osteoporosis

share common mechanisms of development and where the two conditions co-exist, this is known as Osteosarcopenia.

The attainment of peak bone and muscle mass in early adulthood is followed by the gradual decline of both, following similar declining trajectories. The loss of both tissues does not occur in isolation, rather a multifaceted chain of events contributing to a reduction in mass and function (Pasco, 2019). The existence of osteoporosis is more likely in an individual with sarcopenia (Verschueren et al., 2013) supported by data from the Korea National Health and Nutritional Examination Surveys (KNHANES) demonstrating a relationship between low lean mass and osteoporosis (Kim et al., 2014). The muscle-bone relationship is further demonstrated with a positive association of muscle size and strength to bone mass and strength (Edwards et al., 2013). Men with low appendicular lean mass have been observed with narrower bones, thinner cortices and lower section modulus in the distal radius and femoral neck (Szulc et al., 2005). Moreover, the same study also demonstrating associated diminished balance and increased falls risk in those with lower appendicular skeletal mass. Thus, sarcopenia is a one of a number of risk factors for hip fracture, predicting fracture risk in the aging population (Oliveira and Vaz, 2015).

It is not fully understood whether one condition accelerates the progression of the other however, the development of Osteosarcopenia from sarcopenia was more apparent than from osteoporosis (Huo et al., 2015). This provides a suggestion that muscle loss does in fact lead to bone loss (Pasco, 2019). There remains though, the question of whether the impact of lifestyle on bone and muscle mass and strength in early life is irreversible. The attainment of peak bone, lean mass and strength being the most important factor in determining bone health in older age, in the absence of pathological bone conditions is not absolutely certain, while it clearly exerts a huge influence on outcomes in later life. However, before lifestyle factors have contributed to the volume of bone mass, birth weight has been identified as a strong predictor of bone mineral content (BMC) and bone mineral

density (BMD), accounting for 95% of the BMC variation and 86% of total body BMD variation, measured by DXA (Koo et al., 1996). Furthermore, birth weight was significantly ($p<0.001$) associated with fat free mass and grip strength in later life (age: 64.3 years) in a UK cohort study (Sayer et al., 2004). The presence of more favourable measures early in life, have been found to track into later life (Dennison et al., 2005).

Given that early life factors influence so much of later life outcomes, there are more transient effects from influences throughout life that impact bone loss and lean mass. It is well established from studies on bed rest, that disuse results in the loss of bone, atrophy of muscle tissue and loss of strength (Kortebein et al., 2007; Wall et al., 2013). Lack of exercise and inactivity has been linked with lower lean mass compared with older men who participate in regular exercise (Szulc et al., 2004), demonstrating the temporary nature and complex interplay of bone mass, lean mass, and strength (ref). Conversely, the opposite can be seen when individuals engage in physical activity. Loss of mass can be mitigated against, when there is participation in resistance training and high impact exercise (Kramer et al., 2017).

2.7 Falls

There are features that are independent of bone biology that contribute to fracture, of those the most central, it could be argued is the risk posed by falling. A complex risk factor for fracture, in that a number of aspects contribute to an individual falling and yet there are further contributing dynamics that increase the chance of an individual suffering a fracture as a result of the fall. Falls contribute to non-vertebral fracture in approximately 80% of fractures with over 90% of hip and wrist fractures being attributed to falling (Cummings and Nevitt, 1994) and yet falls result in serious injury in a much smaller proportion approximately 10% (Tinetti and Williams, 1997).

The mechanics of the fall that result in fracture in large part, determines the fracture site. The point of impact, direction of fall, surface type, and the potential energy in the fall (Cummings and

Nevitt, 1994) all contribute to fracture, and are unrelated to skeletal biology. Hip fracture occurs when the individual falls to the side or straight down and lands on the hip (Nevitt and Cummings, 1993; Hayes et al., 1993). Wrist fractures most commonly occurred due to a fall onto an outstretched hand (Cummings and Nevitt, 1994), but interestingly, were more likely to be a result of falling backwards rather than forwards (which had a decreased risk of wrist fracture) or sideways (Nevitt and Cummings, 1993). The risk of both hip and wrist fractures increased when falling onto a hard surface, particularly hip (odds ratio: 2.8) more so than wrist (odds ratio: 1.3) (Nevitt and Cummings, 1993). Hip fractures tend to occur in less active individuals (Bischoff-Ferrari, 2011).

The height of the individual has been associated with increased risk of hip fracture. (Hayes et al., 1993) demonstrated that those elderly residents suffering fracture after a fall were taller ($p<0.001$) than those without fracture and that the potential energy of the fall was higher but not significantly so (Hayes et al., 1993). Taller women were also at increased risk of hip fracture when falling on the hip with an odds ratio of 1.5 per 1 standard deviation increase in height (Nevitt and Cummings, 1993).

The effect of potential mitigators of force such as soft tissue padding in order to reduce fracture risk has been investigated. Peak forced was reduced and tissue energy absorption increased as tissue thickness increased (Robinovitch et al., 1995). In the Framingham Study, trochanteric soft tissue thickness was significantly ($p<0.001$) less in women suffering hip fracture compared to the non-fracture group (Dufour et al., 2012). However, there were no significant differences the soft tissue thickness of men in the same study.

Aside from bone density, osteoarthritis (OA) has also been implicated as a risk factor for falling with an increased risk of falling in those with self-reported OA and notwithstanding having superior BMD, there was not a significantly reduced risk of fracture (Arden et al., 1999). An association of knee pain with increased risk of falling and fracture has been demonstrated by Arden and colleagues (Arden

et al., 2006) with greater severity of pain associated with increased risk. Also revealed was an increased risk of nonvertebral fracture in those with knee OA (Arden et al., 2006).

2.8 Athlete health

The mechanism of bone mass and lean tissue accumulation in humans from birth until peak mass in adulthood have been established and critically unpacked/interpreted in previous sections. There follows the inevitable loss of lean tissue following attainment of peak mass. However, given that exercise in the form of resistance and impact work can promote mass development and mitigate loss, athletes would seem at an advantage compared to those that do not engage in regular physical activity. However, it is not known if the benefits gained remain throughout the life cycle or are they predominantly a temporary phenomenon.

Competitive athletes from a variety of sports have demonstrated superiority in several measures compared to the non-athletic population. The life expectancy for athletes in general, is greater than for the non-athlete population (Clarke et al., 2012; Garatachea et al., 2014; Lemez and Baker, 2015). Cardiovascular disease risk was reduced in those engaged in physical activity, although this association was less when the physical activity was not maintained in later life (Paffenbarger and Lee, 1998). An early study (Polednak, 1972) found there was no protective effect from College sports with athletes having a greater mortality risk from cardiovascular disease than non-athletes. In contrast to this, vigorous exercise reduces mortality, largely due to the lower cardiovascular disease mortality in endurance athletes (Teramoto and Bungum, 2010). There is a difference in the incidence of such disease in different sports, between endurance athletes and power athletes. A substantial volume of work was performed on former athletes who represented Finland in a variety of sports between 1920 and 1965 (Kujala et al., 2003). The relative risk of such cardiovascular diseases was less than 1, when compared to controls for each type of athlete group (endurance sports, mixed sports, and power sports), although, in the power athletes there was a higher relative risk, 1.21 for diabetes (Kujala et

al., 2003). A lower incidence of hypertension has been reported in endurance and mixed sports athletes, compared to the power sports athletes (Hernelahti et al., 2002). Moreover, compared to controls, former elite athletes have been shown to have more favourable measures of metabolic health including lower body fat percentage (Laine et al., 2016). Additionally, lower body fat percentage was associated with current levels of physical activity, highlighting the importance of current levels of activity which compound the benefits of previous participation.

By most measures of health, former athletes in general, tend to fare better than non-athletic controls. However, aside from cardiovascular factors, musculoskeletal health is impacted by participation in sports activity earlier in life. For example, former competitive athletes were at increased risk of hospital admission for Osteoarthritis (OA) of the lower limb joints (U M Kujala et al., 1994; Kujala et al., 2003). Furthermore, active athletes have demonstrated increased risk for radiographic knee OA (odds ratio: 2.8) compared to controls, with further increased odds when a history of ACL surgery is noted (Roemer et al., 2015). Ultimately, the end result of severe arthritis is managed by joint replacement (Bachmeier et al., 2001). Knee and hip injury were associated with later joint replacement in retired National Football League athletes (Davies et al., 2019).

Jumping height in a variety of former athletes was less in those with lower limb OA than those without but was still superior to healthy, sedentary controls and comparable to healthy, active controls (Urho M Kujala et al., 1994; Kettunen et al., 1999). However, former endurance and power athletes reported increased odds ratio for hip osteoarthritis, but conversely, reduced odds ratio for hip pain compared to controls (Kettunen et al., 2001). Though, for knee OA and knee pain, both team sport and power sport athletes reported higher odds ratios than controls (Kettunen et al., 2001). This was reflected in higher odds ratios for self-reported knee disability in former team sport and power athletes (Kettunen et al., 2001). In contrast, the remainder of former athletes (endurance, track and field, and shooters)

all demonstrated lower odds ratios for knee disability, added to all athletes reporting reduced odds ratios for hip disability (Kettunen et al., 2001).

2.9 Rugby Player Health

There have been few studies to specifically investigate the longevity of retired rugby players. In those that have, there was no demonstrated difference in life expectancy (Rook, 1954; Beaglehole and Stewart, 1983). Participation in the game and the training involved does confer certain benefits on the players. In studies that have examined the characteristics of currently active rugby players, rugby players were found to have superior strength, bone density and lean mass when compared to controls (Elloumi et al., 2009; Hind et al., 2015). The longevity of these characteristics is unknown, and it is difficult to ascertain from the current literature if the benefits of such participation will remain following retirement. The nature of sport and particularly impact sports such as Rugby does contain risk of injury and while it is difficult to compare the injury rates of different sports, the incidence of injury is high in both codes of rugby. For the English Premiership rugby union, the mean injury rate from 2002-16 was 84/1000 hours of match play (England Professional Rugby Injury Surveillance Project Steering Group, 2018). Compare this to professional football with an injury incidence of 27.5/1000 hours of match play (Ekstrand et al., 2011). It is unclear if the injuries experienced while active, exert a long-term effect on the musculoskeletal health of the player and whether this mitigates the benefits gained from taking part in physical activity during youth and young adulthood.

There have been documented catastrophic spinal injuries in rugby players, although they are relatively rare (Fuller, 2007). The spine, however, is subject to trauma from the impact of rugby. When evaluated clinically and radiologically, retired rugby players were found to suffer from neck pain in greater number compared to controls (51% vs. 32%) (Brauge et al., 2015), and show greater severity of foraminal stenosis than controls (Brauge et al., 2015). In currently active rugby players 121 grade 1 to grade 3 vertebral abnormalities were found in 51 players using lateral vertebral assessment imaging

by DXA, although there was no control group for this study (Hind et al., 2014). Furthermore, evidence of previous clinical fracture was identified on a lateral spine radiographs of 8 out of 150 players vs. 1 from the control group of the same number (Scher, 1990). This same study demonstrating significantly more rugby players showing degenerative changes compared to relatively few in the age-matched control group (Scher, 1990).

There are few studies on the health of rugby players post-retirement. A small study (n=28) questioned former professional rugby league players on the effects of injuries sustained during their playing career (Meir et al., 1997). From this cohort, 23 had suffered injuries that resulted in long term conditions (as determined by medical examination), including arthritis, chronic back pain and restricted joint mobility (Meir et al., 1997). In another questionnaire-based study, of 390 ex-players, 22 (9%) reported significant adverse effect from injury on various aspects of life in retirement, including on employment, family life and health (Lee et al., 2001). A further comprehensive study, again from questionnaire, explored various aspects of retired rugby players (n=259) health compared with general population cohort data (Davies et al., 2017). Standardised morbidity ratios (SMR) were calculated for several physician diagnosed conditions, most notably, increased prevalence of osteoarthritis (4.00) and joint replacement (6.02) and of relevance to the present research project, osteoporosis (2.69) (Davies et al., 2017). Hind et al (2020) determined from a questionnaire, that prevalence of osteoarthritis was greater in former elite rugby players compared with non-contact athletes (51% vs 22%). Furthermore, higher prevalence also seen in other studies, compared with population data (Le Roux et al., 2023).

3.0 Justification of measures employed in this thesis

The study of musculoskeletal physiology in retired rugby players addresses the complex interplay between physical activity, ageing, and the long-term effects of intense athletic training on the human body. This chapter presents a comprehensive account of the methodology employed in this doctoral research, which aims to deepen the understanding of the musculoskeletal health and functional abilities in retired athletes.

The overarching objective of this doctoral research was to investigate aspects of musculoskeletal physiological health that manifests in retired rugby players, shedding light on the potential consequences of their athletic careers and the subsequent transition to a different lifestyle. By exploring the intricacies of musculoskeletal physiology in this specific population, the research in this thesis creates new knowledge to guide healthier aging among retired athletes. To achieve this, a multifaceted methodology was employed. This chapter will outline the various components of the research approach, including participant recruitment and selection criteria, data collection methods, and analytical procedures. The chapter will also elucidate the selection and implementation of various assessment tools, such as a general health questionnaire (GHQ), DXA imaging and neuromuscular analyses. Data analysis and statistical methods will be detailed in terms of the utilisation of appropriate statistical tests and software packages, as well as the interpretation of results, to ensure the validity and reliability of the findings. It should be noted that a separate methods section is included for each subsequent chapter.

3.1 Assessment of Bone Mineral Density

DXA is the current, preferred technique for the measurement of BMD for the clinical diagnosis of osteoporosis (Blake and Fogelman, 2007) and thus the most commonly employed method for fracture risk assessment (Jain and Vokes, 2017). It is widely used both clinically and in research, measuring a variety of populations including the elderly and the athletic. The densitometry data is acquired using X-ray beams of two different energies generated by an x-ray tube, transmitted through the subject and received by the detector to form the image and make the necessary calculations. The X-rays are attenuated by the body tissues of the subject. It is this attenuation by the subject of the two energies that allow the differentiation of tissue components to be made and a subsequent estimation of tissue density (Laskey, 1996). The bone mineral content (BMC), bone area and areal BMD are calculated, and it is areal BMD that is used as a surrogate for bone strength (Adams, 2013).

Using the measured areal BMD ($g.cm^{-2}$) a clinically valuable T-score or Z-score is calculated, using reference databases. The T-score is the difference between the subjects BMD and that of the mean healthy young adult, matched for gender and ethnicity, expressed relative to the young adult population (Blake and Fogelman, 2007; Watts et al., 2013). The Z-score is calculated using the same method but is expressed relative to the healthy adult subject, matched for age, sex and ethnicity (Blake and Fogelman, 2007). The World Health Organisation defines osteoporosis as a T-score of -2.5 standard deviations (SD)below the young adult mean. Low bone mass or osteopenia was defined as a T-score of below -1 SD but above -2.5 (World Health Organisation, 1994).

DXA scans have a number of advantages including low radiation dose (1-6μSv) (Adams, 2013; Damilakis et al., 2010), short scan time and good precision (Blake and Fogelman, 2007). BMD results can be incorrectly inflated in the presence of increased density, such as that from vertebral fractures, osteoarthritis, aortic calcification and metallic artefacts (Adams, 2013). These anomalies can be mitigated by vigilant image analysis from the operator.

3.2 Body Composition

DXA provides a three compartment (bone mineral content, fat and lean mass) method of assessing body composition; (Bonnick and Lewis, 2013). The distribution of fat, lean and bone mass can also be determined (Bonnick and Lewis, 2013). Excellent precision has been demonstrated with % coefficient of variation (CV) values of 0.82% for total body fat and 0.6% for total body bone mineral content (Hind et al., 2011). While it is not considered the 'gold standard' technique for assessing body composition (Messina et al., 2018), a strong correlation has been demonstrated between DXA and CT to assess fat mass (Bredella et al., 2010) and also between DXA and MRI (Kullberg et al., 2009). Furthermore, significant correlations were found between CT-measured thigh muscle mass and DXA measured thigh fat-free mass (Levine et al., 2000).

3.3 Bone Specific Physical Activity Questionnaire (BPAQ)

The Bone Specific Physical activity Questionnaire (Weeks and Beck, 2008) is a method of self-recording current and historical levels of physical activity. The method has been developed to account for the bone loading capability of different exercises and the relationship to BMD (Kim et al., 2016). It has been shown that athletes from high impact sports such as gymnastics have the highest BMD versus those from non-weightbearing sports such as swimming who have the lowest (Kohrt et al., 2004). Exercise can impose strain on the bone and depending on the type of exercise can produce an osteogenic effect (Mantovani et al., 2018). In particular high impact exercise, exerting a high magnitude of force has been shown to increase bone density (Bassey and Ramsdale, 1994; Weeks and Beck, 2008).

The BPAQ is a method to quantify the osteogenic effect of exercise and its relationship with BMD (Kim et al., 2016). Bone responds to stimuli in such a way that is unique to the type of stimulus (Weeks and Beck, 2008). The osteogenic index allows for estimation of bone formation with exercise (Turner and Robling, 2005). Large magnitude forces applied to the skeleton at fast rates are though to offer the optimal benefits (Weeks and Beck, 2008). Given that bone strain is difficult to measure

at sites such as the femoral neck or lumbar spine, ground reaction forces (GRF) offer a surrogate measure of bone strain. The BPAQ uses an algorithm developed using weighting factors of load intensity (peak magnitude of stress multiplied by loading frequency) based on the osteogenic index (Turner and Robling, 2005; Weeks and Beck, 2008).

In the absence of the ability to appropriately record strain on bone in vivo, alternate measures of ground reaction forces have been used to develop the BPAQ algorithms and calculate a BPAQ score for a given activity history (Weeks and Beck, 2008). The calculation accounts for when the activity has taken place in the lifespan i.e. in childhood or adulthood and also the most recent activity. The scores are produced: past BPAQ, current BPAQ and total BPAQ (which is an average of past and current). Thus a 'bone relevant' activity history is gathered, and a commensurate score is assigned (Weeks and Beck, 2008). Relevant to the current thesis, a relationship between the BPAQ score and BMD has been demonstrated in middle aged men (Bolam et al., 2014) and young men (Weeks and Beck, 2008).

3.4 Balance Testing

The assessment of balance is of paramount importance in understanding and evaluating the functional capacity of individuals, as well as identifying potential risk factors for falls and musculoskeletal injuries. Balance, the ability to maintain postural stability and control during static and dynamic activities, is a complex motor skill that involves the integration of sensory information, neuromuscular coordination, and cognitive processing. Accurate and reliable assessment of balance is crucial for evaluating the musculoskeletal health and function of retired athletes. Static and dynamic balance were assessed with the Neurocom VSR sport force plate and Balance Manger software. The VSR sport offers a number of assessment protocols:

The Stability Evaluation Test (SET), Unilateral Stance (US) for functional assessment; the Modified Clinical Test of Sensory Interaction on Balance (mCTSIB) for sensory assessment; the Limits of Stability

(LOS), Rhythmic Weight Shift (RWS) and Weight Bearing Squat (WBS) for voluntary motor assessment. (Natus Medical Incorporated, 2014b)

Balance is maintained if the centre of gravity (COG) is preserved vertically over body's the base of support (Nashner, 1997). Gravitational force acts downward on the body and if there is displacement of the COG (sway) from its position above the base of support, angular acceleration is initiated with the larger the displacement angle resulting in larger acceleration (McCollum and Leen, 1989).

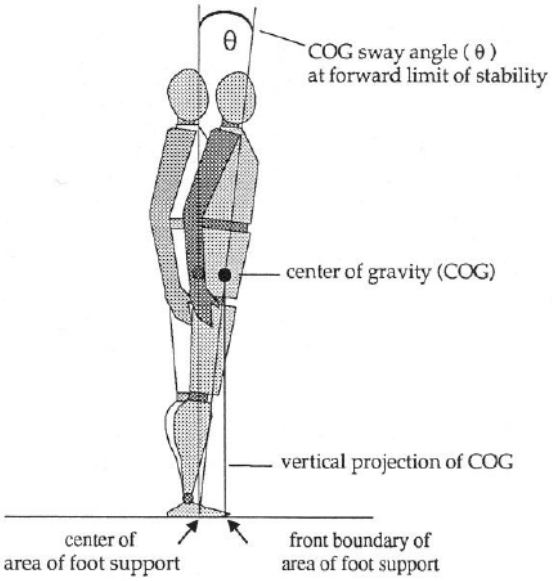


Figure 3.1 (Natus Medical Incorporated, 2013, p. 2)

When an individual is working to maintain the COG above the base of support, this cannot be done without movement. There is a sway from side to side and forwards and backwards (Nashner, 1997). The limits of stability (LOS) is the theoretical maximum sway possible without falling (Natus Medical Incorporated, 2013; Nashner, 1997; Koozekanani et al., 1980). When the limits of sway exceed the limits of stability the individual loses balance (Nashner, 1997). There are two issues of note: the limits of sway will be as large as the LOS when the COG is preserved over the base of support. However, if the COG is offset then it is closer to the LOS in one direction and balance will be disturbed as it will take less displacement to exceed the LOS than a correctly aligned COG (Nashner, 1997). The second issue being that the sway frequency impacts the actual LOS (Nashner et al., 1989) with higher sway

frequencies the LOS is subsequently reduced (Nashner, 1997). Increased sway velocity means poorer balance (Wang et al., 2018).

This complex task of balance maintenance is achieved through a co-ordinated effort of processing information from the visual, vestibular and somatic senses (Nashner and McCollum, 1985). The force plate tracks the movement of the centre of pressure and from this, the centre of gravity and sway angle are calculated, subsequently producing a sway velocity (Natus Medical Incorporated, 2014a).

3.4.1 Stability Evaluation Test (SET)

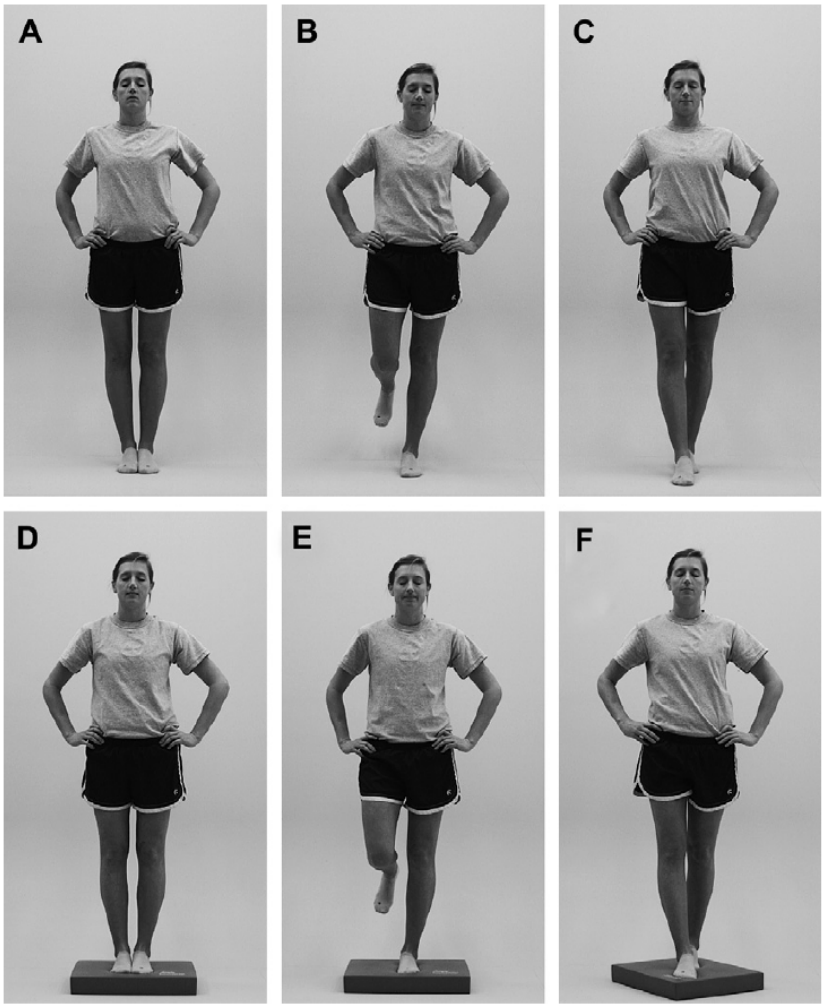


Figure 3.2 The Balance Error Scoring System (Guskiewicz, 2011, p. 96).

The SET is an objective version of the established Balance Error Scoring System (BESS). The BESS is used in the management of concussion and is a low cost option for assessing postural deficits in the

athletic population (Guskiewicz, 2011). The SET provided by Neurocom is an objective version of the BESS, adopting the same positions for the testing protocol as those executed in the BESS. However, unlike the BESS, sway velocity is recorded in place of the subjective scoring system. The SET has demonstrated good reliability (Williams et al., 2017). The participant performs six trials: the double stance, single leg and tandem stance on a firm surface for 20 seconds each. The same three trials are completed on a foam surface. All trials are performed with eyes closed. Image:

From the SET a sway velocity (degree per second) is presented for each trial.

3.4.2 Modified Clinical Test of Sensory Interaction on Balance (mCTSIB)

The mCTSIB has been designed as a simplified version of the sensory organisation test (SOT) (Natus Medical Incorporated, 2014a). The SOT being the gold standard of balance assessment. The participant is positioned on the force plate on both feet a given distance apart (as per on-screen instruction), depending on height, with a wider stance for taller people. The integration of the vestibular, vision and somatosensory systems is required for correct balance control (Cimino et al., 2018). In static tests the COG sway is a reflection of the competence to organise the input from the sensory systems in order to preserve balance control (Cimino et al., 2018). The mCTSIB has demonstrated reliability and correlated with other clinical balance measure (Cimino et al., 2018).

The participant undergoes 3 trials each under 4 different conditions: eyes open on the firm surface, eyes closed and firm surface, eyes open on the foam surface and eyes closed on the foam surface. Each of the 12 trials last 10 seconds. Results are given as a mean COG sway velocity for each condition and also a composite sway velocity.

3.4.3 Limits of Stability (LOS)

Participants stand in a given position on the force plate and are instructed to move in 8 given directions. The centre of pressure (COP) is represented on screen as an icon. The object is to move the

icon as quickly and smoothly as possible to the given target without any foot movement (Tsang and Hui-Chan, 2003). The positions are set at the theoretical limits of stability. The measurements given for this test are movement velocity, reaction time, end point excursion, maximum excursion and directional control. The LOS has been shown to be reliable with good test-test reliability (Lininger et al., 2018; Pickerill and Harter, 2011) and compared well, providing more dynamic postural stability information that other equipment (Pickerill and Harter, 2011). Poorer performance in the LOS test are suggestive of poor motor control or lower extremity weakness (Schieppati et al., 1994; NeuroCom International Inc., 2007). Deficiencies in the LOS may have implications for activities of daily living such as reaching for items (NeuroCom International Inc., 2007).

3.5 Muscle Strength

Muscle strength can be assessed in a number of ways. Grip strength using a handheld dynamometer is widely used in clinical and research settings (Beaudart et al., 2019). Hand grip strength testing serves as a valuable tool in assessing upper body strength and overall physical function, with numerous applications in various populations, including athletes and non-athletes. In the context of the current research study, comparing retired rugby players to non-athletes, hand grip strength testing provides useful insights into the potential long-term effects of athletic training and its impact on musculoskeletal health. This rationale outlines the specific justifications for incorporating hand grip strength testing in this comparative study.

Indicator of General Muscular Strength: Hand grip strength is widely recognized as a reliable indicator of overall muscular strength and function (McGrath, 2021). It reflects the strength and coordination of the muscles in the forearm, wrist, and hand, which are crucial for numerous daily activities, including grasping objects, manipulating tools, and maintaining postural stability. By measuring hand grip strength, it is possible to assess the general muscular strength of individuals and compare the strength levels between retired rugby players and non-athletes (Beaudart et al., 2019).

Reflection of Upper Body Strength: Rugby, being a physically demanding sport that involves intense upper body engagement, places substantial demands on the muscles of the arms, shoulders, and hands. Hand grip strength is closely related to the strength of these upper body muscles, making it an important measure to evaluate the possible long term impact of rugby-specific training on the strength and functionality of these muscles in retired players. Comparing hand grip strength between retired rugby players and non-athletes can elucidate the potential long-term effects of rugby training on upper body strength.

Correlation with Overall Physical Performance: Hand grip strength has also been shown to correlate with various aspects of physical performance, such as functional mobility, balance, and overall muscle power (Beaudart et al., 2019). Assessing hand grip strength in retired rugby players and non-athletes can help with understanding of how differences in upper body strength relate to overall physical performance in these two populations. This information is particularly relevant in identifying potential functional limitations or performance advantages associated with rugby training.

Although there are other measures which could offer additional and potentially superior predictor of health such as leg extension power (Beaudart et al., 2019), the handgrip strength assessment does provide procedural ease. This is relevant given the variety of testing taking place. It is advised that 3 measurements on each hand are taken (Beaudart et al., 2019).

Widely-used Screening Tool for Musculoskeletal Health: Hand grip strength is widely used as a screening tool for musculoskeletal health, with lower grip strength associated with an increased risk of musculoskeletal disorders and functional limitations (Sousa-Santos and Amaral, 2017; Beaudart et al., 2019; McGrath, 2021). By comparing hand grip strength between retired rugby players and non-athletes, it is possible to explore if rugby training may have a long-term protective effect on musculoskeletal health. The Takei hand grip dynamometer has demonstrated reliable performance and validity, with validity coefficients above 0.90 ($p<0.001$) (Balogun and Onigbinde, 1991).

In conclusion, hand grip strength testing provides valuable insights into upper body strength, physical performance, and musculoskeletal health in retired rugby players compared to non-athletes. By incorporating this assessment into the research, it was possible to gather objective data to support the evaluation of the possible long-term effects of rugby training on the upper body strength and overall musculoskeletal health of retired athletes.

Chapter 4.0 Study 1: Vertebral anomalies in retired rugby players and the impact on bone density calculation of the lumbar spine

This study has been published in a peer reviewed journal:

Entwistle, I., Hume, P., Francis, P. and Hind, K. 2021. Vertebral Anomalies in Retired Rugby Players and the Impact on Bone Density Calculation of the Lumbar Spine. *Journal of Clinical Densitometry*. **24**(2), pp.200–205.

4.1 Introduction

Dual energy X-ray absorptiometry (DXA) is the current gold standard for bone mineral density (BMD) assessment in the diagnosis of osteoporosis, given that BMD is a strong predictor of fracture risk (Kanis, 2002; Lewiecki et al., 2016). Population-based data indicates that for every standard deviation reduction in BMD, there is a 1.5 to 3-fold higher risk of fracture (Marshall et al., 1996; Kanis, 2002). Along with scans of the total hip or femoral neck, the lumbar spine is also routinely assessed, as recommended by the International Society for Clinical Densitometry (ISCD) and the World Health Organisation (World Health Organisation, 2007b; Blake et al., 2013; ISCD, 2015). For both lumbar spine and hip scans, BMD is compared to either the young adult mean or age and sex-matched reference data. In post-menopausal women and men at or over the age of 50 years (≥50 y), a T-score is calculated by comparing the BMD to the young adult mean (ISCD, 2015). In premenopausal women and men under the age of 50 years (<50 y), a Z-score is calculated according to age and sex-matched reference data, and adjusted for weight and ethnicity (ISCD, 2015).

A high level of scrutiny is required during the scan acquisition and subsequent interpretation of lumbar spine scans to obtain an accurate BMD. This is because lumbar spine BMD can be subject to artificial elevation in the presence of various disease states, most notably osteoarthritis and other degenerative conditions (Yu et al., 1995). The presence of structural abnormalities such as fracture,

osteoarthritis, osteophytes and sclerosis, contribute to the assessed bone density, and result in spurious increase (Adams, 2013). Osteophytes cannot be measured independently of the vertebral body and so elevate the recorded bone density (Pye et al., 2006). Therefore, it is important that DXA technicians identify and exclude such anomalies from the scan images. It is recommended that adjacent vertebrae with a T-score difference of >1 SD, should be excluded from the lumbar spine bone density measurement. The visible presence of a structural abnormality would also provide a justification for exclusion of a vertebrae (ISCD, 2015). There are currently no recommendations for men aged under 50 years, specifically for the use of the Z-score for removal of vertebrae from the clinical evaluation, and this is likely because degenerative changes and artefacts at the spine are more common with age (Adams, 2013).

Approximately one third of the UK population over 45 years seek treatment for osteoarthritis (Arthritis Research UK, 2013) and contact sports populations appear to be at particularly high risk. Across multiple injury types, past participation in rugby union and rugby league, particularly at elite level, has been associated with a high cumulative musculoskeletal injury load and a continued impact of previous injuries post-retirement including osteoarthritis (Hind et al., 2020). The high prevalence of degenerative disease such as osteoarthritis, in retired rugby players (Davies et al., 2017) appears to occur with premature onset (Scher, 1990). Furthermore, a study of professional rugby players reported a high number of mild to severe vertebral deformities by DXA vertebral fracture assessment (Hind et al., 2014). Athletes from contact sports including rugby, are reported to have higher lumbar spine bone density compared to athletes from other sports and to non-athlete populations (Elloumi et al., 2009; Hind et al., 2015). However, in these studies, it was not clear if vertebral exclusions were made for vertebral bodies with signs of degeneration or fracture.

The aims of this study were first, to investigate the presence of vertebral anomalies on lumbar spine bone density DXA scans in retired rugby players compared to an age-matched control group, and second, to determine the effect of vertebral deformity exclusion on the bone density outcome.

4.2 Methods

This research was performed as a cross sectional analysis of 138 male participants from the UK Rugby Health Project and was approved by the University Research Ethics Committee. The study design and recruitment protocol have been described previously (Hind et al., 2020). All participants provided signed informed consent prior to taking part.

4.2.1 Participants

Participants were from the United Kingdom, and were either retired rugby players, retired non-contact athletes or non-athletes. All participants in this study were male and aged between 24 and 78 years and retired from competitive sport. Rugby participants (n=87) were drawn from amateur and professional rugby union and rugby league codes. Participants in the non-rugby group (n=51) may have played sport at any competitive level and consisted of retired non-contact athletes (n=30), predominantly cricketers (n=24) or those that had never taken part in organised sport (n=21). Participants were grouped by age to enable the application of T-scores (≥50 y) or Z-scores (<50 y).

4.2.2 Procedures

Participants received DXA scans (Lunar iDXA^{TM}; GE Healthcare, WI; Encore software v 15.0) of the posterior-anterior lumbar spine (L1-L4) and dual femur (total hip and femoral neck) on one occasion. Participants were advised to abstain from intensive exercise, alcohol and caffeine in the 12 hours prior to scanning. For the lumbar spine scans, the legs were elevated with flexion at the hip and of the knees at 90^{o}, and with the lower legs resting on the iDXA positioning foam block (GE Healthcare, Madison, WI). This positioning enabled a widening of the intervertebral space so that individual vertebrae in the lumbar region were clearly visualised. Positioning for the dual femur BMD scan was assisted using the

GE-dual femur positioning device which allowed both legs to be abducted and inwardly rotated 15-25°. Quality assurance using the calibration block was made during the study period, and no drifts were observed. In-vivo precision (CV) for the DXA measurements in adults are 0.4% for lumbar spine BMD, 0.6% for total hip and 1.4% for femoral neck BMD (Hind et al., 2010).

The scans were performed by an experienced radiographer and an ISCD certified clinical densitometrist. T and Z-scores were evaluated both pre and post exclusion of vertebral anomalies (UK reference population). The presence of vertebral anomalies was determined in consensus between the two experienced densitometrists. T-score (ISCD, 2015) or Z-score discrepancies that were greater than 1 SD indicated vertebral exclusion. Vertebrae were also excluded from the evaluation if there was visible evidence of anatomical anomalies based on criteria reported by the ISCD (ISCD, 2015). Following removal, scans resulting in less than two evaluable vertebrae were deemed unreportable and were not assigned a T/Z-score. Each participant was assigned a 'hip T/Z-score', which was the lowest score from the femoral neck or total hip and a 'lowest T/Z-score', that is the lowest score of the lumbar spine or hip (ISCD, 2015).

4.2.3 Statistical analysis

Statistical analysis was performed using IBM SPSS® Statistics, Version 24. Data were assessed for normality visually and using the Kolmogorov-Smirnov test. The number of excluded vertebrae were compared using Mann Whitney U-test. The number of unreportable spines and instances of the lowest T/Z-score arising from the spine were also recorded and compared (Fishers Exact Test). The Related-Samples Sign test was used to compare the paired T/Z-scores from the spine and the lowest T/Z-score. The significance of the difference in the proportions of lowest T/Z-scores being generated by the spine (pre and post exclusion of vertebrae) was assessed with the McNemar's test. The significance of the difference in proportions of participants with ≥ 1 vertebral body excluded from BMD assessment was calculated using Fishers Exact Test. Significance was identified at <0.05.

4.3 Results

The study group descriptive results are presented in Table 4.1. In total, 122 vertebral exclusions were made, 79 in retired rugby players and 43 in the non-rugby group. Following exclusion of vertebrae, 12 scans were un-reportable (eight from the rugby ≥50 y group versus two from the non-rugby control ≥50 y group [p = 0.159] and two from the rugby <50 y group).

Table 4.1: Descriptive results

Table 4.1		N	Age (y)	Height (cm)	Weight (kg)	BMI (kg/m^2)	Hip T/Z-score
≥50 y	Rugby	31	57.4 ± 7.0	175.7 ± 7.4	94.5 ± 16.5	30.5 ± 4.6	-0.4 ± 1.3
	Non-Rugby	25	62.4 ± 6.6	174.4 ± 5.4	89.4 ± 16.9	29.3 ± 4.7	-0.7 ± 1.0
	p		0.006	0.471	0.264	0.347	-
<50 y	Rugby	56	39.9 ± 6.1	182.9 ± 6.9	102.7 ± 13.5	30.7 ± 4.0	0.4 ± 1.1
	Non-Rugby	26	37.4 ± 7.3	179.6 ± 5.9	84.2 ± 11.6	26.1 ± 3.1	-0.1 ± 1.0
	p		0.136	0.034	<0.001	<0.001	-

Table 4.2 displays the mean and standard deviation for the Lumbar spine T/Z-score (with and without vertebrae excluded) and the lowest T/Z-score from the lumbar spine or hip. The removal of anomalous vertebrae from the evaluation of the lumbar spine scans significantly reduced the lumbar spine T/Z-score across all four groups.

Table 4.2: Lumbar spine and Overall T/Z scores

Table 4.2		N	Lumbar spine T/Z-score			Overall Lowest T/Z score			Mean number of excluded vertebrae per person	Number of participants with ≥ 1 vertebrae excluded (%)
			Pre exclusion	Post exclusion	*p*	Pre exclusion	Post exclusion	*p*		
≥50 y	Rugby	31	0.7 ± 1.5	0.1 ± 1.3	0.006	-0.5 ± 1.2	-0.6 ± 1.1	0.031	1.5 ± 1.3	20 (65)
	Non-Rugby	25	0.5 ± 1.6	0.1 ± 1.5	<0.001	-0.8 ± 1.0	-0.9 ± 1.1	0.250	1.1 ± 1.2	14 (56)
	p								0.247	0.588
<50 y	Rugby	56	0.5 ± 1.4	0.3 ± 1.3	<0.001	0.0 ± 1.1	0.0 ± 1.1	<0.001	0.6 ± 0.9	22 (39)
	Non-Rugby	26	0.2 ± 1.3	-0.1 ± 1.0	0.004	-0.4 ± 1.0	-0.4 ± 0.9	0.125	0.6 ± 0.9	10 (39)
	p								0.873	1.000

There was a non-statistically significant increase in the incidence of lowest T-score arising from the spine (as opposed to the hip) following exclusion of anomalous vertebrae in each group, other than controls <50 years. The prevalence of the lowest T/Z-score arising from the spine significantly increased when vertebrae with anomalies were removed (47 versus 57, p=0.002). The exclusion of anomalous vertebrae identified an additional two cases of low bone density (T-score <1.0 or Z-score < -2.0).

4.4 Discussion

The aim of this study was to determine the prevalence of vertebral anomalies in retired rugby players and to investigate the effect of exclusion of these vertebrae on the lumbar spine T/Z-score and the resultant bone density outcome. In doing so, a high prevalence of vertebral anomalies were found across all study groups regardless of age, and the exclusion of these vertebrae, impacted on the resultant T/Z-score in 62 cases. The highest proportion of anomalies were found in the rugby group over ≥50 y, this group also had the largest number of unreportable lumbar spine scans of any group.

A main finding was that anomalous vertebrae were not limited to those in the over 50 years category. Previous studies demonstrating increased BMD with degenerative change in the spine have largely been limited to older populations (Yu et al., 1995; Jones et al., 1995; Liu et al., 1997; Pye et al., 2006). Our findings of reduced lumbar spine T-scores following exclusion of anomalies in those over 50 years, together with most (10 out of 12) unreportable spines found in those over 50 years, would be consistent with previous reports that the spine is more susceptible to elevated BMD due to osteoarthritis (Liu et al., 1997). The impact of this on the overall lowest T/Z-score was significant in the rugby groups of both age categories but not in the control groups. The exclusion of vertebrae with anomalies had the most significant effect on lumbar spine scan Z-score results in the <50 y retired rugby player group. A possible explanation could be the presence of early degenerative changes, which supports reports elsewhere of spine osteoarthritis in male rugby players, also aged under 50 years (Scher, 1990). The risk of osteoarthritis is increased with a previous history of injury (Wilder et al., 2002; Hind et al., 2020). Rugby players are exposed to a high risk of spinal injury, with a high incidence of cervical and lumbar spine injuries demonstrated in match play and training (Fuller et al., 2007).

The total number of lumbar spine scans producing the lowest T/Z-score was significantly increased following exclusion. However, there were no differences in the proportion of unreportable lumbar spine scans and the scan site producing the lowest T/Z-score between the rugby and control groups of both age categories. Furthermore, there were no differences in the number of vertebrae excluded between rugby and controls groups. The lumbar spine producing the lowest T/Z-score in significantly more cases when vertebrae had been excluded is possibly reflective of the spine BMD being subject to spurious increase from degenerative change particularly more so than the hip (Liu et al., 1997).

There are several considerations to make when interpreting the results of this study. First, our study focused on vertebral anomalies in former rugby players and therefore further studies would be

valuable to explore the prevalence of deformities in other populations. None-the-less, we also found vertebral anomalies in both the <50 y and ≥50 y control groups. It should also be considered that the evaluation and removal of vertebrae in DXA analysis can vary according to technician. In this study, the technician performing the scans was a registered radiographer and vertebral exclusions were verified by two densitometrists, including a certified clinical densitometrist.

4.5 Conclusion, limitations and recommendations for further work

In conclusion and based on these findings, vertebral exclusions should be considered for lumbar spine BMD scans performed in individuals under age 50 years, where there is evidence of vertebral anomalies. Currently, recommendations are restricted to over 50 years only and through the use of T-scores (ISCD, 2015). Considering the possibility of bone and joint degenerative changes at an earlier age in contact sports such as rugby, DXA scans in these athletes regardless of age, should be performed with careful evaluation to support the exclusion of vertebral anomalies where required, in order to avoid a falsely elevated BMD. Further research would be necessary to focus on athletes under 50 years from other sports to study the number of vertebral anomalies and potentially elevated BMD.

The next chapter aims to address the second aim and compare bone density at the hip and spine between the retired rugby groups and the retired non-contact athletes. Given the population being studied and the potential for BMD elevation in the presence of vertebral anomalies and resultant impact on BMD calculation, vertebrae affected by anomalies will be excluded from the assessment in the following study. Accurate assessment of bone density at the spine and avoidance of spurious increase in BMD will provide a reliable comparison of BMD without artificial elevation due to the potential presence of osteoarthritis, previous fracture and degenerative change.

Chapter 5.0 Study 2: Bone mineral density in retired rugby players and associations with previous and current bone-specific physical activity

Introduction

5.1 Bone health across the lifespan

The development of bone mass over the life course is a complex and multifactorial process. The timing of the physical activity over the lifespan can be optimised to promote adaptation. As ossification occurs throughout early life, longitudinal bone growth takes place. The optimum window for bone development is during puberty when there is rapid bone development and it is insufficient bone accrual at this stage that would results in suboptimal peak bone density (Weaver, 2002). Performing osteogenic activity such as weight bearing and resistance based exercise further compounds the effect of puberty, particularly when performed prior to the onset of puberty (Weaver, 2002). Evidence suggests that the period prior to puberty rather than post puberty is more successful in generating a response from the developing bones. This has been demonstrated in the arms of racquet sport players, where larger side to side differences in bone mineral content were seen in those starting prior to puberty rather than after (Kannus et al., 1995; Kontulainen et al., 2003). Forwood and Burr, (1993) concluded that exercise in childhood and adolescence is of greater importance than that performed in adulthood for achieving higher peak bone mass. Therefore, it is important to take advantage of this apparent window of opportunity, within which mechanical stimulation can promote bone building to achieve optimal peak bone mass, augmented by the already active modelling.

Later in life bone mass declines and in women the decline is more dramatic, at the onset of menopause compared to men, where the decline is more gradual, in line with declining levels of testosterone and oestrogen blood levels (Weaver and Peacock, 2019). Athletes from sports such as rugby, having higher bone density (Hind et al., 2015), is thought to offset the loss of bone density in later life. The main strategies to avoid low bone density in later life would be to achieve high bone

density at peak or attenuate loss in older age through continued osteogenic activity such as weight bearing and impact exercise (Watson et al., 2018).

There have been few studies to investigate the longevity of retired rugby players specifically. In those that have, there was no demonstrated difference in life expectancy (Rook, 1954; Beaglehole and Stewart, 1983). Participation in the game and the training involved does confer certain benefits on the players. In studies that have examined the characteristics of currently active rugby players, rugby players were found to have superior strength, bone density and lean mass when compared to controls (Elloumi et al., 2009; Hind et al., 2015). The longevity of these characteristics is unknown, and it is difficult to ascertain from the current literature if the benefits of such participation will remain following retirement. There is also a lack of data concerning the health of rugby players in retirement. A comprehensive study, again from questionnaire, explored various aspects of retired rugby players (n=259) health compared with general population cohort data (Davies et al., 2017). Standardised morbidity ratios were calculated for several physician diagnosed conditions, most notably, increased prevalence of osteoarthritis (4.00) and joint replacement (6.02) and of interest, osteoporosis (2.69) (Davies et al., 2017).

5.2 Bone health in athletes.

The association of muscle strength, size and skeletal mass has been established (Chalhoub et al., 2018) and given that retired rugby players have demonstrated superior lean mass to non-contact controls (Entwistle et al., 2022), it could be suggested that rugby players will have superior bone density in retirement. The mechanostat theory states the bone responds to muscle contractions via the tendon attachment, which initiates strain on the bone, which in turn promotes osteogenesis (Frost, 1987; Frost, 2003). Following cessation of sports related exercise, when the stimulus is absent and loading of bone is not beyond the threshold to initiate bone strains, the architecture and volume of bone is reduced up to a point where the strain is within an effective range (Frost, 1987; Giangregorio and

Blimkie, 2002). Therefore, when muscle mass is lost, as in the ageing individual, there is less strain generated by the contraction, resulting in a reduced action on the bone (Giangregorio and Blimkie, 2002). However, skeletal benefits appear to be maintained following retirement in athletes. Gymnasts have demonstrated superior bone strength (Eser et al., 2009). Furthermore, the site of bone mass development and retention appears to be sport specific. Hockey players have demonstrated superior BMD in the humerus, compared to controls following cessation, despite the loss of BMD at weight bearing sites (Nordström et al., 2005). Tennis players demonstrate a similar phenomenon in the forearm of the dominant arm (Kannus et al., 1994). While loading the bone via muscle strain is necessary, it appears less effective than impact loading, with athletes from swimming and rowing having lower BMD than athletes from rugby and soccer and fighting sports (Morel et al., 2001). Moreover, it is the upper limb that seems more dependent on strain as opposed to impact loading (Morel et al., 2001).

The aims of this study were firstly to determine the bone mineral density, measured at the lumbar spine and hips. Secondly, to measure the and establish pBPAQ and cBPAQ scores and compare between the groups. Thirdly, to assess for any relationships between BPAQ scores and bone density measurements.

5.3 Methods

This study was a cross sectional analysis of 118 male participants. The outcome variables of the study were bone mineral density (BMD) of measured at the hip and spine. Current and previous history of bone specific physical activity assessed using the bone specific physical activity questionnaire (BPAQ) (Weeks and Beck, 2008).

5.31 Participants

Participants were recruited using social media, television, radio and printed outlets together with word of mouth between September 2016 and December 2018. Further to completion of a General

Health Questionnaire (GHQ), 108 retired male athletes attended for clinical testing. Former elite (n=42) and amateur (n=46) rugby union and league players together with retired non-contact athletes ((n=30), predominantly cricket (n=24) players), received Dual energy X-ray absorptiometry (DXA) scans and completed a BPAQ. Participant demographic data is presented in Table 5.1.

Table 5.1		Age (y)*	Body mass (kg)	Height (m)	Body mass index (kg/m^2)*
Full group n=118		47.4 ±11.2	97.8 ±15.1	1.80 ±0.08	30.1 ±4.2
Elite Rugby, n=42		43.9 ±10.3	101.1 ±13.4	1.82 ±0.09	30.7 ±3.8
Amateur Rugby, n=46		48.0 ±10.5	98.9 ±16.6	1.79 ±0.07	30.7 ±4.5
Non-contact, n=30		51.3 ±12.5	91.3 ±13.4	1.79 ±0.07	28.6 ±3.8
Difference, *P*	Elite vs. Amateur	0.117	1.000	0.493	1.000
	Elite vs. Non-contact	0.019	0.019	0.303	0.050
	Amateur vs. Non-contact	1.000	0.088	1.000	0.074

* Data not normally distributed

5.32 Procedures

Participants were asked to arrive in a fasted and hydrated state, having abstained from alcohol, caffeine and intensive exercise for the 12 hours prior. Basic anthropometry was performed; height was assessed using a stadiometer (SECA Alpha, Birmingham, UK) and body mass was measured using electronic scales (SECA Alpha 770).

5.33 Bone mineral density

Participants received DXA scans (Lunar iDXA; GE Healthcare, WI; Encore software v 15.0) of the dual femur (total hip and femoral neck) and the posterior-anterior lumbar spine (L1-L4). The scanning method and technique has been described in detail elsewhere (Entwistle et al., 2021). Quality assurance using the calibration block was made during the study period, and no drifts were observed. In-vivo precision (CV) for the DXA measurements in adults are 0.4% for lumbar spine BMD, 0.6% for total hip and 1.4% for femoral neck BMD (Hind et al., 2010). As per recommendations from the ISCD and WHO (World Health Organisation, 2007b; ISCD, 2019), T-scores and Z-scores were derived. T-scores provide a comparison to a young adult female mean. A Z-score is calculated by comparing the

BMD to age and sex matched data, and adjusting for weight and ethnicity (ISCD, 2019). T-scores were reanalysed using female Caucasian reference database and updated software (Encore software v 18.0, GE Healthcare, WI). In the lumbar spine, T-score or Z-score discrepancies greater than 1 SD and/or a visual abnormality indicated the exclusion of the vertebrae from the bone density calculation (Entwistle et al., 2021).

5.34 Bone specific physical activity questionnaire (BPAQ)

Participants completed a paper based, BPAQ. This questionnaire details current and historical physical activity, documenting the number of years participating in a given activity. An online calculator is used to produce a BPAQ score for current (cBPAQ) and past (pBPAQ) physical activity (Weeks and Beck, 2008).

5.35 Statistical analyses

Statistical analyses were performed using IBM SPSS Statistics (Version 26; IBM Corp., Armonk, NY, USA). Normality was assessed visually and using Kolmogorov-Smirnov tests. Between group differences were assessed using a one way analysis of variance (ANOVA), where distributions were normal. When normality could not be assumed, non-parametric Kruskal-Wallis tests were performed. Post hoc tests were performed, and significance values adjusted by the Bonferroni correction for multiple tests. All data are posted as mean ± standard deviation (SD). A Pearson (where distribution was normal) or Spearman correlation coefficient (where distribution was not normal) was calculated to assess the correlation between several outcomes in the full group (n=118)

5.4 Results

Retired elite rugby players had the highest BMD at the hip neck, when compared to retired amateur rugby group ($p = 0.032$) and also the non-contact group ($p = 0.01$) with moderate effect size of

Cohens d = 0.55 and 0.77 respectively. There were no significant difference between the groups for total hip BMD. The elite group also had higher BMD at the spine compared to the non-contact group. However, when Z-score were compared at the hip and spine, that is adjusting for weight and age, no significant differences were seen between groups but there were moderate effects size seen (Cohens d = 0.43 and 0.35).

Table 5.2: BMD scores

Table 5.2	Elite Rugby	Amateur Rugby	Non-contact	*P*			Cohen's *d* (95% CI)		
				Elite vs. Amateur	Elite vs. Non-contact	Amateur vs. Non-contact	Elite vs. Amateur	Elite vs. Non-contact	Amateur vs. Non-contact
Hip Neck BMD	1.150 ±0.150	1.060 ±0.177	1.032 ±0.155	0.032	0.010	1.000	0.55 (0.12-0.97)	0.77 (0.28-1.26)	0.17 (-0.30-0.63)
Total Hip BMD*	1.203 ±0.147	1.136 ±0.182	1.102 ±0.145	0.166	0.065	1.000	0.41 (-0.02-0.83)	0.69 (0.20-1.18)	0.20 (-0.27-0.66)
T-score Hip	0.8 ±1.1	0.2 ±1.3	0.0 ±1.1	0.031	0.010	1.000	0.55 (0.12-0.97)	0.77 (0.28-1.26)	0.16 (-0.31-0.63)
Z-score Hip*	0.5 ±1.0	0.1 ±1.2	0.0 ±1.2	0.133					
Spine BMD*	1.352 ±0.140	1.247 ±0.148	1.275 ±0.150	0.236	0.016	1.000	0.72 (0.26-1.18)	0.53 (0.03-1.03)	-0.19 (-0.66 -0.29)
T-score Spine	1.5 ±1.2	0.6 ±1.2	0.8 ±1.3	0.007	0.139	1.000	0.72 (0.26-1.18)	0.50 (0.01-1.00)	-0.21 (-0.68-0.27)
Z-score Spine*	0.6 ±1.2	-0.1 ±1.2	0.4 ±1.3	0.157			0.55 (0.09-1.00)	0.16 (-0.33-0.65)	-0.38 (-0.85-0.10)
Final T-score	0.6 ±0.9	0.0 ±1.2	-0.2 ±1.1	0.024	0.015	1.000	0.57 (0.14-1.00)	0.75 (0.27-1.24)	0.10 (-0.36-0.56)
Final Z-score	0.1 ±0.9	-0.3 ±1.2	-0.2 ±1.1	0.142	0.547	1.000	0.43 (0.01-0.86)	0.35 (-0.13-0.82)	-0.10 (-0.56-0.36)
BPAQ Current*	4.8 ±6.4	2.4 ±3.6	1.8 ±2.2	0.007	0.018	1.000	0.47 (0.04-0.89)	0.59 (0.11-1.06)	0.19 (-0.27-0.65)
BPAQ Past*	129.4 ±44.2	125.6 ±42.7	95.0 ±49.8	1.000	0.001	0.005	0.09 (-0.33-0.51)	0.74 (0.25-1.22)	0.67 (0.20-1.14)
BPAQ Total*	67.1 ±22.7	64.0 ±21.9	48.4 ±25.3	1.000	0.001	0.004	0.14 (-0.28-0.56)	0.79 (0.30-1.27)	0.67 (0.20-1.14)
T-score >50 Hip	0.3 ±1.2	-0.3 ±1.2	-0.1 ±1.1	0.398	0.948	1.000	0.57 (-0.20-1.33)	0.43 (-0.38-1.22)	-0.18 (-0.83-0.47)
T-score >50 Spine	1.4 ±1.2	0.1 ±1.1	0.9 ±1.5	0.117	1.000	0.199	1.13 (0.13-2.11)	0.32 (-0.64-1.27)	-0.67 (-1.37-0.05)
Final T-score >50	0.2 ±0.9	-0.4 ±1.2	-0.3 ±1.1	0.403	0.915	1.000	0.59 (-0.19-1.35)	0.44 (-0.36-1.24)	-0.16 (-0.81-0.49)

* Data not normally distributed

The BPAQ score of current physical activity (cBPAQ) was higher in elite rugby group compared to the amateur (p = 0.007) and the non-contact (p = 0.018). Past BPAQ (pBPAQ) scores were higher in the rugby groups compared to the non-contact group.

In a separate sub-analysis of the participants over 50 years, where T-scores are clinically relevant in the diagnosis of osteopenia (T <-1.0) and osteoporosis (T ≤2.5), there were no differences

In incidence of osteoporosis or osteopenia. In the amateur over 50y group (n=21) there was 1 case of osteoporosis and 5 cases of osteopenia. The non-contact over 50y group (n=16) had no cases of osteoporosis and 3 cases of osteopenia.

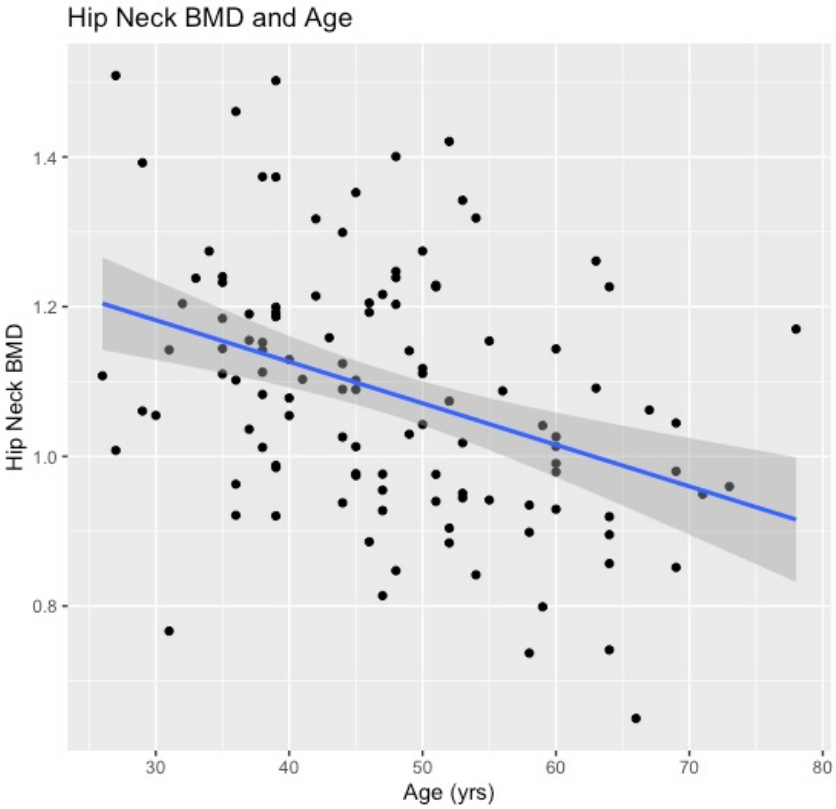


Figure 5.1: Hip Neck BMD and Age

A number of relationships were seen within the full group (n=118). As could be expected, significant correlations were demonstrated between age and BMD at the hip neck (r=-.369, p<0.001) and total hip (r=-.258, p=0.005) but not in the spine BMD. There was also a significant relationship between age and the cBPAQ score (r=-.278, p=0.002). The cBPAQ positively correlated with hip neck

BMD (r=.279, p=0.002), total hip BMD (r=.244, p=0.008), hip T-score (r=.281, p=0.002), hip Z-score (r=.227, p=0.014), spine BMD (r=.201, p=0.038), spine T-score (r=.202, p=0.037). There were no correlations between previous BPAQ and measures of bone density.

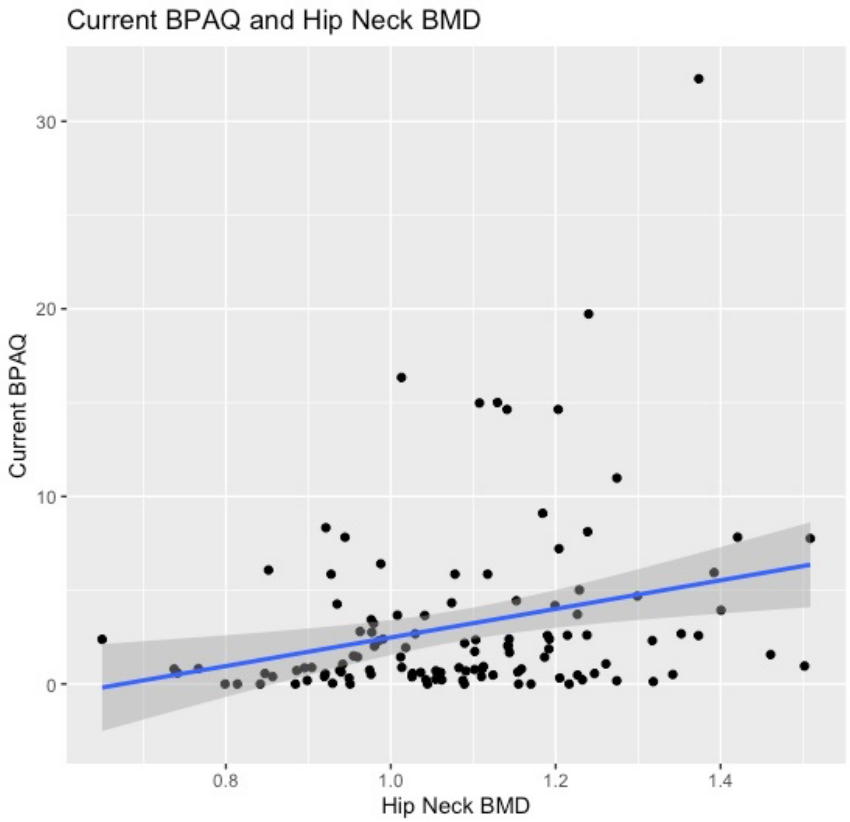


Figure 5.2: Current BPAQ and Hip Neck BMD

5.5 Discussion

This work addressed the aims of this study by documenting the bone density at the hip and spine of retired rugby players in comparison to non-contact athletes. Data on bone density in this particular group did not previously exist. Previous and current levels of bone-specific physical activity were measured and compared. The main findings were that while the elite rugby group had greater BMD hip neck compared to amateur and non-contact groups, there were no differences between the amateur rugby group and the non-contact group in all measures of bone density. Secondly, when BMD was adjusted for age, sex and weight, to calculate Z-scores, there were no differences between the

groups. cBPAQ scores were higher in the elite rugby group compared to the amateur and non-contact groups, as were pBPAQ scores in both rugby groups compared to non-contact athletes.

The negative relationship between age and BMD is in keeping with the expected bone mass decline in later life (Khosla and Riggs, 2005). Given that age is an important predictor of bone density and therefore fracture risk. However, after the adjustment for sex, age and weight to calculate a Z-score, there was still a relationship between cBPAQ and hip Z-score. When controlling for sex, weight and age there were no differences between the three groups. The mechanostat theory (Frost, 1987) of the bone dynamically altering in response to the level of strain experienced may provide an explanation. as elite rugby players may have achieved a high threshold, following a career of intense physical training with osteogenic stimulus available and therefore requiring a greater stimulus to achieve a response. The previously seen greater volumes of lean mass in retired rugby players but no superiority in muscle quality compared to non-contact athletes (see Chapter 6: Study 3) may also provide further understanding of the higher bone density in rugby players without higher Z-scores. The muscle providing strain on the bone, promoting an osteogenic reaction. However, the higher T-scores in the elite group will yield functional benefit due to the association between T-scores and fracture risk. While there is higher bone density seen in retirement, it should be noted that the average age of each group is under 50 years and would therefore not be of an age where osteoporosis could be diagnosed. The presence of higher BMD in the elite group that no longer exists in the over 50 years subgroups suggests that benefits from an active youth may have been mitigated in older age. However, where osteoporosis could be diagnosed (T score <2.5 in the over 50 years group) there was only one case of osteoporosis seen in the entire group, in the amateur rugby group. This is of note, as previous work by Davies et al (2017) presented a high prevalence of osteoporosis in retired rugby players compared to general population data. Significantly, this data was self-reported and therefore may not accurately reflect the true DXA-diagnosed prevalence.

5.6 Conclusion, limitations and recommendations for further work

The absence of any correlation between pBPAQ and measures of bone density suggests the past history of physical activity is less important than current physical activity. Therefore, it would appear that continued participation in of weightbearing physical activity is important to maintain bone mass and attenuate the loss experienced due to ageing. Although, the limitations of the BPAQ should be considered here, given that there is no differentiation between the elite and amateur rugby in the weighting assigned to rugby training. It could be assumed that professional sport would be at a higher, more intense level and would have an impact on the response. This may account for the lack of relationship between past physical activity and bone density. The impact of genetics should not be ignored and those predisposed to superior mass and strength may be attracted to sports such as rugby. The higher cBPAQ scores in elite rugby group is noteworthy in demonstrating an ability to continue to be physically active in retirement. The high injury toll on rugby players and the impact in retirement (Hind et al., 2020) may prevent long term practice of osteogenic activity. However, elite rugby players participated in more bone specific activity than the other groups. It should be noted that this study has several limitations. Firstly, the cross-sectional study design does not allow any cause and effect inferences. The mean age of the full group is 47 years and osteoporosis in men cannot be diagnosed under 50 years.

The close relationship between the muscular contraction (and therefore muscle mass and strength) alongside bone development as demonstrated by the similar trajectory of growth in young adulthood and subsequent loss in older age, must be considered. A comparison of lean mass and strength between these groups could identify the lean mass profile of retired rugby athlete and the relationship with strength, thus allowing a measure of muscle quality. As the decline in bone density can result in osteopenia and osteoporosis, so the loss of lean tissue can result in sarcopenia. Osteosarcopenia may be outcome if both bone and muscle mass were to decline. The following study will address this.

Chapter 6.0 Study 3: Lean mass, muscle strength and muscle quality in retired rugby players

This study has been published in a peer reviewed journal:

Entwistle, I., Francis, P., Lees, M., Hume, P. and Hind, K. 2022. Lean Mass, Muscle Strength, and Muscle Quality in Retired Rugby Players: The UK Rugby Health Project. *International Journal of Sports Medicine*. **43**(11), pp.958–963.

6.1 Introduction

Physical activity and exercise promote the accumulation of lean mass which is important for reducing the risk of sarcopenia (Peterson et al., 2011). Competitive athletes engage in a high volume of exercise throughout their sporting careers, and this is reflected in their unique physique and body composition (Olds, 2001; Reale et al., 2020). Rugby players have demonstrated greater body mass and lean mass (Hind et al., 2015) as well as muscle strength (Elloumi et al., 2009)compared to non-athletes of the same age. The assumed performance advantage gained from greater stature and mass means that this is a differentiating characteristic of rugby players at the elite level (Johnston et al., 2014) as well as higher lean and lower fat mass compartments (Johnston et al., 2014; Till et al., 2015). The contact nature of rugby places importance on the ability to generate momentum, thus making speed and mass distinguishing characteristics of successful players (Baker and Newton, 2008).

Lean mass and strength accumulate from birth until adulthood but from around midlife, there is a progressive loss of lean mass (Dodds et al., 2014; Cruz-Jentoft et al., 2019). Sarcopenia is the term originally proposed to describe this age-associated loss of muscle mass (Rosenberg, 1989), developing later to encompass the loss of strength (Morley et al., 2001). The most recent working definition has since incorporated additional measures, including muscle quality and physical performance (Cruz-Jentoft et al., 2019). Importantly, the loss of strength with age is more pronounced and occurs earlier

than the loss of muscle mass (Goodpaster et al., 2006). Sarcopenia is an important area of focus given its association with several undesirable outcomes such as an increased risk of falls, prolonged hospital stays, morbidity and mortality (Mayhew et al., 2019). In particular, muscle strength rather than mass is associated with mortality in later life (Newman et al., 2006). In a study by Ruiz et al. (2008), older males grouped in the lowest third for strength had a mortality rate 50% greater than those in the upper third (Ruiz et al., 2008; McLeod et al., 2016). Therefore, the maintenance of lean mass and muscle strength with age are important goals for supporting longevity and functional capacity.

The variation in muscle mass and strength between individuals is a multifactorial issue explained by both the attainment of peak mass and strength in young adulthood and the rate of muscle loss thereafter (Sayer et al., 2008). While there is loss of muscle mass and strength over time, the loss of mass alone does not fully explain the loss of strength, given that where mass has been maintained, strength has still declined (Goodpaster et al., 2006).

Muscle quality, as measured by the amount of force per unit mass (kg/kg) (Lynch et al., 1999), is a complementary tool for musculoskeletal health and sarcopenia assessment (Barbat-Artigas et al., 2012; Lees et al., 2019). Given that strength deteriorates more rapidly that lean mass (Lauretani et al., 2003), muscle quality has been shown to decrease with advancing age (Moore et al., 2014). Although cut-points for muscle quality of <5.76 kg/kg for men have been developed (Cooper et al., 2014), robust and specific thresholds for older populations remain to be determined.

While it is established that athletes from sports such as rugby have superior lean mass and strength during their playing careers, little is known about these qualities post-retirement. Therefore, the purpose of this study was to investigate the impact of previous participation in rugby codes by comparing lean mass, muscle strength and muscle quality in former competitive rugby players and non-rugby players.

6.2 Methods

This study comprised a cross-sectional analysis of 139 participants from the UK Rugby Health Project. With a multidisciplinary research focus, the UK Rugby Health Project was initiated in 2016 and has been described elsewhere (Hind et al., 2020). The primary outcome variables for this current study were lean mass, muscle strength and quality. The project was approved by the University Research Ethics Committees and informed consent was obtained from all participants.

6.2.1 Participants

Participants were recruited using past player/athlete associations, printed and televised media outlets, word of mouth and social media between September 2016 and December 2018. Upon completion of a general health questionnaire (GHQ), participants were invited to attend an appointment at the University for clinical testing, including body composition and strength assessment. A total of 254 participants completed the GHQ and 108 retired athletes attended for clinical testing. Retired rugby players (n=88) had previously competed in rugby union, rugby sevens or rugby league for at least 3 years and included 42 former elite and 46 amateur rugby players. The non-contact group (n=30) consisted of retired non-contact athletes, predominantly (n = 24) retired cricket players. Participant characteristics are reported in Table 6.1 (mean ± standard deviation).

Table 6.1		**Age (y)***	**Body mass (kg)**	**Height (m)**	**Body mass index (kg/m^2)***
Elite Rugby, n=42		43.9 ±10.3	101.1 ±13.4	1.82 ±0.09	30.7 ±3.8
Amateur Rugby, n=46		48.0 ±10.5	98.9 ±16.6	1.79 ±0.07	30.7 ±4.5
Non-contact, n=30		51.3 ±12.5	91.3 ±13.4	1.79 ±0.07	28.6 ±3.8
Difference, *P*	**Elite vs. Amateur**	0.117	1.000	0.493	1.000
	Elite vs. Non-contact	0.019	0.018	0.303	0.050
	Amateur vs. Non-contact	1.000	0.088	1.000	0.074
Cohen's *d*	**Elite vs. Amateur**	0.39	0.15	0.29	-
	Elite vs. Non-contact	0.66	0.73	0.38	0.55
	Amateur vs. Non-contact	0.29	0.49	0.11	0.49

* Data not normally distributed

6.2.2 Procedures

Participants were asked to arrive at the laboratory in a fasted and hydrated state and abstain from alcohol, intensive exercise, and caffeine for the previous 12 hours. Following a brief schedule of events, participants completed an adapted screening questionnaire and received basic anthropometry. Wearing only light, loose clothing, height was assessed using a stadiometer (SECA Alpha, Birmingham, UK) and body weight was measured using calibrated electronic scales (SECA Alpha 770, Birmingham, UK). Blood pressure was measured as part of the screening procedure. Participants were excluded from strength testing if they had a history of cardiovascular disease, high blood pressure (>140/90 mmHg) or a relevant injury to the upper limb (n = 35). Following exclusion of participants that failed the screening process there were 31 elite rugby, 28 amateur rugby and 24 non-contact group participants for strength and muscle quality measures.

6.2.2.1 Body composition

Each participant received one total-body dual-energy X-ray absorptiometry (DXA) scan (GE Lunar iDXA, GE Healthcare, Madison, WI). Daily quality assurance tests were performed on testing days and all scans were performed by a registered radiographer or certified densitometrist. Participants were scanned in the supine position, with arms placed close to sides, hands mid-prone and legs immobilised in position using the provided ankle strap. In 21 cases where the arm of the participant did not fit within the scan area, the contralateral arm data was replicated. Three-component (bone mineral content, lean tissue mass and fat mass) total body composition data were acquired. Regional composition data were also derived, allowing for the calculation of appendicular lean mass (ALM; total lean mass of the arms and legs) and muscle quality as the amount of force per unit mass (Cooper et al., 2014). *In vivo* precision for total body composition in adults for the DXA used in the current study is 0.51% for total body lean tissue mass, 0.82% for total body fat mass and 0.86% for percentage total body fat (Hind et al., 2011).

6.2.2.2 Strength assessment

Handgrip strength was assessed in 59 rugby and 24 non-rugby participants using a Takei T.K.K. 5401 handgrip dynamometer (Takei Scientific Instruments Co., Ltd, Niigata, Japan). Both hands were tested in an upright standing position, with the arm positioned straight by the side, with the shoulder slightly abducted and feet approximately shoulder width apart (NCHS, 2011). The participant was asked to breathe in and squeeze the dynamometer as they breathed out. Each hand was tested three times, alternately, ensuring a one-minute interval between each attempt on the same hand. The maximum effort recorded from the six attempts is presented. Muscle quality was calculated as grip strength relative to upper body ALM/arm lean mass (kg/kg).

6.2.2.3 Sarcopenia assessment

In the absence of a single consensus, there are several measures recommended to classify an individual as sarcopenic, with several recommended cut-points (Mayhew et al., 2019). The European Working Group on Sarcopenia in Older People (EWGSOP) (Cruz-Jentoft et al., 2019)recommend several cut-points for the identification of sarcopenia, based on European populations. These include, for men, grip strength of <27 kg (Dodds et al., 2014), ALM of <20 kg (Studenski et al., 2014) and appendicular lean mass index (ALMI; ALM/height^2) of <7.0 kg/m^2 (Gould et al., 2014). Alternative, higher cut-points have been employed in other studies, such as <37.2 kg for grip strength (Cooper et al., 2014) and ALMI of <7.23 kg/m^2 (Fielding et al., 2012). We assessed the presence of sarcopenia in each group using these higher cut-points to compare prevalence among each group.

6.2.3 Statistical analyses

Statistical analyses were performed with IBM SPSS Statistics (Version 26, IBM Corp., Armonk, NY). Normality was assessed visually and using Kolmogorov-Smirnov tests. Between-group differences, where distributions were normal, were assessed using one way analysis of variance (ANOVA). For distributions where normality could not be assumed, non-parametric Kruskal-Wallis tests were

performed. Post-hoc tests were performed, and significance values adjusted by the Bonferroni correction for multiple tests. All data are reported as mean ±standard deviation (SD). Fisher's Exact Test was used to determine the significance of differences between proportions of those categorised as sarcopenic. Cohen's d effect size calculations were performed using the R programming language (Version 4.0.3) and classified using the following thresholds: <0.2 = trivial; 0.2 – 0.5 = small; 0.5 – 0.8 = moderate, and >0.8 = large (Cohen, 1988).

6.3 Results

The body composition and strength outcomes for the retired elite and amateur rugby and non-contact groups, are presented in Table 6.2.

Table 6.2	Elite Rugby	Amateur Rugby	Non-contact	*P*			Cohen's *d*		
				Elite vs. Amateur	Elite vs. Non-contact	Amateur vs. Non-contact	Elite vs. Amateur	Elite vs. Non-contact	Amateur vs. Non-contact
Total lean mass (kg)	71.6 ±9.5	65.2 ±7.6	60.0 ±7.8	0.002	<0.001	0.027	0.74	1.31	0.68
Appendicular lean mass (kg)	34.6 ±5.1	30.9 ±4.1	28.3 ±4.2	<0.001	<0.001	0.051	0.81	1.33	0.62
Arms lean mass (kg)	9.7 ±1.7	8.4 ±1.1	7.5 ±1.3	<0.001	<0.001	0.031	1.00	1.47	0.72
Legs lean mass (kg)	24.9 ±3.6	22.5 ±3.2	20.8 ±3.0	0.003	<0.001	0.086	0.70	1.22	0.55
Lean mass index (kg/m^2)	21.6 ±2.0	20.2 ±1.7	18.7 ±1.7	0.001	<0.001	0.002	0.76	1.53	0.86
Appendicular lean mass index (kg/m^2)	10.5 ±1.1	9.6 ±0.9	8.8 ±1.0	<0.001	<0.001	0.009	0.87	1.51	0.77
Body fat percentage (%)	25.8 ±6.3	30.6 ±6.6	31.1 ±7.0	0.003	0.003	1.000	0.74	0.80	0.08
Muscle quality (kg/kg)	5.57 ±0.86	5.92 ±0.86	5.93 ±0.96	0.428	0.438	1.000	0.40	0.39	0.01
Grip strength (kg)	54.3 ±8.9	48.8 ±8.0	45.5 ±7.3	0.038	<0.001	0.435	0.64	1.07	0.43

Table 6.2. Body composition, muscle strength and muscle quality in retired rugby code and non-rugby participants, presented as mean ±standard deviation.

6.3.1 Body composition

The elite rugby group had significantly higher appendicular lean mass compared to amateur rugby and non-contact groups ($p < 0.001$, Cohen's $d > 0.8$).

6.3.2 Strength and muscle quality

Grip strength was greater in the retired elite rugby group compared to the non-contact group ($p < 0.001$, constituting a large effect) and the community rugby group ($p = 0.038$). There were no significant differences in muscle quality between the amateur rugby group and the non-contact group (small effect). No significant differences in muscle quality were seen across the groups. However, muscle quality was lower in the elite rugby group compared to the amateur rugby and non-rugby groups (small effect).

6.3.3 Prevalence of sarcopenia

Sarcopenia criteria	**Elite Rugby**	**Amateur Rugby**	**Non-contact**	***P***
Grip strength <27 kg [9]	0%	0%	0%	-
Appendicular lean mass (ALM) <20kg [28]	0%	0%	3.3%	0.244
ALM/height^2 < 7.0 kg/m^2 (Gould et al., 2014)	0%	0%	0%	-
Grip strength <37.2 kg (Cooper et al., 2014)	3.2%	3.6%	12.5%	0.434
ALM/height^2 < 7.23 kg/m^2 [38]	0%	0%	0%	-
Muscle Quality <5.76 kg/kg [24]	64.5%	46.4%	45.8%	0.282

Table 6.3. Sarcopenia prevalence according to the designated assessment tool in retired rugby code and non-rugby participants.

No significant differences were seen in sarcopenia prevalence among the three groups. In the non-contact group, 12.5% (n = 3) were below the 37.2 kg threshold for grip strength (Cooper et al., 2014) compared to 3.2% (n = 1) in the elite rugby group and 3.6% (n = 1) in the amateur rugby group (p = 0.434). Prevalence was highest using the <5.76 kg/kg threshold for upper body muscle quality [24]. In the elite rugby group 64.5% (n = 20) were below the cut off compared to 46.4% (n = 13) and 45.8% (n = 11) in the amateur rugby and non-contact groups respectively. Sarcopenia prevalence is presented in Table 6.3.

6.4 Discussion

This study investigated lean mass, strength and muscle quality in retired elite male rugby players compared to an age- and sex-matched amateur rugby group and a sex-matched non-contact group. In doing so, the significant findings were firstly, that while retired elite rugby players had greater overall lean mass and strength, compared to both groups there were no differences in muscle quality between groups. Secondly, there were no differences in strength between the amateur rugby group and non-contact group. Thirdly, the prevalence of sarcopenia was low in all three groups when using most sarcopenia cut-points, except for muscle quality [24]. A further observation was of a lower body fat percentage in elite compared to the amateur rugby group and non-rugby group. There were no differences in body fat percentage between the amateur rugby and non-rugby groups.

The elite rugby group had greater lean mass and strength compared to retired amateur rugby players and non-contact athletes. Higher body mass has been observed in currently active elite rugby union and rugby league players compared with non-athletes (Hind et al., 2015) and this was present in retirement with a significantly higher BMI in former elite rugby players (p = 0.050, moderate effect), corroborating the findings of Hind and colleagues (Hind et al., 2015). Conversely, former elite college athletes performed worse than controls in several measures, including body composition and

strength, with the authors suggesting a limited ability to perform physically due to previous injury as an explanation for such outcomes (Simon and Docherty, 2017). It might be plausible that the high injury toll in rugby and two-fold increased prevalence of osteoarthritis in retired players (Hind et al., 2020) may limit the ability of a former player to remain active. However, in the current study, higher volume of lean mass and significantly higher grip strength were seen in former elite rugby players.

When comparing the values to those from currently competitive professional rugby players in the UK, the retired elite rugby code players had greater body mass (101.1 vs. 96.5 kg) but less lean mass (71.6 vs. 74.6 kg), arm lean mass (9.7 vs. 10.0 kg) and leg lean mass (24.9 vs. 25.3 kg) (Till et al., 2016). Notwithstanding, the volume of lean mass, the lean mass index and the ALMI of the retired rugby players were all higher than mean values for players aged 20-29 years in published reference data (Imboden et al., 2017). Moreover, the elite rugby group placed in the 80th percentile of the 40-49 yr age group for measures of lean mass and ALMI (Imboden et al., 2017). Taken together, these findings suggest that retired rugby players may experience lasting benefits for body composition long after their careers have concluded.

No significant difference in muscle quality between the rugby and non-rugby groups was seen in the current study. However, it is noteworthy that the elite rugby group had the lowest recorded muscle quality compared to both amateur rugby and non-contact athletes (small effect). Previous studies have demonstrated an inverse relationship between muscle mass and muscle quality (Barbat-Artigas et al., 2013) with higher volume of upper limb lean mass associated with lower muscle quality and lower muscle quality associated with functional impairments (Barbat-Artigas et al., 2013). This is consistent with the findings from the current study, given that retired elite rugby players have higher levels of lean mass and strength but lower muscle quality. It may be possible that there has been a loss of strength since peak, at a greater rate than loss of mass. However, the cross-sectional design of the current study only allows for speculation, although strength is known to decrease at a greater rate than mass (Barbat-Artigas et al., 2013). Given that the rugby group demonstrated higher volume of

lean mass and ALMI, this would yield functional benefit of higher volume of muscle with similar quality.

No differences were found in body fat percentage between the retired amateur rugby and non-contact group, although lower volume of body fat were seen in the elite rugby group compared to both groups ($p = 0.003$, moderate effect). Higher BMI in rugby players has previously been seen with comparable volume of body fat (Hind et al., 2015) and this should also be considered in retirement given risk for cardiometabolic disease (McHugh et al., 2020). Furthermore, in other studies of former endurance athletes, lower body fat percentage was found compared to controls (Laine et al., 2016a). Both rugby groups had higher BMI at 30.7 kg/m^2, largely as a result of higher volume of lean mass. It should be noted that BMI is employed as a measure of cardiometabolic health but is somewhat limited by an inability to differentiate lean mass from fat mass. The rugby groups would be classified as obese on this basis, a potentially misleading conclusion given the benefits of higher volume of lean mass with advancing age.

The elite rugby group were heavier and had greater lean mass and grip strength compared to non-contact controls, with no significant differences in muscle quality. Furthermore, the prevalence of sarcopenia was not significantly different across the groups. The low prevalence of sarcopenia in the rugby and non-contact athletes suggests there may be a beneficial effect of rugby participation in youth and young adulthood for muscle health in ageing. However, all groups had much higher prevalence of sarcopenia when muscle quality was the chosen measure (64.5% elite; 46.4% amateur; 45.8% non-contact). Given that volume of lean mass would have likely been superior during young adulthood, a dramatic loss of mass seems unlikely. However, the performance advantage gained from possessing higher body mass makes those with a genetic advantage in this area likely to be identified as such, and thus attain success in the sport. The significance and practical impact of lower muscle quality (small effect) in the elite rugby group compared to both the community and non-rugby groups, given the superior strength of the rugby group, warrants additional exploration. Moreover, further

work is needed to determine the relevance of muscle quality as a measure of sarcopenia in the absence of low grip strength.

There were several considerations to make when interpreting the results of this study. First, the study design was cross-sectional and therefore cause and effect inferences could not be made. Secondly, participants self-selected and the impact of the potential genetic predisposition to superior muscle mass and strength of those who participated in rugby limited the conclusions to be drawn from such a study. Thirdly, the median age of this cohort was 47 years and relatively early in the ageing process, and thus did not allow for inferences to made for the older ageing population. Moreover, sarcopenia cut-points (Cooper et al., 2014) were developed in older adults, over 60 years. Fourthly, the lack of exercise and diet data restricted the investigation of the impact of such factors. Therefore, future studies to focus on exercise and diet habits in an older retired population would be valuable.

In conclusion, the retired elite and community rugby players had higher, lean mass and superior grip strength compared to their non-rugby counterparts. However, no significant difference was seen in muscle quality. The aim of efforts to reduce sarcopenia, focus on preventing the loss of lean tissue from peak levels attained in adulthood. The potential benefits of previous rugby participation on muscle mass and strength appear to somewhat be retained and this is relevant given the positive functional implications for activities of daily living. Future research with a focus on the longitudinal assessment of muscle quality, diet and exercise in rugby players post retirement, would be valuable.

A further assessment to be made with implications for fracture risk, given that mass appears to be retained, is falls risk. The risk of fracture from falling, is compounded by low bone density and sarcopenia. Therefore, a strategy to prevent fracture must assess fall risk. The next chapter will explore the balance performance of elite and amateur rugby groups compared to non-contact athletes.

Chapter 7.0. Study 4: Balance and dynamic posturography in retired rugby players

7.1 Introduction.

Falling is a risk in the elderly and is known to increase with advancing age (Peel, 2011). The rate of fatal falls increases exponentially with age (World Health Organisation, 2007a). Falls are the most common reason for emergency hospital attendance in older people (OHID: Office for Health Improvement and Disparities, 2022). Falls can occur due to a number of factors including muscle weakness, poor balance and visual impairment (OHID: Office for Health Improvement and Disparities, 2022). A fragility fracture can occur when a low energy impact has taken place where there are other risk factors such as osteoporosis and has an annual cost of £4.4 billion, of which £2 billion is accounted for by hip fractures (OHID: Office for Health Improvement and Disparities, 2022).

Balance is maintained if the centre of gravity (COG) is preserved vertically over body's the base of support (Nashner, 1997). Gravitational force acts downward on the body and if there is displacement of the COG (sway) from its position above the base of support, angular acceleration is initiated with the larger the displacement angle resulting in larger acceleration (McCollum and Leen, 1989). When an individual is working to maintain the COG above the base of support, this cannot be done without movement. There is a sway from side to side and forwards and backwards (Nashner, 1997). The limit of stability (LOS) is the theoretical maximum sway possible without falling (Koozekanani et al., 1980; Nashner, 1997; Natus Medical Incorporated, 2013). When the limits of sway exceed the limits of stability the individual loses balance (Nashner, 1997). There are two issues of note: the limits of sway will be as large as the LOS when the COG is preserved over the base of support. However, if the COG is offset then it is closer to the LOS in one direction and balance will be disturbed

as it will take less displacement to exceed the LOS than a correctly aligned COG (Nashner, 1997). The second issue being that the sway frequency impacts the actual LOS (Nashner et al., 1989) with higher sway frequencies the LOS is subsequently reduced (Nashner, 1997). Increased sway velocity means poorer balance (Wang et al., 2018). This complex task of balance maintenance is achieved through a co-ordinated effort of processing information from the visual, vestibular and somatic senses (Nashner and McCollum, 1985). The force plate tracks the movement of the centre of pressure and from this, the centre of gravity and sway angle are calculated, subsequently producing a sway velocity (Natus Medical Incorporated, 2014a).

Rugby is a demanding collision sport. The nature of the game demands that players are required to have excellent physical fitness and agility. Moreover, with agility further improving with age and experience suggests rugby training is beneficial to balance performance (Chow et al., 2016). However, rugby has a high cumulative injury load with continued impact in retirement (Hind et al., 2020). Furthermore, concussion was the most frequently reported injury, reported in over 75% of rugby athletes (Hind et al., 2020). Additionally, osteoarthritis was reported twice as much in retired rugby players compared with retired non-contact athletes (Hind et al., 2020).

Given the immediate effects of concussion on balance performance are well documented (Broglio and Puetz, 2008; Parker et al., 2008b), it is of note that concussion was the most frequently reported injury in retired players (Hind et al., 2020), and it has been the most common injury in the English Premiership in terms of incidence and associated days absence since between 2015-2020 (England Professional Rugby Injury Surveillance Project Steering Group, 2021). The potential long term effect on balance and falling has not been studied to date. Furthermore, a concussed player is more likely to sustain further musculoskeletal injury (Cross et al., 2016). However, even in the absence of a confirmed concussion, contact sports athletes performed worse in tests of memory than control subjects (Killam et al., 2005), the authors speculating as a result of sub concussive impacts.

The number of musculoskeletal injuries in rugby is also high (Hind et al., 2020; Hume et al., 2022) with further impact in retirement. Previous injury is a substantial risk factor for the development of degenerative change in the joint (Roos, 2005). Davies et al demonstrated high rates of osteoarthritis in former rugby players (Davies et al., 2017; Davies et al., 2017). There are deficits in balance and increased risk of falling in those with knee osteoarthritis (Kim et al., 2011; Khalaj et al., 2014). Poor balance is associated with difficulty in activities of daily living, this in turn reduces physical activity and confidence, further reducing balance performance. Lean mass reduces with age, together with strength and reaction time. Physiological changes in the muscle spindle bring about poorer proprioception and a loss of sensitivity in addition to reduced vibration sense. Visual acuity is also reduced (Henry and Baudry, 2019).

Balance is controlled by complex interaction of the vestibular, proprioceptive and visual systems. The sensorimotor function of maintaining postural control will depend on the setting. However, the primary goals are to maintain the centre of mass over the base of support, ensuring the trunk remains upright and coordinating the effective movement of the limbs (Horak, 1997). The body must respond to postural challenges in either a reactive or anticipatory control of posture (MacKinnon, 2018). The ability and efficiency to carry out these tasks declines with age, with the potential to ultimately impair balance in such a way as to result in falls, fracture and morbidity(Henry and Baudry, 2019).

There are a variety of musculoskeletal and neurological conditions that impair balance and failure in any of these interactions will reduce balance performance with the potential to result in a fall. Athletes have demonstrated superior performance in balance testing compared to non-athlete controls, comparable to that of younger subjects (Räty et al., 2002). Furthermore, there was no significant differences between athletes from different sports (Räty et al., 2002).

The aim of this study is to assess the balance performance of retired rugby players and non-contact athletes and determine any relationships between parameters of balance and previous injury.

7.2 Methods

The study was a cross-sectional analysis of 112 participants from the UK Rugby health project and has previously been described (Hind et al., 2020). The focus of the study was to assess the balance performance using the Neurocom VSR Sport testing platform (Natus Medical Incorporated, San Carlos, CA). The project was approved by the University Research Ethics Committees. Participants also received whole body DXA scans to determine body composition and underwent hand grip strength assessment.

7.2.1 Participants

Participants were recruited using past player networks, social media and television/radio outlets. In the first part, participants completed a General Health Questionnaire (GHQ), providing details of playing and injury history. Upon completion of the GHQ, participants were invited to attend the University for clinical testing. Retired professional (n=40), amateur (n=46) rugby players and non-contact athletes (n=26, predominantly cricket players) received DXA scans, grip strength testing and balance assessment. Table 7.1 presents participant characteristics.

Table 7.1		Age (y)*	Body mass (kg)	Height (m)	Body mass index (kg/m^2)*
Full group n=112		47.0 ±11.2	97.6 ±15.2	1.80 ±0.08	30.1 ±4.2
Elite Rugby, n=40		44.0 ±10.5	100.6 ±13.3	1.81 ±0.09	30.7 ±3.8
Amateur Rugby, n=46		48.0 ±10.5	98.9 ±16.6	1.79 ±0.07	30.7 ±4.5
Non-contact, n=26		50.0 ±12.6	90.8 ±13.4	1.79 ±0.07	28.3 ±3.7
Difference, *P*	**Elite vs. Amateur**	0.058	1.000	0.864	1.000
	Elite vs. Non-contact		0.028	0.678	0.038
	Amateur vs. Non-contact		0.080	1.000	0.057
Cohen's *d*	**Elite vs. Amateur**	0.38	0.11	0.23	-
	Elite vs. Non-contact	0.53	0.74	0.29	0.62
	Amateur vs. Non-contact	0.18	0.53	0.08	0.55

* Data not normally distributed

7.2.2 Procedures

Participants were asked to abstain from alcohol and caffeine in the previous 12 hours and to arrive at the laboratory in a fasted and hydrated state. On arrival, a screening questionnaire was administered, together with basic anthropometry. Height was measures using a stadiometer (SECA Alpha, Birmingham, UK) and body weight was assessed using electronic scales (SECA Alpha 770). A total body dual-energy X-ray absorptiometry (DXA) scan (GE Lunar iDXA; GE Healthcare, Madison, WI, USA) was received by each participant. Handgrip strength assessment was performed by 58 rugby players and 20 non-rugby players using a Takei T.K.K. 5401 handgrip dynamometer (Takei Scientific Instruments Co., Ltd, Niigata, Japan). Muscle quality was calculated as grip strength relative to upper body appendicular lean mass (kg/kg). These procedures have been described, in detail elsewhere (Entwistle et al., 2022).

7.2.3 Balance assessment

Static and dynamic balance were assessed with the Neurocom VSR sport force plate and Balance Manger software (Natus Medical Incorporated, San Carlos, CA). The VSR sport offers several assessment protocols:

The Stability Evaluation Test (SET), for functional assessment; the Modified Clinical Test of Sensory Interaction on Balance (mCTSIB) for sensory assessment; the Limits of Stability (LOS), for voluntary motor assessment (Natus Medical Incorporated, 2014b). Following on-screen instructions re foot placement, the participants underwent the three assessment protocols, consecutively with bare feet to minimise slipping on the metal surface.

Stability Evaluation Test (SET)

The SET is an objective version of the established Balance Error Scoring System (BESS). The BESS is used in the management of concussion and is a low cost option for assessing postural deficits in the athletic population (Guskiewicz, 2011). The SET provided by Neurocom is an objective version of the

BESS, adopting the same positions for the testing protocol as those executed in the BESS. However, unlike the BESS, sway velocity is recorded in place of the subjective scoring system. The SET has demonstrated good reliability (Williams et al., 2017). The participant performs six trials: the double stance, single leg and tandem stance on a firm surface for 20 seconds each. The same three trials are completed on a foam surface. All trials are performed with eyes closed. Image: (Guskiewicz, 2011). Sway velocity (degree per second) is presented for each trial. The participants failed to record measures for every trial.

Modified Clinical Test of Sensory Interaction on Balance (mCTSIB)

The mCTSIB has been designed as a simplified version of the sensory organisation test (SOT) (Natus Medical Incorporated, 2014a). The SOT being the gold standard of balance assessment.

The participant is positioned on the force plate on both feet a given distance apart (as per on-screen instruction), depending on height, with a wider stance for taller people. The integration of the vestibular, vision and somatosensory systems is required for correct balance control (Cimino et al., 2018). In static tests the COG sway reflects the competence to organise the input from the sensory systems in order to preserve balance control (Cimino et al., 2018). The mCTSIB has demonstrated reliability and correlated with other clinical balance measure (Cimino et al., 2018). The participant underwent 3 trials each under 4 different conditions: eyes open on the firm surface, eyes closed and firm surface, eyes open on the foam surface and eyes closed on the foam surface. Each of the 12 trials lasted 10 seconds. Results are given as a mean COG sway velocity for each condition and a composite sway velocity.

Limits of Stability (LOS)

Participants stand in a given position on the force plate and are instructed to move in 8 given directions. The centre of pressure (COP) is represented on screen as an icon. The object is to move the icon as quickly and smoothly as possible to the given target without any foot movement (Tsang and

Hui-Chan, 2003). The positions are set at the theoretical limits of stability. The measurements given for this test are movement velocity (MV), reaction time (RT), end point excursion (EE), maximum excursion (ME) and directional control (DC). The LOS has been shown to be reliable with good test-test reliability(Pickerill and Harter, 2011; Lininger et al., 2018) and compared well, providing more dynamic postural stability information than other equipment (Pickerill and Harter, 2011). Poorer performance in the LOS test is suggestive of poor motor control or lower extremity weakness (Schieppati et al., 1994; NeuroCom International Inc., 2007). Deficiencies in the LOS may have implications for activities of daily living such as reaching for items (NeuroCom International Inc., 2007). Seven participants failed to record a score for all trials in the LOS test protocol.

7.2.4 Statistical analyses

Statistical analyses were performed with IBM SPSS Statistics (Version 26; IBM Corp., Armonk, NY, USA). Visual assessment and Kolmogorov-Smirnov tests were used to assess normality. Where normality was established, between group differences were assessed using one-way analysis of variance (ANOVA). Non-parametric Kruskal-Wallis tests were used when normality could not be established. Bonferroni corrections were made for multiple comparisons and significance values adjusted. Cohen's d effect size calculations were performed using the R programming language and the following classifications were used: <0.2 = trivial; 0.2-0.5 = small; 0.5-0.8 = moderate; >0.8 = large (Cohen, 1988). Assessment of correlation was performed using a Pearson correlation coefficient and a Spearman rank correlation when normality could not be assumed.

Further analysis was performed with group further separated by osteoarthritis diagnosis, as determined form the GHQ. In this case, between group comparisons were performed using independent samples T-test or Mann-Whitney U-test. Covariance was analysed using Quades ANCOVA

7.3 Results

The outcomes for the LOS, mCTSIB and SET protocols are presented in Table 7.2.

Table 7.2	Elite Rugby	Amateur Rugby	Non-contact	*P*			Cohen's *d*		
				Elite vs. Amateur	Elite vs. Non-contact	Amateur vs. Non-contact	Elite vs. Amateur	Elite vs. Non-contact	Amateur vs. Non-contact
MV Comp	5.40 ±1.78	5.60 ±1.52	5.53 ±1.68	1.000	1.000	1.000	0.12	0.08	0.04
EE Comp	76.0 ±9.2	76.8 ±9.8	75.0 ±8.6	1.000	1.000	1.000	0.08	0.12	0.20
ME Comp	90.6 ±6.8	92.1 ±6.7	88.5 ±7.7	1.000	0.801	0.166	0.22	0.29	0.50
RT Comp*	0.63 ±0.15	0.64 ±0.12	0.61 ±0.13	0.568			0.06	0.11	0.19
DC Comp*	78.2 ±6.3	77.7 ±5.8	74.2 ±8.3	0.099			0.08	0.57	0.52
Firm EO*	0.24 ±0.07	0.24 ±0.06	0.26 ±0.09	0.535			0.06	0.33	0.29
Firm EC*	0.38 ±0.16	0.35 ±0.13	0.42 ±0.26	0.508			0.02	0.18	0.34
Foam EO*	0.51 ±0.14	0.53 ±0.19	0.53 ±0.15	0.875			0.11	0.12	-
Foam EC*	1.21 ±0.32	1.11 ±0.29	1.11 ±0.36	0.238			0.33	0.29	0.02
Comp mCTSIB*	0.59 ±0.13	0.57 ±0.12	0.59 ±0.17	0.791			0.15	-	0.13
Firm Double*	0.91 ±0.35	0.90 ±0.28	0.97 ±0.45	0.610			0.03	0.16	0.20
Firm Single*	2.96 ±1.18	3.06 ±1.40	3.76 ±2.01	0.262			0.08	0.51	0.43
Firm Tandem*	3.45 ±2.35	3.23 ±2.24	3.29 ±2.18	0.952			0.10	0.07	0.03
Foam Double*	2.85 ±0.97	2.62 ±0.94	2.81 ±1.11	0.455			0.24	0.03	0.20
Foam Single	4.94 ±1.46	4.94 ±1.39	5.33 ±1.54	1.000	0.883	0.852	-	0.26	0.27
Foam Tandem*	7.19 ±2.81	7.31 ±2.62	8.05 ±3.00	0.393			0.04	0.30	0.27
SET Comp	3.72 ±0.94	3.68 ±0.99	4.01 ±1.14	1.000	0.791	0.557	0.05	0.28	0.32

* Data not normally distributed

There were no significant differences between any of the groups in all the measures of balance.

7.3.1 Age and balance

When the full group (n=112) was analysed to determine relationships, several notable correlations were found. Increasing age was associated with increasing sway velocity in all measures of the mCTSIB (mCTSIB Composite, r =0.467, p <0.001). Strong associations (p <0.005) were observed between age and outcomes of the limits of stability (LOS) protocol. Negative correlations were seen with age and movement velocity (MV), endpoint excursion (EE), maximum excursion (ME) and directional control (DC). As age increased, reaction time (RT), also increased (r =0.235, p =0.016). Furthermore, in all conditions of the stability evaluation test (SET), there were strong associations between age and performance. The SET composite score correlating strongly (r = 0.526, p <0.001).

7.3.2 Weight, BMI, body composition and balance

Weight was positively associated with the directional control outcome in the LOS protocol (r = 0.216, p =0.027). BMI was positively associated with sway velocity in the 'firm eyes open' and 'foam eyes closed' conditions of the mCTSIB protocol (mCTSIB composite r =0.220, p =0.02). In the LOS protocol, associations were seen with reaction time (RT; r = 0.2, p =0.041), movement velocity (MV; r =-0.218, p =0.025) and directional control (DC; r = 0.196, p =0.046). The SET demonstrated an association with sway velocity in the 'foam double' condition (r =0.299, p =0.001). Lean mass in the legs positively correlated with directional control in the LOS protocol (r =0.213, p =0.029) and negatively with sway velocity in the single leg conditions (firm and foam) of the SET (p <0.05). Higher body fat percentage was associated with higher sway velocity in most conditions of the mCTSIB (p <0.05), lower movement velocity in the LOS (r =-0.292, p =0.003). In the SET protocol, higher body fat percentage associated with higher sway velocity in the double leg conditions (firm and foam, p <0.05). The only association with muscle quality was observed with movement velocity of the LOS protocol (r =0.276, p =0.017). No associations seen with concussion and any of the balance outcomes.

7.3.3 Osteoarthritis and balance

Of the 112 participants, n=109 answered questions in the GHQ regarding the presence of physician diagnosed osteoarthritis (OA) in the lower limb (hip, knee, ankle, or foot). 29% (n=33) confirmed a diagnosis of lower limb osteoarthritis. The prevalence of lower limb osteoarthritis was 46% in the elite group (n=18/39), 22% in the amateur group (n=10/45) and 16% in the non-contact group (n=4/21). In total, 32 participants confirmed the presence of lower limb OA compared to 77 who did not report lower limb OA. The OA group were significantly ($p = 0.001$) older (52.1y vs. 44.6y). However, no differences were seen in height, weight, BMI, lean mass, and muscle quality. There were several balance measures where the OA group performed poorer. However, when further analysis was performed, controlling for age, the difference was no longer significant, except for end point excursion ($p = 0.012$) and maximum excursion ($p = 0.037$) from the LOS protocol.

7.4 Discussion

This study investigated the balance performance of a retired elite male rugby player group compared to an age and sex-matched amateur rugby group and a non-contact group. The aims of the study were met by quantifying the balance performance of the groups and determining there were no significant differences in any of the balance performance measurements were seen between the groups. Secondly there were several associations; strong correlations were seen with age and reduced performance measures. Those with self-reported, physician diagnosed lower limb osteoarthritis performed poorer than those without but when age was controlled, the difference was no longer present.

In the context of other studies, athletes have generally performed better than controls in measures of balance (Räty et al., 2002). The demands of the game require that players possess certain key attributes, including strength power and agility (Duthie et al., 2003). Players will be expected to meet many challenges such as change of direction, acceleration, jumping and collision (Wilczynski et al., 2021). However, given the high injury toll in rugby, including concussion (Hind et al., 2020), and

the impact these injuries have on balance performance, rugby athletes may not be expected to have superior performance. The longer term balance performance in rugby players and the potential effects of concussion on balance is not known. Although the incidence of concussion in retired in rugby players has been previously reported as higher than other athletes (Hind et al., 2020), and the detrimental effects of concussion on balance are well documented (Guskiewicz et al., 1996; Parker et al., 2006), there was no difference in balance performance in all tests in the current study. Furthermore, musculoskeletal injury has an impact on balance performance, with increased sway in the injured limb compared to the uninjured limb (Holder-Powell and Rutherford, 2000). The incidence of musculoskeletal injury in rugby is high (Hind et al., 2020), but no significant differences were detected in performance.

The prevalence of lower limb osteoarthritis in the elite group, amateur group and non-contact athletes was higher than that found in other similar studies (Hume et al., 2022), but comparable to findings by Davies et al. (2017). Balance in those with severe OA was compromised compared to those with mild disease (Kim et al., 2011). Moreover, falls risk was increased in those with bilateral knee osteoarthritis (Khalaj et al., 2014). In the current study, sway velocity was significantly increased ($p = 0.005$) in those with lower limb OA in the 'foam eyes-open' condition of the mCTSIB and the 'foam single' condition of the SET. These differences were no longer significant when controlling for age. However, there remained differences in conditions of the LOS protocol. End point excursion and maximum excursion were both less in those with lower limb OA after controlling for age. Less range in this test suggest difficulty in certain tasks of daily living (NeuroCom International Inc., 2007).

Several associations were seen with age and several measures of balance with performance being poorer with increasing age. Sway velocity and reaction time increased with age and movement velocity decreased. There are declines in proprioception with age, potentially accounting for the reduced balance control (Ribeiro and Oliveira, 2007). The ageing process dictates that lean mass decreases and body fat increases (St-Onge and Gallagher, 2010). This study demonstrated associations

between increased lean mass and reduced body fat with lower sway velocity and therefore superior balance performance. This could provide a potential explanation for the decline on balance performance with age. Proprioception is critical in balance control, in particular in the joints of the lower limb (Henry and Baudry, 2019). The reduction of lean mass and strength with age contributes to a reduced level of proprioception. Conversely, while low BMI is associated with increased falls risk (OHID: Office for Health Improvement and Disparities, 2022), results from this study demonstrate reduced measures of balance with higher BMI. Potentially, above a given BMI balance deteriorates in relation to BMI.

There are a number of considerations to make when interpreting the results of this study. Firstly, the mean age of the cohort is 47 years, limiting the application to an ageing population. Secondly, this was a cross-sectional analysis, therefore cause and effect could not be inferred. Thirdly, participants self-selected. Future research should focus on the older population.

In conclusion, ageing was associated with a decline in balance performance. There were no differences in the balance performance between retired elite and community rugby players and retired non-contact counterparts. Those with lower limb osteoarthritis performed worse in the limits of stability test, demonstrating lower limits of stability and at higher risk of falling. Future efforts should focus on treatment and prevention of osteoarthritis, given the high prevalence in retired players.

Chapter 8.0 Overall Discussion

These studies present a body of work which strives to make a comparative analysis of retired male rugby union and rugby league players with retired non-contact athletes in several areas of musculoskeletal health. They form a thesis where each study follows on from the next but could be read as an individual piece of work. This final chapter aims to bring together the separate studies with a discussion of the findings and where this work contributes to the literature, alongside limitations of the research and some areas for future research.

8.1 Research aims

This research addressed the following aims:

1. Identify a method for assessing lumbar spine bone density in men under 50 years and determine the impact of vertebral anomalies in the lumbar spine and the effect on bone density calculation.
2. Determine the spine and hip bone density in retried rugby players compared to age-matched, retired non-contact athletes.
3. Identify past and current levels of participation in osteogenic physical activity and determine the association with the bone density.
4. Compare muscle mass, strength, muscle quality and body fat levels across retired rugby players and retired non-contact athletes.
5. Assess balance performance between retired rugby players and non-contact athletes and associations with previous injury. Elucidate potential risk of falling and difficulties with tasks of daily living.

8.2 Summary of key findings

- There was a high number of vertebral anomalies that significantly altered the BMD calculation at the lumbar spine and excluding these vertebrae from the BMD assessment

reduced final BMD scores. This method was developed by applying guidelines reserved for those over 50 years, and assessing BMD, particularly where there is risk of vertebral anomalies in younger athletes, such as contact sports athletes.

- The elite rugby group achieved a higher bone density score at the hip neck, but there were no differences between the amateur rugby group and the retired non-contact athletes in all measures of bone density. Furthermore, when bone density measures were adjusted for sex, weight and age to calculate Z-sores, there were no differences across all groups.
- Higher volume of current (bone specific) physical activity were seen in the elite rugby group in comparison with the amateur and non-contact groups.
- There were no correlations between past (bone specific) physical activity and any measures of bone density.
- The cBPAQ score positively correlated with hip and spine BMD.
- Retired elite rugby players had greater lean mass and grip strength than amateur rugby and non-contact athletes.
- No differences were seen in muscle quality across all groups.
- Low prevalence of sarcopenia and osteoporosis in all groups.
- No differences seen in balance performance across all groups.
- Superior balance performance was positively associated with lean mass in the lower limbs and negatively associate with body fat percentage. Worse balance performance was seen in those with osteoarthritis and was also negatively associated with age.
- There were no associations with a history of concussion and balance performance.

8.3 Discussion

To address the first aim of this thesis was to identify a method for assessing bone density in the lumbar spine in men under 50 years and determine if exclusion of vertebral anomalies from the bone density calculation would affect the bone density score. There are currently guidelines for the

scanning and assessment of bone density in the lumbar spine for men over 50 years and post-menopausal women (ISCD, 2019), which recommend the exclusion of anomalous vertebrae. There are none such guidelines for men under 50 years. Therefore, the recommended methods were applied in this study to determine if exclusion of vertebrae altered the outcome. It is known that the lumbar spine BMD can be subject to spurious elevation in the presence of vertebral anomalies, particularly those from osteoarthritis and previous fracture (Yu et al., 1995; Adams, 2013). A high number of vertebral anomalies were found in the rugby group, not limited to those over 50 years and the exclusion of such vertebrae had a significant effect on the final BMD Z-score and T-score in the rugby group. This may represent the presence of degenerative changes, which have been seen in the rugby players under 50 previously (Scher, 1990). Moreover, a high incidence of vertebral fractures have been demonstrated in currently active professional rugby players, suggesting they may be at risk for such anomalies (Hind et al., 2014), which would potentially impact the BMD assessment. The high injury toll in rugby (Hind et al., 2020) and in particular a high incidence of cervical and lumbar spine injuries (Fuller et al., 2007) would increase the risk of osteoarthritis (Davies et al., 2017) and therefore further influence the measured BMD. The resultant impact on bone density calculation in this population suggests that it would be necessary to apply this method of assessment to allow accurate measurement of BMD at the lumbar spine. Therefore, anomalous vertebrae were excluded from the BMD calculation when performing DXA scans of the lumbar spine for the purposes of the second aim.

The second aim was to determine bone density at the hip and spine and make comparisons between groups. The elite rugby group did demonstrate higher BMD at the hip neck but there were no differences between the groups in other measures of BMD. Furthermore, when BMD was adjusted for age, sex and weight there were no differences between groups. If it were to be assumed that these former players would have had superior bone density during their active careers (Elloumi et al., 2009; Hind et al., 2015), a lack of difference in BMD would suggest that superiority is not maintained into retirement. Comparable results in a sub analysis those over 50 years would also

suggest that age related decline does happen at a rate greater in the rugby groups. The mechanostat theory (Frost, 1987; Frost, 2003), suggests that the bone responds to stimulus is a similar way to a thermostat with temperature and that bone responds depending on the stimulus. The stimulus of rugby training during their careers may potentially increase the tolerance to such stimulus. Therefore, this would then require a greater stimulus to elicit a response (Frost, 1997; Frost et al., 1998; Frost, 2003). The osteocyte cells within the bone are sensitive to the mechanical loading and elicits a biochemical response (Bullock et al., 2019).

There was negative correlation between age and bone density, which would be consistent with the expected age-related decline in bone density (Khosla and Riggs, 2005). However, in relation to the third aim, to identify the levels of bone specific physical activity (Weeks and Beck, 2008); elite rugby players had higher cBPAQ scores. Moreover, higher cBPAQ score were associated with higher BMD scores. There were no relationships between pBPAQ and bone density. The conventional wisdom has suggested that bone development in adolescent years is of greater importance than in that achieved in adulthood (Forwood and Burr, 1993; Kannus et al., 1995), and it is not known how well this is preserved in adulthood. The lack of association between previous (bone specific) physical activity but a positive correlation between current (bone specific) physical activity suggests that it is important to maintain exercise into retirement in order to maintain bone density or offset the age-related loss. Given the elite rugby group had higher cBPAQ score than the amateur rugby and non-contact groups would imply that these athletes are able to maintain some level of physical activity despite the high injury toll and lasting effects in retirement (Hind et al., 2020). Bone density can be impacted in later life with appropriate osteogenic activity (Watson et al., 2018).

The fourth aim was to compare muscle mass, strength and body fat composition across the three groups of elite and amateur rugby players and non-contact athletes. The elite rugby group had greater lean mass and strength compared to the amateur and non-contact group, however there

were no differences in muscle quality. Higher body mass in currently active rugby players (Hind et al., 2015) is present in retirement. Higher levels of lean mass compare well to published reference data (Imboden et al., 2017). However, muscle quality was similar across the groups but given that the rugby group had higher volume of lean mass this would provide a higher functional benefit, with the same muscle quality. Lower muscle quality is consistent with previous studies demonstrating an inverse relationship between muscle mass and quality (Barbat-Artigas et al., 2013). It may be that strength has deteriorated at a greater rate than mass since peak in adulthood. Lower volume of body fat was observed in the elite group compared to the other groups.

The final aim was to assess and compare balance performance across the groups in order to determine a potential risk of falling. There were no differences in balance performance across the groups. The demands of rugby suggest that maintaining good balance is an important attribute and indeed, rugby training is beneficial to balance (Chow et al., 2016). However, the high injury toll may compromise balance in retirement. In particular, concussion is a common injury in rugby (England Professional Rugby Injury Surveillance Project Steering Group, 2021) and is known to have an effect on balance (Broglio and Puetz, 2008).The long term effects of concussion on balance are not known. However, no relationships were seen between concussion and balance performance in this study.

In order to appreciate the full scope of this work, it is necessary to evaluate the separate studies together. If we consider the ageing process and the development of lean mass and bone density across the lifespan, this generally builds to a peak in adulthood. Sports such as rugby have demonstrated that participation is associated with higher bone density, more lean tissue and greater strength than comparable individuals. The ageing process results in reduced bone density, lean tissue and strength, which in turn results in functional decline. This is significant, as reduced lean mass and strength can impact bone density. Reduced bone density increases the risk of fracture. Reduced lean mass and increases the chances of falls, compounded by the reduced bone mass, and increasing the likelihood of fragility fracture.

The retired elite rugby athletes in this study demonstrated superior bone density, lean mass and strength, compared retired non-contact athletes. This confers a functional benefit and with a similar balance performance, affords a reduced risk of fracture. The previous high injury toll does not appear to impact these outcomes

Conclusions, limitations and recommendations for future work

This thesis aimed to build a picture of the musculoskeletal health of rugby players in retirement. While there was no superiority in muscle quality and balance performance, elite rugby players did demonstrate greater levels of bone density, lean mass and strength that would provide a functional benefit when performing tasks of everyday living. Higher levels of physical activity would suggest that although there is a high injury toll in elite rugby this does not appear to prevent participation in beneficial activity in retirement. Furthermore, with comparable balance performance to retired non-contact athletes, there does not appear to be any adverse effect on balance from injuries including concussion. This may represent the benefit of lower limb lean mass offsetting the long term impact of injury and degenerative change.

It should be noted that the study design was cross-sectional and therefore inferences of cause and effect cannot be made. Where the elite rugby group demonstrated superiority in certain measures, a genetic component cannot be ruled out given that a predisposition for greater strength and mass would afford a natural inclination to a sport where these features would provide an obvious advantage. Other influences on the outcome are not limited to the measures performed in this research. The societal influence on an individual to choose to participate in rugby such as previous parental involvement in rugby or 'nurture' and their geographical location and socioeconomic group. Rugby union and Rugby league have been generally not separated in this work, but there are differences in the pathway to these two sports, not least the location of birth, with rugby league very much rooted in northern working class towns (Collins, 2006).

Lifetime and current nutrition has not been accounted for in this research but is a crucial influence on several musculoskeletal factors such as bone density, lean mass and strength (Weaver and Haney, 2010; Weaver et al., 2019). This did not fall within the scope of this study but would be area for future research to explore longitudinally and prospectively.

There exists selection bias, given that participation was voluntary. There may have been a reluctance of those with a perceived loyalty to their sport to engage with the process, with concerns about the outward image of the sport. Conversely, those with a negative view and possible health complications may have been eager to receive a medical assessment and opportunity to seek blame. In addition, while effort was made to assign participants to the appropriate groups using the inclusion criteria, the non-contact group constituted of those whose main sport had been a non-contact sport (predominantly cricket). However, there exists the possibility of some recreational participation in the very popular sport of football.

Further work should focus on long term prospective study of these athletes, beginning at the point of retirement to determine the features developed during an active career. Decline in characteristics of bone mass and muscle strength could be monitored over a given period. Other components of health could also be measured such as volume of visceral fat and markers of cardiometabolic health.

Practical applications

This work provides a view of the musculoskeletal health of retired rugby players. The importance of continued physical activity should be highlighted, and support should be provided to promote opportunities for participation in exercise in retirement. Governing bodies are in a position to provide a structure of support following retirement for these players, namely continued involvement with the game, albeit in a less collision-based format, such as tag rugby. The continued presence of a sporting network could provide physical benefits from exercise and also mental health benefits from continued peer support. Specifically, the benefits of resistance work in a relatively low-risk

environment, such as the gym should be encouraged and opportunities provided for retirees to participate. These athletes have experience performing resistance training such as weight lifting and could continue this activity in retirement, with benefits to bone density, lean mass and strength (O'Bryan et al., 2022). Furthermore, there are benefits for regarding the degenerative conditions associated with a career in rugby. Resistance exercise can improve the symptoms associated with osteoarthritis such as pain and reduced function (Turner et al., 2020) . The provision and monitoring of training regimes could be provided by former clubs and governing bodies to ensure management of pain and selection of the most appropriate exercises (Vincent and Vincent, 2012).

Tremendous impact could be seen by continuing work to reduce injury in current players. The high injury toll in rugby has impact in later life and a reduction in injury levels could mitigate these outcomes. Identifying areas of the game more likely to cause injury, such as tackle height (Tucker et al., 2017) is necessary to inform intervention such as rules altering the tackle height. Further work and intervention is required by governing bodies to identify and reduce injury risk in these areas.

Regarding the care of current rugby players and other contact sports athletes, it is recommended that DXA monitoring of bone density, as exists in elite environments should consider this unique group and their composition. Guidelines from such authorities as the ISCD should include provision and guidance for assessment of the lumbar spine, specifically to remove those vertebrae affected by degenerative disease associated with rugby.

Appendix A: Abstracts of published works

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Original Article

Vertebral Anomalies in Retired Rugby Players and the Impact on Bone Density Calculation of the Lumbar Spine

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Abstract

Dual energy X-ray absorptiometry (DXA) lumbar spine bone mineral density (BMD) measurements are subject to artificial elevation in the presence of structural abnormalities that are more common with age and injury, including osteoarthritis, fracture and osteophytes. The aims of this study were to investigate the presence of vertebral abnormalities on DXA scans in retired rugby players and a nonrugby control group, and to explore the effect of vertebral exclusion on the BMD diagnostic outcome. Eigty-seven male retired rugby players and 51 non-rugby controls from the UK Rugby Health Project participated in the study. Lumbar spine, total hip and femoral neck BMD were measured by DXA and scans were analyzed pre and post exclusion of anomalous vertebrae. Data were analyzed by age group to enable application of T-scores (≥50 y) and Z-scores (<50 y). From 138 lumbar spine scans, 66 required adjustment. One hundred twenty-two vertebral exclusions were made, and 12 lumbar spine scans (10 in retired rugby athletes) were un-reportable (<2 evaluable vertebrae). Vertebral exclusion significantly lowered lumbar spine BMD across all groups ($p<0.01$) and lowered the overall lowest T/Z-score. This effect was more pronounced in rugby groups (age <50 y, $p < 0.001$; age ≥50 y, $p = 0.031$) than in the control groups (age <50y, $p = 0.125$; age ≥50 y, $p = 0.250$). Vertebral abnormalities detected on lumbar spine scans, were highly prevalent and impacted final the T/Z-score in this cohort of retired rugby players. Current guidelines recommend exclusion of abnormalities from lumbar spine scans in adults aged ≥50 years. Our findings suggest that vertebral exclusions should also be applied to lumbar spine scans performed in those aged <50 years, particularly in former contact sports athletes, given their high risk for vertebral deformity.

Key Words: DXA, Bone density, Sport, Athlete; Spine; Vertebral exclusions.

Thieme

Lean Mass, Muscle Strength, and Muscle Quality in Retired Rugby Players: The UK Rugby Health Project

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ABSTRACT

Although athletes from sports such as rugby have greater lean mass and strength during their playing careers, little is known about these characteristics post-retirement. Therefore, this study investigated lean mass, strength, and muscle quality in retired elite and amateur rugby players and non-contact athletes. Retired elite male rugby players (n = 42, 43.9 ± 10.3 y; 101.1 ± 13.4 kg; 1.82 ± 0.09 m), amateur rugby players (n = 46, 48.0 ± 10.5 y; 98.9 ± 16.6 kg; 1.79 ± 0.07 m) and non-contact athletes (n = 30, 51.3 ± 12.5 y; 91.3 ± 13.4 kg; 1.79 ± 0.07 m) received one total body dual-energy X-ray absorptiometry assessment of appendicular lean mass (ALM) and ALM index (ALMI). Grip strength was measured, and muscle quality (grip strength/unit of arm lean mass) was calculated. Sarcopenia was identified as ALMI < 7.23 kg/m^2 and handgrip strength < 37.2 kg. Total lean mass, ALM and grip strength were greater in the elite rugby compared to amateur rugby and non-contact groups ($p < 0.01$). There were no significant differences in muscle quality or sarcopenia prevalence. Retired elite rugby players had greater lean mass and grip strength than amateur rugby and non-contact athletes, although muscle quality was similar. The greater lean mass and strength might reflect genetic influences or previous participation in a highly physical sport.

Sports Medicine
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ORIGINAL RESEARCH ARTICLE

Cumulative Sport-Related Injuries and Longer Term Impact in Retired Male Elite- and Amateur-Level Rugby Code Athletes and Non-contact Athletes: A Retrospective Study

Karen Hind[1,2] · Natalie Konerth[1] · Ian Entwistle[1,2] · Alice Theadom[3] · Gwyn Lewis[4] · Doug King[5] · Paul Chazot[2,6] · Patria Hume[5,3]

Abstract

Background Rugby union and rugby league are popular team contact sports, but they bring a high risk of injury. Although previous studies have reported injury occurrence across one or several seasons, none have explored the total number of injuries sustained across an entire career. As the first to do so, the aim of this study was to report on cumulative injuries and their perceived long-term impact in retired rugby code athletes compared to athletes from non-contact sports.

Methods One hundred and eighty-nine former rugby code athletes (rugby union $n=145$; rugby league $n=44$) and 65 former non-contact athletes were recruited to the UK Rugby Health Project between September 2016 and December 2018. Details on sports participation, sports injuries and concussion history, sports injury-related surgeries, and previous and current health were obtained from a validated, online self-report questionnaire.

Results Former elite rugby code athletes ($n=83$) reported more total injuries per player (median 39, IQR 35) than former amateur rugby code athletes ($n=106$; median 23, IQR 30; $p=0.014$) and non-contact sports athletes ($n=65$; median 7.5, IQR 15; $p<0.001$). Concussion was the most frequently reported injury for the elite and amateur rugby code groups, followed by upper/lower back and knee ligament injuries. These injuries also presented with the highest recurrence. Rugby code groups reported a higher continued impact of previous concussion, neck injuries, shoulder dislocation, ACL tears, and knee ligament injuries ($p=0.003$–0.045). The reported prevalence of osteoarthritis was more than twofold greater in the elite rugby code group than in non-contact athletes (51% v 22%, $p<0.001$). The prevalence of back pain and/or severe and regular joint pain was high across all groups (47–80%), particularly the elite rugby code group. The total number of joint injuries and sport injury-related surgeries was higher in those who reported current osteoarthritis and current severe and regular joint pain ($p<0.001$–$p=0.028$).

Conclusion Across multiple injury types, past participation in rugby union and rugby league, particularly at elite level, is associated with a high cumulative injury load and a continued impact of previous injuries post-retirement. Given the high number of reported concussions (and their recurrence) and associations between previous injuries during a player's career and current musculoskeletal conditions, efforts should be prioritized to reduce the occurrence and recurrence of injuries in rugby codes at all levels of the sport. Strategies should also be developed for supporting the specific physical health needs of rugby code athletes post-retirement.

Sports Medicine
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ORIGINAL RESEARCH ARTICLE

Mental Health and Wellbeing of Retired Elite and Amateur Rugby Players and Non-contact Athletes and Associations with Sports-Related Concussion: The UK Rugby Health Project

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Abstract

Background Concerns have intensified over the health and wellbeing of rugby union and league players, and, in particular, about the longer-term effects of concussion. The purpose of this study was to investigate whether there were differences in mental health, sleep and alcohol use between retired elite and amateur rugby code players and non-contact athletes, and to explore associations with sports-related concussion.

Methods 189 retired elite (ER, $n=83$) and amateur (AR, $n=106$) rugby code players (rugby union $n=145$; rugby league $n=44$) and 65 former non-contact athletes (NC) were recruited to the UK Rugby Health Project between 2016 and 2018. Details on sports participation and concussion history were obtained by questionnaire, which also included questions on mental health, anger, sleep, mood, alcohol use, social connections and retirement from injury. Data were compared between sports groups (ER, AR and NC), between exposure of three or more or five or more concussions and for years in sport.

Results ER reported more concussions than AR (5.9 ± 6.3 vs. 3.7 ± 6.3, $p=0.022$) and NC (0.4 ± 1.0, $p<0.001$). ER had a higher overall negative mental health score (indicating poor mental health) than AR (10.4 ± 6.3 vs. 7.4 ± 6.5, $d=0.47$, $p=0.003$) and NC (7.1 ± 4.8, $d=0.57$, $p=0.006$) and a lower overall positive score (indicating good mental health) than NC (8.9 ± 4.1 vs. 10.7 ± 3.4, $d=0.46$, $p=0.021$). Negative scores were highest and positive scores lowest in those reporting three or more concussions ($d=0.36$, $p=0.008$; $d=0.28$, $p=0.040$, respectively) or five or more concussions ($d=0.56$, $p<0.001$; $d=0.325$, $p=0.035$, respectively). Reported symptoms for sleep disruption were more prevalent in ER than NC, and in former athletes with three or more concussions ($d=0.41$–0.605, $p<0.05$). There were no significant differences in alcohol score ($p=0.733$). Global anger score and covert anger expression was higher in former athletes with five or more concussions ($d=0.32$, $p=0.035$; $d=0.37$, $p=0.016$). AR reported greater attachment to friends than NC ($d=0.46$, $p=0.033$) and 20% of ER reported that they would not turn to anyone if they had a problem or felt upset about anything.

Conclusion There was a significantly higher prevalence of adverse mental health and sleep disruption in ER and in former athletes who reported a higher number of concussions. Anger and irritability were more prevalent in former athletes with a history of five or more concussions. Strategies are needed to address mental health and sleep disturbance in elite rugby code athletes, who are also less likely to seek help should they need it. Further research is needed to elucidate causation, and the neurobiological connection between concussion, sub-concussions and longer-term psychological health and wellbeing.

Appendix B: Participant information sheet and Consent form

Retired Athlete Health Project - Clinical Assessments

PARTICIPANT INFORMATION SHEET

BACKGROUND TO THE STUDY

In any sport, injuries are common and an expected risk of the game. Efforts to reduce the risk of serious injury have been made by the governing bodies over the last decade, however little is known about the long term health of players following retirement from the game. The aim of this project is to investigate, in depth, player health post retirement so that strategies can be identified for supporting long term player welfare.

INVITATION TO PARTICIPATE

You are invited to take part in **Part C, D and E - Clinical Assessments** of the Retired Athlete Health Project. *Parts A (general health questionnaire) and B (CNSVS) should already have been completed, but if not, please let the research team know.* Your participation in this research is voluntary. You are free to withdraw consent and discontinue participation at anytime without influencing any present and/or future involvement with the involved organisations.

Your consent to take part in this research will be indicated via the consent form. Consenting indicates that you are freely happy to participate, and that there has been no coercion or inducement to participate by the researchers.

WHAT IS THE PURPOSE OF THE STUDY?

- The purpose of the study is to investigate the long-term effects of sport on musculoskeletal health (bone, joint and muscle), neuromuscular/balance capability, vision and cardiometabolic health in retired international/national and community level Rugby players from the UK in comparison with retired 'non-contact' athletes, and non-athlete controls.

- Your clinical data will be combined with your results from the online General Health Questionnaire and CNSVS test.

- Some of the data from this UK project will be combined with data from other countries including New Zealand, Canada and Australia, under the umbrella of 'Global Athlete Health'. *All data will be fully anonymised before sharing.*

- The anonymised results of this study will be used towards a PhD, presented at conferences, reports to governing bodies, the general public and submitted to peer-reviewed journals for publication.

HOW WAS I CHOSEN TO BE ASKED TO PARTICIPATE IN THE STUDY?

You have been invited to take part because you are either:

a) A retired elite/professional level rugby player

b) A retired amateur level rugby player

c) An individual who has **not** played rugby or any other contact sport (e.g. football, hockey, ice hockey, martial arts, boxing etc.) post-school.

d) An individual who has not taken part in any organised, competitive sport post-school

WHAT WILL HAPPEN IF I DECIDE TO TAKE PART?

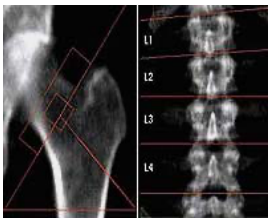
You will be invited to a clinical appointment at the Carnegie Research Institute, Headingley Campus, Leeds Beckett University, LS6 3QS. Your appointment can be arranged at a time suitable to you, and will last around 3 hours in total. You should not exercise within 24 hours of the appointment, or consume stimulants (such as alcohol or caffeine) 12 hours prior to the study. You should also make sure that you are fully hydrated. A morning appointment will be made for you and we ask that you attend after an overnight fast (eg before breakfast) so that the blood test and DXA results are as accurate as possible. A snack and drink will be provided for mid appointment.

PART C: MUSCULOSKELETAL ASSESSMENT

Physical Activity Questionnaire

You will be asked to complete a questionnaire, giving details of any sports and other physical activity you have regularly participated in previously.

Dual energy X-ray absorptiometry (DXA) scans



Dual energy X-ray absorptiometry (DXA) provides quick and accurate assessments of bone strength, vertebral fracture and body composition. You will receive five scans with a total assessment time including feedback = 30 minutes.

1 x total body scan for body composition (muscle mass, fat mass and bone mineral content). 7-12 minutes

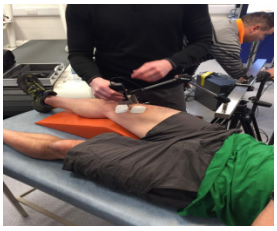
1 x lumbar (lower) spine scan for bone density. ~60 seconds

1 x total hip scan for bone density, bone geometry and osteoarthritis ~2 minutes

1 x knee scan for osteoarthritis ~2 minutes

1 x vertebral fracture assessment to identify any existing/old fractures of the spine. 4-5 minutes

Muscle quality assessment



We will use tensiomyography (TMG) to evaluate the quality of your muscle. You do not need to exercise for this test. The assessment is made by placing a probe on the middle of the muscle at rest, through which a small electrical pulse is delivered. We will record how quick your muscle responds and relaxes. This test is NOT invasive. The total test time will be 20 minutes.

Strength assessment

Hand grip strength will be assessed using a handgrip dynamometer. This requires you to squeeze the handle of the dynamometer in order to measure your grip strength. This test should take approximately 15 minutes.

Successful completion of a screening questionnaire, and a blood pressure measurement will be required prior to the strength assessment and TMG.

PART D: NEUROMUSCULAR AND KING DEVICK TEST

Your balance/neuromuscular capabilities will be tested using the Natus VSR Sports Balance System. You will be asked to stand on a force plate and adopt a variety of stances (including squat). The balance tests should take no longer than 25 minutes.

You will also be asked to complete the King Devick Test which is increasing in use for the diagnosis of concussion. This test is conducted on an IPad and takes around 10 minutes. It involves you reading out a series of numbers from left to right.

PART E: CARDIOMETABOLIC TESTS

The cardiometabolic tests include a short questionnaire that is specific to cardiac risk. You will be invited to receive a resting electrocardiogram (ECG) which will record the electrical activity of your heart using electrodes placed on the skin. These electrodes detect the tiny electrical changes on the skin that arise from the heart muscle depolarizing during each heartbeat. This test should take no longer than 15 minutes.

You will also be invited to provide small finger-tip and venous blood samples for analysis of cardiometabolic, inflammation and neurological markers. The blood tests will be undertaken by specialist, trained personnel.

WHAT ARE THE DISCOMFORTS AND RISKS?

- The DXA scans involve a dose of ionising radiation which is 17μSv. This dose has been evaluated and approved by our medical physics expert and clinical radiation expert.
- From our studies and studies by other groups to date, no discomfort or side effects have been reported for TMG.
- Risk of injury from the strength tests is low, following successful screening.
- There is a risk of falling during the balance test, and we will minimise this risk by making a member of the team is close to you during all testing.
- The risk of discomfort from blood sampling will be minimised through our experienced and certified phlebotomist, following our standardised procedures.

WHAT ARE THE BENEFITS?

- All individuals taking part in the Clinical tests will receive a copy of the DXA results and feedback on all other tests. This will give you useful information about your current health.
- Information gained from this research has potential to help shape player welfare strategies, care for players post retirement and develop prognostic indicators of value to athletes, clinicians, physical

conditioners and coaches within sport towards a strategy to tackle concussion and degenerative joint disease.

HOW IS MY PRIVACY PROTECTED?

- Data will be coded and held in secure storage under the responsibility of the principal investigator.
- Names or personal identifying information will not be shared outside of the research team and will not be published.
- Only the investigators will have access to computerised data.

OPPORTUNITY TO CONSIDER INVITATION

- Please take the necessary time you need to consider the invitation to participate in this research.
- It is reiterated that your participation in this research is completely voluntary.
- If you require further information about the research topic please feel free to contact Dr Karen Hind (details are at the bottom of this information sheet).
- You may withdraw from the study at any time without there being any adverse consequences of any kind.
- If you would like to withdraw, please contact Ian Entwistle or Dr Karen Hind, at any time stating your intention to withdraw (see contact details below).
- You may ask for a copy of your results at any time and you have the option of requesting a report of the research outcomes at the completion of the study.

HOW DO I AGREE TO TAKE PART?

By completing and signing the consent form. This also means that you have read and understood all the information contained in this participant information sheet and have clarified any details prior to taking part.

CONTACT DETAILS

If you have any questions please feel free to contact the Principal Investigators, Mr Ian Entwistle (PhD student) or Dr. Karen Hind (Director of Studies and UKRHP Lead)

Email: i.entwistle@leedsbeckett.ac.uk and k.hind@leedsbeckett.ac.uk

Any concerns regarding the nature of this project should be notified in the first instance to the Principal Investigators.

For independent advice on the project please contact Local Research Ethics Coordinator, Dr Matthew Barlow, matthew.barlow@leedsbeckett.ac.uk.

Research Team Address: Fairfax Hall, School of Sport, Carnegie Faculty, Leeds Beckett University, Headingley Campus, Leeds, LS6 3QS, United Kingdom.

Telephone: 0113 8123539

Approved by the Carnegie Faculty Research Ethics Committee on 15th June 2016

UK Rugby Health Project - Parts C, D and E - Clinical Assessments

CONSENT FORM

	Please circle
1. I have read and understood the Participant Information Sheet, and have had my questions answered.	Yes No
2. I am happy to complete the physical activity questionnaire.	Yes No
3. I am happy to receive the DXA scans and understand that this involves exposure to a dose of ionising radiation.	Yes No
4. I am happy to receive TMG testing.	Yes No
5. I am happy to complete the screening questionnaire and undergo blood pressure/heart rate assessment.	Yes No
6. I am happy to undergo the strength assessment using the handgrip dynamometer.	Yes No
7. I am happy to take part in the Balance Test and complete the King-Devick Test.	Yes No
8. I am happy to donate small samples of blood (finger-tip and venous) for analysis of health markers. I will notify researchers if I have a needle phobia or I am susceptible to fainting prior to the procedure.	Yes No
9. I am happy to receive ECG testing and complete the cardiovascular health questionnaire.	Yes No
10. I understand that I am free to withdraw from the study at anytime without giving reason and without this affecting my future care, and that I understand how to withdraw.	Yes No
11. I understand that upon my request any personal data will be removed from the study database should I wish to withdraw my participation.	Yes No
12. I am happy for my results to be used confidentially for this research project, a PhD and for subsequent publications and presentations, including those to the sport governing bodies.	Yes No
13. I understand that if my results suggest incidental health findings, that I will be advised to seek medical attention from my GP.	Yes No

Participant Name:	Signed:	Date:
Researcher Name:	Signed:	Date:

Appendix C: Screening Questionnaire

UK Rugby Health Study Screening Questionnaire

Name: _____ **ID:** _____

LEEDS BECKETT UNIVERSITY

Preliminary Questions		
Have you had any alcohol to drink in the last 24 hours?	**Yes**	**No**
If yes, how many units and how long ago?	**Units……..**	**Hours……..**
Have you exercised in the last 24 hours?	**Yes**	**No**
Have you eaten in the last 12 hours?	**Yes**	**No**
Have you consumed caffeine in the last 12 hours?	**Yes**	**No**
Are there any other factors that may affect your results today? E.g. viral infection, injury, smoking, exercise. If so, please give details		

Signs or Symptoms	**S/S**	**No S/S**
Q 1. Do you ever have pain or discomfort in your chest or surrounding areas (neck, jaw, arms or other areas)?	**Yes**	**No**
Q 2. Are you ever short of breath at rest or with mild exertion?	**Yes**	**No**
Q 3. Have you ever experienced dizziness or loss of consciousness during or shortly after exercise?	**Yes**	**No**
Q 4. Have you ever been short of breath at rest in the recumbent position or had an attack of breathlessness in the middle of the night which was relieved by sitting up?	**Yes**	**No**
Q 5. Do your ankles ever become swollen (other than as a result of an injury)?	**Yes**	**No**
Q 6. Do you ever have palpitations (the unpleasant awareness of the heart beating in your chest) or an unusual period of rapid heart rate?	**Yes**	**No**
Q 7. Do you ever suffer from cramp-like pains in your legs, brought on by exertion and relieved after 1-2 minutes of rest?	**Yes**	**No**
Q 8. Has a doctor ever said you have a heart murmur?	**Yes**	**No**
Q 9. Do you feel unusually fatigued or find it difficult to breathe with usual activities?	**Yes**	**No**
SIGNS/SYMPTOMS OF DISEASE	**YES / NO**	

Personal History of Disease	**History of Disease**	**No History of Disease**
Q 10. Heart disease	**Yes**	**No**
Q 11. Peripheral vascular disease	**Yes**	**No**
Q 12. Cerebrovascular disease (e.g. stroke)	**Yes**	**No**
Q 13. Chronic obstructive pulmonary disease (emphysema/chronic bronchitis)	**Yes**	**No**
Q 14. Asthma	**Yes**	**No**
Q 15. Interstitial lung disease	**Yes**	**No**
Q 16. Cystic fibrosis	**Yes**	**No**
Q 17. Diabetes mellitus	**Yes**	**No**
Q 18. Thyroid disorder	**Yes**	**No**
Q 19. Renal disease	**Yes**	**No**
Q 20. Liver disease	**Yes**	**No**
HISTORY OF DISEASE	**YES / NO**	

Other conditions	Condition	Condition
Q 21. Do you have any bone or joint problems such as arthritis or a past injury that might get worse with exercise? **(Exercise testing may need delaying or modifying)**	**Yes**	**No**
Q 22. Do you have any other problem that might make it difficult for you to do strenuous exercise? **Details**:	**Yes**	**No**
Q 23. Are you or have you recently been pregnant?	**Yes**	**No**
Q 24. Are you on any prescription medications? **List:**	**Yes**	**No**

Blood pressure

Resting blood pressure: SBP = _____ *mmHg, DBP* = _____ *mmHg*

Do you have an implanted cardiac pacemaker or defibrillator or deep brain stimulator?	**Yes**	**No**
Do you have sensitive skin, surface inflammations, skin irritations, broken skin or scarring?	**Yes**	**No**
Do you have any muscle soreness as a result of exercise?	**Yes**	**No**
Do you have any other major previous or current health conditions? E.g. Cancer, if so please give details:	**Yes**	**No**

- I confirm that the above information which I have provided to Leeds Beckett University is true and accurate to the best of my knowledge and belief and I understand that I must notify promptly of any changes to the information.
- I understand that the information I have provided above may be used as part of an anonymised dataset by staff or students of the School of Sport for completion of coursework or for research or audit purposes (with the appropriate ethical approval in place).

Participant Signature: **Date:**

Researcher Signature: **Date:**

Bone-Specific Physical Activity Questionnaire (BPAQ)

SUBJECT ID:	DATE:

1. **Please list <u>any</u> sports or other physical activities you have participated in regularly. Please <u>tick</u> the boxes to indicate how old you were for each sport/activity and how many years you participated for.**

Age: / Activities	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30

Age: / Activities	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60

Age: / Activities	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80	81	82	83	84	85	86	87	88	89	90

Bone-Specific Physical Activity Questionnaire (BPAQ)

SUBJECT ID:	DATE:

2. **Please list the sports or other physical activities (be as specific as possible) you participated in regularly during the last 12 months and indicate the average frequency (sessions per week).**

Activity: _____ Frequency (per week): _____

Activity: _____ Frequency (per week): _____

Activity: _____ Frequency (per week): _____

Activity: _____ Frequency (per week): _____

Activity: _____ Frequency (per week): _____

Activity: _____ Frequency (per week): _____

Activity: _____ Frequency (per week): _____

Activity: _____ Frequency (per week): _____

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