The University of Rome "Foro Italico"
The German Sport University, Cologne
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The Norwegian School of Sport Sciences, Oslo
The University of Vienna

European Master in Health and Physical Activity

(Laurea Magistrale Internazionale in Attività Fisica e Salute)
LM 67- I

"Bone health in Norwegian endurance athletes"

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Academic Year 2015-2017

Acknowledgements

This master thesis concludes five interesting and educational years at the Norwegian School of Sport Science (NIH) and the University of Rome “Foro Italico”. I would like to express my gratitude to the staff at both universities and partner universities. It has been a great experience.

I would like to direct a special thanks to my supervisors, Trine Stensrud and Benjamin Clarsen. Thank you for your continuous support, encouraging words, and academic insight. Furthermore, I would like to thank Ina Garthe and Morten Mørland at the Norwegian Olympic Training Centre for their help with data analysis and recruitment. I would like to thank Erling Hisdal at NIH, Veslemøy at Norwegian Athletic Federation, Kaja Sætre at NIMI and Carl Erik Pedersen at the Norwegian Cycling Federation for the help with recruitment of athletes.

Thank you to all the test subjects who have participated in the study.

A special thanks to my mother for giving a valued second opinion and helping me with the structure of my paper. Thank you to my dear girlfriend, for always being there and believing in me.

Abstract

Background: Physical activity is generally accepted to be good for skeletal health. However, the effect seems limited to weight-bearing activity. Multiple studies have demonstrated that athletes competing in non-weight-bearing activities such as swimming and cycling, are at risk of developing low bone mineral density (BMD). Moreover, certain weight-bearing activities such as long distance running have been associated with low BMD.

Objective: The primary objective was to evaluate bone health in Norwegian male and female endurance athletes and to identify cases of low BMD. A secondary objective was to identify possible risk factors associated with low BMD.

Methods: Twenty-one runners, 11 females and 10 males, and 19 cyclists, 7 females and 12 were enrolled in this cross-sectional study. DEXA measurement of BMD in total body, dual proximal femur (DP femur) and lumbar spine was measured. Furthermore, a questionnaire regarding training, injuries, nutrition and health variables was administered on the test subjects.

Results: Cyclists had significantly lower BMD for all measured sites ($p \leq 0{,}05$). 10 of 19 cyclists were classified as having low bone mass per ASCM criteria (Z-score ≤ -1), despite reporting to train heavy resistance training on the lower extremities. Low BMD was most prevalent at the lumbar spine. Type of sport was the only significant predictor of low BMD.

Conclusion: Elite Norwegian cyclists had lower BMD compared to runners, and a large portion were classified as having low BMD as per ASCM criteria, despite that cyclists reported to perform heavy resistance training. Interventions to increase BMD in cyclists are necessary.

Abbreviations		**Unit**
ASCM	American Collage of Sport Medicine	-
BMD	Bone mineral density	g/cm^2
BMI	Body mass index	kg/m^2
CI	Cumulative incidence	-
DEXA	Dual Energy X-ray absorptiometry	-
DP Femur	Dual proximal femur	-
FFM	Fat free mass	kg
FM	Fat mass	kg
IGF-1	Insulin-like growth factors	
IQR	Interquartile range	-
Mdn	Median	-
NIH	Norwegian School of Sport Sciences	-
OPG	osteoprotegerin	
OR	Odds ratio	-
PA	Physical activity	
PBM	Peek bone mass	
PTH	Parathyroid hormone	
pQCT	Peripheral quantitative computer tomography	-
RR	Risk Ratio	-
RANK	Receptor activator of NF-kappaB	
RANKL	Receptor activator of NF-kappaB ligand	
SD	Standard deviation	-
QCT	Quantitative computer tomography	-
WHO	World Health Organization	

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1.0. Introduction and background of the study

It is generally acknowledged that physical activity (PA) has a beneficial effect on the skeleton. However, the effect seems limited to weight-bearing activity. About 10 percent of the skeleton is remodeled every year, and the relationship between bone resorption and bone remodeling is affected by the strain put on the skeleton (Sand, Sjaastad & Haug, 2011). Peak Bone Mass (PBM) represents the peek density of the skeleton and is achieved in the third decade (Garthe & Helle, 2011). From this point on, PBM can only be maintained. Thus, weight-bearing activity during childhood and adolescent years are imperative for bone health later in life.

Non-weight-bearing activity, such as swimming and cycling does not seem to elicit the bone remodeling process (Stewart & Hannan, 2000; Medellin, Shabani, Lounana, Fardellone & Campion, 2009). It is a growing concern that the lack of weight-bearing activity, combined with low weight and nutritional deficits, could lead to a sub-optimal PBM and poor preservation of bone mass in cyclists (Nagle & Brooks, 2011).

Previous research has demonstrated high prevalence of low BMD in competitive cyclists (Smathers, Bemben, M.G. & Bemben, D.A., 2009; Nichols, Palmer & Levy, 2003). Furthermore, low BMD has been observed in elite middle- and long distance runners (Chen, Tenforde & Fredericson, 2013; Burrows & Bird, 2000). Similarly, to cyclists, performance in middle- and long distance runners is associated with low weight. Low weight and energy deficit has been linked to low BMD and hormonal disturbances in several studies (Lucia et al., 2001; Wheeler, Singh, Pierce, Epling & Cumming, 1991; Hackney, 1989). Thus, it is likely

that runners at elite levels have similar risk of developing low BMD, despite competing in a weight-bearing activity.

Cycling has gained popularity in Norway the last ten years. Number of UCI continental teams (third level in professional cycling) has increased from 2 to 5 teams in one decade. Furthermore, number of continental riders has increased from 21 to 46. Furthermore, a Norwegian professional women's team was founded in 2008. As the sport was considered obscure until recently, very little research has been conducted on Norwegian cyclists and bone health. Based on the alarming results from international research, mapping the BMD status in Norwegian elite cyclists is important. Middle and long distance runners are chosen as a comparison group as they have similar training volumes and performance is related to leanness.

2.0. The purpose of the study

The main objective of the present study is to evaluate bone health in Norwegian endurance athletes and the prevalence of low bone mass. A secondary objective is to evaluate whether anthropometrical parameters, differences in training modalities, nutrition and prevalence of secondary amenorrhea is associated with BMD.

The purpose of this study is;

A) To compare total body, lumbar spine and dual proximal femur (DP femur) BMD in male- and female elite cyclists to male- and female middle- and long distance runners.
B) Identify prevalence of low BMD classified as athletes having Z-scores of < -1 at one or more of the measured sites.
C) Identify potential association between independent variables and low BMD.

2.1. Hypothesis

Based on the purpose of the study and previous research the main hypothesis of the present study is:

H0: There is no difference in BMD between cyclists and runners.

H1: Runners have higher BMD for at all sites compared to cyclists, based on the difference in mechanical loading of the different sports.

3.0. Theoretical constructs

3.1. Osteoporosis

Osteoporosis is defined as a systematic skeleton disease characterized by low BMD and micro structural impairments in the bone, which increases the risk of fractures (Ribom & Piehl-Aulin, 2009). World Health Organization (1994) classifies osteoporosis as having a T-score of -2.5 or less (see table 1).

Table 3: WHO classification of BMD (Cranney, Tugwell, Wells & Guyatt, 2002)

Classification	T-score
Normal	−1.0 or greater
Low bone mass (osteopenia)	Between −1.0 and −2.5
Osteoporosis	−2.5 or less
Severe osteoporosis	−2.5 or less with a fragility fracture

Table 1: T-score compares the subjects BMD to that of a young-adult sex-matched reference population. It is used to classify BMD in men and women aged 50 or older (Lewiecki & Lane, 2008). BMD = Bone mineral density

3.2. Bone biology

The skeleton is a living dynamic tissue that adapts to its surroundings and serves multiple purposes. The skeleton is responsible for giving humans its body shape by giving structural support to muscles. Furthermore, it protects vital organs and it is a part of the musculoskeletal system that enable us to move (National Library of Medicine, 2017). Moreover, the skeleton acts as a reservoir for minerals such as calcium and phosphate, and it harbors hematopoietic

stem cells (Negishi-Koga & Takayanagi, 2012). The bone can be divided into two main tissues, cortical and trabecular bone. Cortical bone makes up about 80 percent of the total human bone mass (Riggs et al., 2008). Osteons is the main structure of cortical bone and consists mostly of lamella. Cortical bone is thought to play a fundamental role in strengthening of the bone, by resisting bending (Amgen, 2012a). Thus, loss of cortical bone could lead to fractures. Dual proximal femur is mainly made up of cortical bone, and is a common site of osteoporotic fractures (Leib, Lewiecki, Binkley, Hamdy & International Society for Clinical Densitometry, 2004). Trabecular bone is spongy, and is found in the center of the bone. A large percent of the trabecular bone is found in the spine. (Iloascon et al., 2013).

3.3. Bone remodeling

The skeleton is a dynamic tissue that adapts to its surroundings. The removal and formation of bone is necessary to maintain bone homeostasis, bone quality and mineral homeostasis (Hattner, Epker & Frost, 1965; Nakashima, 2013). The main cells responsible for the skeletons remodeling process are osteoclasts and osteoblasts, which brake down and build up bone (Hattner, Epker & Frost, 1965). This process is universal and similar in all types of bone (Väänänen, 1993).

The bone remodeling process can be broken down to three phases, initiation phase, where osteoclasts resorb old bone, the transition from resorption to new bone formation, and bone formation, where osteoblasts lay down new bone (Sims & Gooi, 2008). The process is coordinated by osteoclasts, osteoblasts and other osteoblast lineage cells, such as bone-lining cells and osteocytes (Negishi-Koga & Takayanagi, 2012).

3.4. The remodeling process

Osteoclasts are derived from either macrophages or monocytes. Osteoclasts bind themselves to the bone through a protein called integrin. Between the surface of the bone and the osteoclasts, it is formed a microenvironment, referred to as the sealed zone (Teitelbaum & Ross, 2003). The Ph level within this zone is lowered, thus creating acid which allows osteoclasts to remove or dissolve bone efficiently. This process is necessary to maintain bone homeostasis as osteoclasts remove old and damaged bone.

When all the bone collagen is removed, osteoblast fills the cavities that are created by osteoclasts. Osteoblasts produce osteoid, which is the unmineralized organic portion of the bone matrix prior to the maturation of bone tissue (Amini, 2017). Osteoid are primarily made of collagen. Minerals crystalize around the collagen scaffold and forms a mineral called hydroxyapatite. Hydroxyapatite consists mainly of calcium and phosphate and is together with collagen, the fundamental component of bone (Amgen, 2012b). After the resorbed areas are refilled with newly synthesized bone, some of the osteoblasts undergo apoptosis (cell death), while other revert to lining cells, which covers 90 percent of the bone surface (Manolagas, 2000). Some of the osteoblasts gets trapped within the matrix, and becomes osteocytes (Amgen, 2012b).

3.5. Regulation of osteoclastic and osteoblastic activity

Osteoclast and osteoblast activity is regulated by a various of different signalling pathways and signalling molecules, which are not yet fully understood (Boyce & Xing, 2008). Two of the molecules that are thought to play a key role in maintaining bone homeostasis is RANK Ligand (RANKL) and osteoprotegerin (OPG). Both molecules are secreted from osteoblasts

and exert their effect on osteoclast activity (Negishi-Koga & Takayanagi, 2012). On the surface of osteoclasts RANKL binds to its receptor RANK. This causes osteoblasts to become active. Thus, increasing bone resorption. To counteract this process OPG is secreted from osteoblasts. They act as a decoy for RANKL by binding to its receptor. This causes less osteoclasts activity. Thus, decreasing bone resorption (The Basics of Bone in Health and Disease, 2004).

The balance between RANKL and OPG is modulated by a various of hormones. In particular, parathyroid hormone (PTH) and estrogen plays an important role. PTH increases the expression of RANKL, resulting in increased net resorption. Conversely, oestrogen increases the expression of OPG, which causes net modelling (Amgen, 2012c). These hormones have been shown to be effected by physical activity. Prolonged and intensive exercise, which is typical training modalities for the cohort in the present study, have been demonstrated to effect both PTH and oestrogen (Bouassida et al., 2006; Matthews, 2012). The effect is discussed at a later point.

3.6. Skeleton and its response to physical activity

Hypothesises regarding mechanical loading and its effect on bone structure has been proposed as early as in 1638 by Galileo (Ascenzi, 1993). It is generally accepted that strain put on the skeleton will affect the remodelling process, as illustrated by Wolff's law; "As bones are subjected to stress demands in weight-bearing posture, they will model or alter their shape accordingly" (Wolff, J. The law of bone transformation. Berlin 1892). Skeleton loading shifts the balance in bone turnover toward net formation, which causes bone to grow stronger and more resilient to strain. Conversely, decreased loading causes increased activity in the

osteoclast, resulting in net resorption. This process is referred to as “bone functional adaptation” (Ruff et al., 2006). The skeleton’s response to mechanical strain is depended on the strain magnitude, rate, distribution and cycles in the bone (Lanyon L., 1996).

Harold Frost developed The Mechanostat Theory in the1960s. This was the first comprehensive theory of how mechanical loading resulted in bone adaptation (Hughes & Petit, 2010). The theory assumes that there is a certain magnitude of mechanical stress required to efficiently remodel bone. Anything below this threshold will result in net resorption. Conversely, if the loading exceed a certain threshold, bone remodelling will take place. Stress of 50-200 με is referred to as the trivial loading zone, and will result in net bone resorption. 200 – 2000 με is referred to as the physiological loading zone, which maintains the bone strength, 2000- 4000 με is referred to as the overload zone, which causes an increased osteoblastic activity, which strengthen the bone. Strain over 4000 με is referred to as pathological overload zone. At this strain rate, bone damage is accumulated faster than it can be repaired (Robling & Turner, 2009). This causes microfractures, and could eventually result in complete fracture.

The mechanostat theory is supported by recent research. Mechanical loading is thought to be one of the most important modifiable predictor of bone mass (Tenforde, Barrack, Nattiv & Fredericson, 2016). It is estimated that children with the absence of mechanical loading would only develop 30 – 50 percent of normal bone mass (Robling & Turner, 2009). The dramatic degenerative effect on the skeleton of staying in a non-weight-bearing environment for long periods have been demonstrated in astronauts in space (Ohshima, 2006). Conversely, increased mechanical loading will elicit bone modelling, leading to stronger and more resilient bone. This is illustrated in studies in professional tennis players, where the BMD in

the playing arm has been shown to be approximately 30 percent higher, compared to the other arm (Jones, Priest, Hayes, Tichenor & Nagle, 1977).

3.7. Mechanotransduction

Mechanotransduction is the theory of how bone cells reacts to mechanical loading (Hughes & Petit, 2010; Duncan & Turner, 1995). Mechanotransduction in bone can be divided into four steps. (1) Mechanocoupling, (2) Biochemical coupling, (3) transmission of signal and (4) effector cell response (Duncan & Turner, 1995).

In mechanocoupling, mechanical loading creates strain on the skeleton and the extracellular fluid which is found within the bone (Duncan & Turner, 1995). Biochemical coupling refers to the possible pathways and signaling molecules which are responsible for transferring mechanical strain into biochemical signals (Duncan & Turner, 1995). Transmission of signals were theorized to occur in osteoblasts, osteocytes and bone lining cells through gap junctions. OPG, Insulin-like growth factors (IGF-1) and prostaglandins are possible mediators of this process. The effector cell response, is the final outcome of the skeletal response to mechanical strain. Which is as previously mentioned, depended on the strain magnitude, rate, distribution and cycles in the bone (Lanyon L., 1996).

Recent discoveries have shown that osteocytes play a crucial role in the skeletons reaction to mechanical stress (Dallas, Prideaux & Bonewald, 2013). Osteocytes are the most numeric cell in bones and are found in small cavities within the mineralized bone matrix (Robling & Turner, 2009). The extracellular fluid surrounding the osteocytes are pushed back and forth due to mechanical loading, which creates tension on the cell membrane of osteocytes (Han,

Cowin, Schaffler & Weinbaum, 2004). The pressure exerted on the osteocytes is proportional to the mechanical stress applied on the skeleton (Robling, Duijvelaar, Geevers, Ohashi & Turner, 2001). The mechanical strain is converted to biomechanical signals through several pathways, regulated by osteocytes. Recent research has demonstrated that the Wnt/β pathway is the most important pathway in regulating skeletal adaption to mechanical strain (Roblin et al., 2008; Sapir-Koren & Livshits, 2014). Osteocytes produce a signalling molecule called sclerostin, which inhibits Wnt/β signaling and bone formation, by binding to osteoblasts receptors called receptor related proteins (LRP) 4, 5 and 6 (Amgen, 2012; van Bezooijen et al., 2004). Osteocytes reacts to mechanical loading by producing less sclerostin, thereby increasing the osteoblastic activity and net bone formation. OPG, IGF-1 and prostaglandins are secreted from the bone mineral matrix within minutes after being exposed to mechanical loading through multiple pathways. However, the exact way they mediate the response in osteocytes are still being researched (Bonewald, 2007).

3.8. Nutrition and hormonal factors affecting bone metabolism

3.8.1. Calcium and Vitamin D

Calcium and phosphate are the main constituent of bone along with collagen (Amgen, 2012b). These minerals are also found in the blood, where they are kept at constant levels. A steady state of calcium in serum is necessary as it is essential for muscle contraction and nerve conduction (Garthe & Helle, 2011). Thus, low levels of calcium in the blood will result in the deliberation of calcium from the skeleton to keep the calcium homeostasis. The release of calcium from the skeleton is regulated by PTH. Vitamin D counteracts the effect of PTH. It increases the absorption of calcium from the gut, thus decreasing PTH activity and calcium resorption from the bone (Yan et al., 2009). Vitamin D, is mainly produced in the skin. A cholesterol called 7-Dehydrocholesterol is converted to Vitamin D3 by exposure to the UV

rays from the sun. It goes through several transformation phases, before finally being converted to 1,25 dihydroxy VD3, which is the active form of Vitamin D (Masterjohn, 2006). Thus, an adequate calcium and Vitamin D intake leads to decreased calcium deliberation from the skeleton, and an increased calcium absorption from the gut, which has a protective effect on the skeleton. Conversely, prolonged sub-optimal calcium and Vitamin D intake is associated with fragile bones.

3.8.2. Calcium and vitamin D recommendations

Calcium intake is closely linked to the total energy consumption. Calcium is mainly found in milk, cheese, green vegetables and almonds. It is recommended that both females and males have a calcium intake of minimum 800 mg (Garthe & Helle, 2011). However, in endurance athletes, the need for calcium might be elevated due to the calcium secreted from the skin.

Current recommendations of Vitamin D intake is 7,5 µg for both females and males (Garthe & Helle, 2011). There is no evidence that suggest that athletes require a larger intake, however, there are discussions regarding whether the current recommendations are high enough (Willis et al., 2008). The main source of Vitamin D is sunlight exposure. However, the skin can only synthesize vitamin D when the sun is a leased 40 degrees above the horizon. On northern latitudes, this only happens during the summer months (May-August). This makes it necessary to have an adequate nutritional intake of Vitamin D for most of the year (Garthe & Helle, 2011). When the sun exposure is adequate, there is little required to maintain Vitamin D status. A total of 30 to 45 minutes a day seems adequate to maintain sufficient vitamin D status. This is dependent on the amount of pigments in the skin, age, clothes, air

pollution and other factors. For typical Caucasians, 15-30 minutes of sunlight exposure seems sufficient (Rhodes et al., 2010).

3.8.3. Calcium and vitamin D measurements

There is currently no gold standard of measuring calcium intake. Because of its close regulation within the blood, measurement of calcium in serum does not reveal calcium status (Garthe & Helle, 2011). Thus, questionnaires and food diaries are currently the most used tools for evaluating calcium status. Measurement of BMD could determine long term calcium status; however, it does not reflect current intake.

Vitamin D status is measured by measuring 25(OH)D in the blood (Garthe & Helle, 2011). This reflects the synthesis of the skin and the nutritional intake. Because of the suns important role in Vitamin D synthesis, food questionnaires and diaries are poor predictors of Vitamin D status.

3.8.4. Vitamin K and Vitamin A

Vitamin K deficiency cause a reduction in osteocalcin levels in serum, which have been shown to be associated with bone loss in post-menopausal females (Iwamoto, Takeda & Sato, 2006). Very little research has been conducted on vitamin K intake in young athletes. Rejnmark et al., (2006) showed no effect of Vitamin K intake at 5 and 10- year follow-up in 2016 perimenopausal women (Danish Osteoporosis Prevention Study). Due to limited research and conflicting results regarding Vitamin K and its effect on bone metabolism, it will not be discussed further in the present paper.

Excessive Vitamin A intake has been associated with decreased bone mineral density (Feskanich, Singh, Willett & Colditz, 2002;). An intake above the current recommendations could impair bone health in frail subjects such as post-menopausal women or athletes suffering from triad problematics (Garthe & Helle, 2011). However, it is unlikely to play a central role in bone health as athletes suffering from triad problematics tend to avoid food like liver, milk and fat fish, which are main sources of Vitamin A. No effort has been done to evaluate the status of Vitamin A in the athletes in the present study.

3.8.5. Alcohol, smoking and caffeine

Alcohol and smoking effects the skeleton negatively (Kanis et al., 2005). However, it is unlikely to be relevant for the cohort of the present study given that they are elite athletes. Caffeine has shown to increase bone resorption. However, even small amounts of milk diminish the negative effect caffeine has on the skeleton.

3.9. BMI and fat free mass

An important predictor of bone mass is body mass index (BMI). It is generally accepted that high BMI creates larger mechanical strain on the skeleton, compared to low BMI, which stimulates osteoblastic activity (Kim, Shin, Lee, Im & Lee, 2014). Conversely, a decrease in BMI is associated with lower BMD (Guney, Kisakol, Ozgen, Yilmaz, Yilmaz & Kabalak, 2003). A comprehensive meta analyses including 60 00 men and women, demonstrated that a BMI of 20 versus 25 increased the risk of hip fractures by almost two-fold (RR=1.95; 95% CI, 1.71-2.22) (De Laet et al., 2005).

The relationship of BMI and BMD is likely to be mediated by fat free mass (FFM). Multiple studies have shown FFM to be an important predictor of bone health (Babtista et al., 2012). Higher percentage of lean body mass is associated with weight-bearing exercise such as strength training, which has been shown to positively affect bone health (Tan et al., 2014).

3.10. The female athlete triad

The female athlete triad is a syndrome categorized by low energy availability with or without disordered eating, menstrual dysfunction, and low bone mineral density (Nattiv et al., 2007). The triad was originally described as the interaction and link between disturbed eating, disturbed menstrual cycles and osteoporosis (Yeager, Agostini, Nattiv & Drinkwater,1993; Nattiv, Agostini, Drinkwater & Yeager, 1994)

The athlete triad is prevalent in sports where leanness, aesthetics and apparent benefits of low weight is related to sport performance. Both cyclists and runners have reported high prevalence of triad problematics. Recent studies have shown that these problems are not limited to female athletes. The International Olympic Committee (IOC) introduced a new term, “Relative Energy Deficiency in Sport” (RED-S), to include male athletes. RED-S refers to impaired physiological functioning, caused by energy deficiency relative to energy expenditure (Mountjoy et al., 2014).

3.11. Measurements of BMD

There are three different technologies used to measure BMD, these include dual-energy X-ray absorptiometry (DEXA), quantitative ultrasound (QUS) and (peripheral) quantitative computer tomography (QCT/pQCT) (Lewiecki & Lane, 2008).

3.11.1. DEXA

DEXA is widely recognized as the "gold standard" of BMD measurement (Lewiecki, 2005). Strong correlations between mechanical strength and BMD measured by DEXA have been shown in biomechanical studies (Lotz, Cheal & Hayes, 1991). Furthermore, associations between fracture risk and BMD measured by DEXA have been shown in large epidemiological studies (Marshall, Johnell & Wedel, 1996). World Health Organization (WHO) has based its classification and diagnostics of osteoporosis mainly on reference data obtained by DEXA. DEXA is preferred due to its low radiation exposure and exceptional measurement precision and accuracy (Mazess, Chesnut, McClung & Genant, 1992).

3.11.2. QUS

QUS is a portable tool that is used to measure the peripheral skeleton such as wrists and ankles. It has its advantages in being light, portable and does not use ionizing radiation. QUS measures the speed of sound and broadband ultrasound attenuation, which are factors associated with bone strength. QUS has shown good interrelationship with these parameters and fracture risk (Siris et al., 2001). However, it cannot be used for diagnostic classification and monitoring of the effects of therapy (Lewiecki, Richmond, Miller, 2006). T-scores derived from QUS cannot be used per WHO criteria, as T-scores derived from QUS are generally higher than T-scores derived from DXA (Lewiecki & Lane, 2008).

3.11.3. QCT and pQCT

QCT and pQCT can measure trabecular and cortical volumetric BMD at the axial skeleton and peripheral skeletal sites (Lewiecki & Lane, 2008). It has its advantages over DXA as it can accurate measure changes in BMD and microstructure occurring in cortical and trabecular bone (Luu et al., 2013). It does however, not provide sufficient details regarding bone biology and material properties (Luu et al., 2013). QCT is considered as precise as DEXA in measuring Lumbar spine in post-menopausal women, however, there are currently lacking sufficient evidence regarding men and pre-menopausal females (Engelke et al., 2008). Moreover, QCT is more expensive and has a higher amount of ionizing radiation, compared to DEXA. Furthermore, T-scores derived from QCT cannot be used for classification of osteoporosis, per WHO's criteria, as these criteria are derived from T-scores using DEXA.

3.11.4. T-scores and Z-scores

A common mistake when evaluating bone mineral density in young subjects > 50 years old is to apply T-scores instead of Z-scores. T-score compares the subjects BMD to that of a young-adult sex-matched reference population. It is used to classify BMD in men and women aged 50 or older (Lewiecki & Lane, 2008). A Z-score compares the patient's BMD to an age-, ethnicity- and sex-matched reference population (Lewiecki & Lane, 2008). Z-scores are preferred over T-scores in healthy young males and females, because young subjects are most likely to have different anatomical bone structure compared to postmenopausal females (Lewiecki & Lane, 2008). Furthermore, T-scores are derived from fracture prevalence in post-menopausal females, not pre-menopausal females and young males. The American College of Sport Medicine (ASCM) defines a Z-score between -1 and -2 as "Low BMD" (Nattiv, 2007) and is the criteria used in the present study to characterise sub-optimal bone health status.

4.0. Methods

4.1. Research design

The present study has a descriptive, cross-sectional design. We included 40 healthy male and female elite athletes (19 women and 21 males) competing in either road cycling or middle- and long distance running. The athletes are divided in groups based on their respective sports and gender. The cyclists will be compared to runners, before being separated by gender for further sub-group analyses. BMD expressed as $gCal/cm^2$ is compared between groups. Three key BMD measurements are included; BMD for total body, lumbar spine and dual proximal femur.

Anthropometrical parameters, prevalence of injuries, exercise, nutrition and prevalence of secondary amenorrhea are potential predictors of BMD that was be investigated in the present study. Secondary amenorrhea is defined as disruption of three or more consecutive menstrual cycles (Garthe & Helle, 2011).

The data collection was performed per protocol. Each subject attended a DEXA scan between 7 and 10 am in a fasting state. Twenty-two of the 40 scans were obtained at an earlier point, as part of an annual health scan performed by the Olympic sport center. Of the 22 scans, 16 was conducted the last year, while the remaining six was a maximum of three years old. The subjects received a questionnaire following the DEXA scan. A questionnaire regarding training, injuries, health and nutritional factors was administered to the subjects within a maximum of 2 months after the scan through Questback, an online questionnaire platform. Twelve of the athletes who already had performed a DEXA scan prior to the study, received the questionnaire later than two months after the DEXA scan.

4.2. Recruitment of male cyclists

The sports directors of three Norwegian continental teams were contacted per mail. Two of the three sports directors responded positively to participation. Due to logistical and time aspects, only one sports director and his team chose to participate. Furthermore, professional riders that had performed a DEXA scan at Norwegian School of Sport Sciences (NIH) the last three years were contacted. In total was 12 male cyclists recruited and enrolled in the study. All sports directors and athletes received informed consent and a detailed project description per mail.

All male cyclists had performed a DEXA scan prior to the enrollment and these scans were used in the present study. Six of the scans were performed at NIH, on the same machine (Lunar Prodigy). The scans were conducted by experienced personnel, trained by the same physician. Four scans were performed at the University of Agder, using an equivalent DEXA machine (Lunar Prodigy). Two scans where performed at St. Olavs Hospital, Polyclinic Endocrinology using Hologic. These data were later converted to Lunar Prodigy by cross calibration, using a standardized equation (Genant et al., 1994).

4.3. Recruitment of female cyclists

All female athletes that had competed for the Norwegian national team within the last three years and had performed a DEXA scan were invited to participate in the study. The coach of the national team and the athletes received a mail with a detailed project description and informed consent. In total were 10 athletes contacted. Seven of 10 athletes agreed to participate and were enrolled in the study. All athletes had performed a DEXA scan prior to the study and these scans were used in the present study. All scans were performed on the

same machine (Lunar Prodigy) and conducted by experienced personnel trained by the same physician.

4.4. Recruitment of elite middle- and long distance runners

The runners were recruited from the national team and the best elite clubs (Tjalve, Vidar and BUL) in the Oslo area. The runners were invited by a formal mail sent from the Norwegian Athletics Federation. Furthermore, the leader of each club received a mail or phone call encouraging them to inform their athletes of the project. Moreover, phone calls were made to athletes to get enough participants. A detailed project description and informed consent was sent to all athletes who were interested. Eleven female- and 10 male middle- and long distance runners responded positively, and were enrolled in the study. Eight of the 22 runners were classified as long distance runners (preferred distance > 3000m). Thirteen runners were classified as middle distance runners (preferred distance 800m – 3000m).

4.5. General in- and exclusion criteria

The athletes needed to be between 18 and 35 years old. One exception was made due to the high international level of the athlete. The athlete in question was 17.4 years old at the time of the DEXA scan, however, he/she was 18 at the time of the study enrollment. Moreover, athletes needed to compete at a national level within their respective sport. Athletes were excluded if they had history of tobacco use (smoking), diseases such as Crohn's disease, kidney diseases, rheumatic diseases or bone marrow diseases, which are known to affect the skeletons bone mineral density. Furthermore, athletes were excluded if they had known family historic with early onset osteoporosis (prior to the age of 50).

4.6. DXA measurement and anthropometrical measures

Dual-energy X-ray absorptiometry (DXA) Lunar prodigy machine (GE Lunar Radiation Corp, madison, WI, USA, Software version 5.60) was used to measure fat free mass (FFM), fat mass (FM), BMI (kg/m^2) and BMD ($gCal/cm^2$). Measurements included total body, lumbar spine (L1-L4) and DP femur. Lumbar spine and DP femur was included as they are the most common sites for osteoporotic fractures (Leib et al., 2004). The equipment was calibrated each morning prior to the first measurement. Anthropometrical measurements such as height and weight was measured prior the DXA scan. Two scans where obtained using Hologic, Massachusetts (Discovery, S/N 83817). All measurements were performed in a fasting state before 10 am. Measurement of L1 was missing in two of the athletes. Thus, measurements of L2 – L4 were used when BMD in the lumbar spine was analyzed. L2 – L4 is considered standard measurements when evaluating osteoporosis (Tavakoli, Salamat & Tavakoli, 2015).

4.7. Questionnaire

A self-composed questionnaire regarding training, injuries, health and nutritional variables was administered to the subjects. The questionnaire was developed with guidance from experts within the fields of overuse injuries and nutrition.

Part two of the questionnaire was a standardized questionnaire used to determine Calcium (CA) intake per day. The questionnaire has been validated in osteoporotic patients and is considered an adequate tool in estimating if a subject has a daily CA intake above cut-off limits of 700 or 1000 mg/day (Macdonald et al., 2014). Cut-off in the present paper is 800 mg/day as recommended for male- and female athletes (Garthe & Helle, 2011).

4.8. Statistical analysis

Statistical analysis was performed by using Statistical Package of Social Sciences (SPSS) version 24, SAS (v.9.3). Demographical data are presented as mean and standard deviation if normally distributed, or median and interquartile range (IQR) if skewed. Student's t-test was applied on normally distributed data to compare means between two groups. If skewed data, a non-parametric test was used. Binary univariate and multivariate logistical regression was applied to determine predictors of low BMD. Results of the regression analysis is presented in tables with odds ratio (OR) and 95 % confidence interval (CI). P – value = 0,05 is used for all measurements to determine statistical significance.

4.9. Ethical considerations

The study was reviewed and approved by The Regional Ethics Committee. Each subject gave informed consent prior to enrolment in the study. Data collection was conducted in agreement with The Declaration of Helsinki – Ethical principles for medical research involving human subjects (World Medical Association, 2013).

The athletes were not given their own result immediately, due to the possible health consequences of the results. The results were interpreted and communicated to each athlete by a team physician or a nutritionist at Norwegian Olympic Training Centre. Furthermore, if low BMD where found, athletes were given recommendations for further follow-up.

5.0. Results

5.1. Anthropometrical subject characteristics

Table 2: Subject characteristics with anthropometric measures for runners and cyclists. Values are presented as means and standard deviation (SD) (n=40).

Measure	Runners (n=21)	Cyclists (n=19)
Age (year)	25.4 (4.4)	24,0 (4.0)
Height (cm)	178.1 (11.8)	177.1 (7.6)
Weight (kg)	65.4 (10.3)	70.1 (10.0)
BMI (kg/m^2)	20.21 (1.14)**	21.88 (1.72)**
FMM (%)	81 (4.4)	83 (5.4)
FM (%)	15 (4.5)	14 (5.3)

* = significant $p < 0.05$, ** = significant $p < 0.01$. BMI = body mass index, FMM = fat free mass, FM = fat mass

Whole group analyses demonstrated that runners had significantly lower BMI ($p < 0.001$) compared to cyclists. There were no differences observed for the other anthropometrical measurements between the groups. Age and FM % was not normally distributed for the cyclist's (median (Mdn) 23.3, interquartile range (IQR) 3.4 and Mdn 11.7, IQR 7.6), respectively. Moreover, weight in runners where not normally distributed (Mdn 64.0, IQR 17.9).

5.2. Sports and nutritional subject characteristics

Table 2: Subject characteristics with sport, injuries and calcium measures for female runners, female cyclists, male runners and male cyclists. Values are presented as means and standard deviation (SD) (n=38).

Measure	Runners (n=21)	Cyclists (n=17)[1]
Started competing (age)	17.8 (4.9)	15.8 (1.1)
Years competing	7.0 (3.4)	7.4 (3.0)
Training hours (year)	549 (170)**	909 (124)**
Calcium intake (mg/day)	1503 (674)	1324 (558)
Nr. of athletes training HRT last two years	5	16
CI acute fractures (%)	14*	59*
CI stress fractures (%)	48	12

[1]Two male cyclists did not respond to the questionnaire and where excluded from the present analysis. * = significant p < 0,05, ** = significant p < 0,01. CI = cumulative incidence, HRT = heavy resistance training.

Cyclists had significantly more training hours compared to runners ($p < 0.001$). Calcium intake was similar in both groups. Sixteen of 17 cyclists reported to perform heavy resistance training on lower extremities for minimum two consecutive months during the last two years. There was no difference in total fractures between cyclists and runners. However, there were differences in type of fractures. Cyclists had significantly higher cumulated incidence of acute fractures compared to runners ($p < 0.05$). Numerical differences were observed in the incidence of stress fractures, were runners had higher cumulative incidence of stress fractures

compared to cyclists. The difference was not considered statistically significant (p = 0.07). Started competing was not normally distributed for runners (median 16.0, IQR 8.5).

5.3. DEXA measurements

Table 3: Dual Energy X-Ray Absorptiometry measurements expressed as bone mineral density (g/cm^2) for female runners, female cyclists, male runners and male cyclists. Values are presented as means and standard deviation (SD) (n=40).

Measure	Runners (n=21)	Cyclists (n=19)
L-spine L2-L4 (g/cm²)	1.267 (0.094)*[1]	1.166 (0.144)*[1]
D.P. femur (g/cm²)	1.157 (0.124)*[1]	1.052 (0.123)*[1]
Total BMD (g/cm²)	1.283 (0.090)**	1.195 (0.102)**
Nr. of athletes with low BMD	0	10
Nr. of athletes with Z-score < -2	0	1

* = significant p=0.05, ** = significant p = 0.01. [1] = missing data form one subject. L-Spine = lumbar spine, D.P. femur = dual proximal femur, BMD = bone mineral density, CI = cumulative incidence.

Runners had significantly higher BMD ($p < 0.05$) for all measured sites. The largest difference was observed in Total BMD ($p < 0.01$). Low BMD, classified as having an age matched Z-score of -1 or less for minimum one of the measured sites, was found in 10 out of 19 cyclists (53 %) (Nattiv et al., 2007). None of the runners had an age matched Z-score of -1 or less. The lumbar spine was the most prevalent site of low BMD. 7 of 10 cyclists had low

BMD in the lumbar spine, 4 of 10 cyclists had low BMD in DP femur and 1 cyclist had low total BMD.

5.4. Associations of low BMD

Training hours last year and cumulative incidence of acute fractures during an athlete's sports carrier was identified as significant predictors of low BMD. However, this relationship became insignificant when "sport" was entered as a covariate in multivariate regression analysis. Sub-group analysis reviled no significant relationship between secondary amenorrhea and BMD in female athletes.

Table 4: Univariate binary logistic analysis of factors for low BMD for all athletes (cyclists n=19, runners n=21).

Factor	OR	95 % C.I. for EXP (B)	P
Age (year)	1.068	(0.907 – 1.257)	0.429
Height (cm)	0.985	(0.917 – 1.058)	0.683
Weight (kg)	0.998	(0.932 – 1.069)	0.956
BMI (kg/m^2)	1.124	(0.740 – 1.706)	0.583
FMM (%)	1.166	(0.984 – 1.381)	0.077
FM (%)	0.903	(0.767 – 1.064)	0.223
Started competing (age)[1]	1.020	(0.876 – 1.188	0.795
Years competing[1]	0.940	(0.745 – 1.186)	0.603
Training hours (year)[1]	1.006	(1.001 – 1.011)	0.011*
Ca (mg/day)[1]	0.999	(0.998 – 1.000)	0.205
Nr. of athletes training HRT last two years	0.134	(0.015 – 1.212)	0.074
CI acute fractures (%)[1]	7.000	(1.413 – 34.682)	0.017*
CI stress fractures (%)[1]	-	-	-

Values are presented as means ± standard deviation (SD) when normal distributed or median and range when non-normal distributed. Cyclists are compared to runners. * = significant $p < 0.05$, ** = significant $p < 0.01$. BMI = body mass index, FMM = fat free mass, FM = fat mass, OR = Odds ratio, CI = cumulative incidence, Ca = calcium, [1] = missing data form two subjects

5.5. Anthropometrical measurements categorized by sport and gender

Table 1: Subject characteristics with anthropometric measures for female runners, female cyclists, male runners and male cyclists. Values are presented as means and standard deviation (SD) (n=40).

Measure	Female runners (n=11)	Female cyclists (n=7)	Male runners (n=10)	Male cyclists (n=12)
Age (year)	25.7 (4,1)	23.9 (4.7)	25.1 (4.8)	24.0 (3.8)
Height (cm)	169.2 (6.9)	170.5 (6.6)	187.9 (7.3)*****	181.0 (5.2)*****
Weight (kg)	57.2 (3.9)	61.7 (8.0)	74.5 (7.6)	75.0 (7.6)
BMI (kg/m^2)	19.74 (1.01)	20.97 (1.82)	20.74 (1.09)******	22.41 (1.49)******
FMM (%)	77.5 (3.0)*	79.2 (5.7)	85.0 (2.2)	85.6 (3.7)
FM (%)	18.6 (2.8)	17.7 (6.1)	11.4 (2.4)	11.3 (3.1)

Female cyclists are compared to female runners and male cyclists with male runners. * = significant $p < 0.05$, ** = significant $p < 0.01$. BMI = body mass index, FMM = fat free mass, FM = fat mass

Female cyclists were compared with female runners. There were no significant differences in anthropometrical measurements between female runners and female cyclists. Male cyclists were compared to male runners. Male runners were significantly taller ($p < 0.05$) and had a significantly lower BMI ($p < 0.01$) compared to male cyclists. Age and FM % was not normally distributed for cyclists (Mdn 22.8, IQR 3.1 and 10.1, IQR 5.7), respectively. Moreover, weight was not normally distributed for runners (Mdn 73.3, IQR 5.7).

5.6. Sports and nutritional subject characteristics categorized by sport and gender.

Table 2: Subject characteristics with sport, injuries and calcium measures for female runners, female cyclists, male runners and male cyclists. Values are presented as means and standard deviation (SD) (n=40).

Measure	Female runners (n=11)	Female cyclists (n=7)	Male runners (n=10)	Male cyclists (n=10)[1]
St. competing (age)	18.6 (5.0)	16.6 (5.8)	16.9 (4.9)	15.2 (3.5)
Years competing	6.1 (4.0)	6.4 (2.7)	8.0 (2.4)	8.0 (3.2)
Training hrs. (year)	505 (190)**	908 (126)**	597 (137)**	909 (129)**
Ca (mg/day)	1420 (768)	1251 (608)	1594 (580)	1376 (546)
Nr. of athletes training HRT last two years	4	7	1	9
CI acute fractures (%)	9	71	20	50
CI stress fractures (%)	55	0	40	20
CI Secondary amenorrhea (%)	64	71	-	-

[1]Two male cyclists did not respond to the questionnaire and where excluded from the present analysis. * = significant $p < 0.05$, ** = significant $p < 0.01$. Ca = calcium, HRT = heavy resistance training, CI = cumulative incidence.

Female cyclists had significantly more training hours last year compared to female runners ($p < 0.01$). Male cyclists had significantly more training last year compared to male runners ($p < 0.01$). There were numerical differences in cumulative incidence of fractures between cyclists and runners. Runners had highest prevalence of stress fractures, whereas cyclists had highest prevalence of acute fractures. No differences were observed in the daily calcium intake between either group. The prevalence of secondary amenorrhea was 64 % and 71 % in female runners and female cyclists, respectively. The difference was not considered significant. Training hours was not normally distributed for male runners (Mdn 545, IQR 182). Moreover, years competing was not normally distributed for female cyclists (Mdn 6.4, IQR 2.7).

5.7. DEXA measurements categorized by sport and gender

No significant differences were observed in BMD for all measured sites between female cyclists and female runners. However, 4 of 7 female cyclists (57.14 %) where classified as having low BMD (Z-score < -1) in minimum one of the measured sites. None of the female runners where classified as having osteopenia. Male cyclists had significantly lower BMD compared to male runners for all measured sites ($p < 0.05$). The largest difference was observed in total BMD ($p < 0.01$). Dual proximal femur was not normally distributed for male runners (Mdn 1.184, IQR 0.185). Furthermore, total BMD for female cyclists was not normally distributed (Mdn 1.09, IQR .076).

Table 2: Dual Energy X-Ray Absorptiometry measurements expressed as bone mineral density (g/cm^2) for female runners, female cyclists, male runners and male cyclists. Values are presented as means and standard deviation (SD) (n=40).

Measure	Female runners (n=11)	Female cyclists (n=7)	Male runners (n=10)	Male cyclists (n=12)
L-spine L2-L4 (g/cm²)	1.238 (0.084)	1.140 (0.053)	1.300 (0.097)*	1.182 (0.045)[1]*
D.P. femur (g/cm²)	1.081 (0.122)	1.009 (0.493)	1.241(0.117)**	1.080 (0.035)[1]**
Total BMD (g/cm²)	1.218 (0.019)	1.132 (0.083)	1.356 (0.017)*	1.230 (0.031)*
Nr. of athletes with low BMD	0	4	0	6

Female cyclists are compared to female runners and male cyclists with male runners. [1] = missing data form one subject * = significant p=0.05, ** = significant p = 0.01. L-spine = lumbar spine, D.P. femur = dual proximal femur, BMD = bone mineral density

6.0. Discussion

Our main finding was that, - compared to runners, cyclists had significantly lower BMD for all measured sites. This is consistent with previous research (Rector, Rogers, Rubel & Hinton, 2008; Rector, Rogers, Ruebel, Widzer & Hinton, 2009; Duncan et al., 2002; Beshgetoor, Nichols & Rego, 2000). Furthermore, 10 out of 19 cyclists were classified as having low BMD, despite that all, but one rider, reported to train heavy resistance training on the lower extremities. Low BMD was not confined to females. One male rider was classified as osteoporotic, with secondary clinical risk factors for fracture (Nattiv, 2007). Conversely, no runners had low BMD for any of the measured sites, despite their high cumulative incidence of stress fractures (48 percent), which has been associated with low BMD in previous research (Burrows & Bird, 2000; Lauder, Dixit, Pezzin, Williams, Cambell & Davies, 2000; Chen, Tenforde & Fredericson, 2013).

6.1. Cyclists had high prevalence of low BMD, despite reporting to train heavy resistance training

The novelty of the present study is that the cyclists had high prevalence of low BMD, despite reporting to train heavy resistance training on the lower extremities. Strength training and its osteogenic effect has been well documented in previous research (Giangregorio, 2014; Hinton, Nigh & Thyfault, 2017; Martyn-St James & Carrol, 2006). All but, one rider reported not to have been training heavy resistance training on the lower extremities for minimum two consecutive months the last two years. By contrast, only five of the runners reported to perform heavy resistance training. No significant associations between BMD and resistance training were demonstrated when the whole population was analyzed or in sub-group analyses.

There is an increasing body of evidence demonstrating the beneficial effect of heavy resistance training on cycling economy and performance in cyclists (Sunde et al., 2010; Vikmoen et al., 2015). This has caused a recent change in training structure in many elite endurance athletes, and strength training has gained popularity in cyclists. For example, Vikmoen and colleagues observed a change in muscle fibers from type IIAX-IIX, toward IIA. IIAX and IIX are fast twitch muscle fibers, which cannot be utilized efficiently during endurance training. By contrast, IIA fibers can contribute during prolonged exercise. Thus, a shift in muscle fibers would most likely improve performance.

Bones are slow adapting and it is possible the cyclists in the present study have not been performing strength training for enough time to improve bone strength. Most studies documenting beneficial effect of resistance training on bone mass are longitudinal studies, lasting for minimum 7 – 12 months, with 2-3 sessions per week (Layne & Nelson, 1999). Moreover, the modality of the resistance training may also play an important role. Hawkins et al., (1999) examined the difference between concentric and eccentric resistance training on 20 women. They showed that only eccentric exercise resulted in BMD gain, while concentric resistance training caused no differences in BMD. Usually, strength training in cyclists is performed during off-season, which is the winter month October-January. Thus, three months of strength training might not be sufficient to elicit the bone modelling process. Furthermore, cyclists typically focus primarily on concentric strength training in order to maximize sport-specificity.

The high prevalence of low BMD in cyclists demonstrated in the present study is consisted with previous research. Rector et al., (2007) showed that elite male cyclists were 7 times more likely to have low BMD, compared to runners. Studies involving professional cyclists have

shown that as much as two third of professional cyclists may have abnormal BMD (Medelli, et al., 2009; Campion et al., 2010). Moreover, two systematic reviews conducted in 2011 and 2012 concluded that road cycling does not appear to stimulate osteogenesis, and that professional cyclists may be at risk of developing low BMD (Olmedillas, Gonzalez-Aguero, Moreno, Casajus & Vicente-Rodriguez, 2012; Nagle & Brooks, 2011). Few longitudinal studies have been conducted in cyclists. However, both studies in males and females show a significant decline in BMD during a season of competitive cycling. Sherk et al. (2014) followed 14 female elite cyclists during 1 year of competitive cycling. The cyclists had an average decline of BMD in the hip and spine of 1.4 % and 1.1 %, respectively. This is similar to longitudinal studies in male cyclists. Barry & Kohrt (2008) showed a decline of 1.5 % and 1.0 % in the DP femur and lumbar spine in 14 male cyclists during a competitive season. The loss of BMD demonstrated in both female and male riders corresponds to the accelerated loss of bone mass observed in post-menopausal females (Beshgetoor et al., 2000).

6.2. Training hours and acute fractures was associated with low BMD

Number of training hours and acute fractures were significantly associated with low BMD in whole group analysis. However, both factors become insignificant when controlling for the type of sport. It is reasonable to assume that cyclists can accumulate more training hours compared to runners, due to its non-weight-bearing and low impact characteristics. Furthermore, road cycling is a sport that involves high risk of accidents, which increases the risk of fractures. Thus, it is likely that cyclists would have higher cumulative incidence of fractures compared to runners, regardless of BMD.

6.3. Differences in BMD is most likely attributed to differences in gravitational forces

The difference in BMD observed in the present study appears to be attributed to the difference in mechanical strain put on the skeleton by gravitational forces. Previous research has examined the differences in force development associated with running and cycling. Woodward & Cunningham (1993) measured acceleration in the ankle in various activities. Running had a rate of change of acceleration of 2.14 g/s. By contrast, cycling only resulted in rate of change of acceleration of 0.23 g/s. According to the mechanostat theory, the force from pedaling would not create enough external force to elicit the bone remodeling process (Hattner et al., 1965). Conversely, running would cause bone remodeling as the forces produced exceeds 1 g, which is thought to be the minimum strain needed to maintain bone homeostasis.

Running, depended on speed, could cause impact forces greater than 3g which is thought elicit bone modelling (Weyand, Sternlight, Bellizzi & Wright, 2000). In the present study 8 of the 21 runners were long distance runners (preferred distance > 3000m). The remaining runners were classified as middle-distance runners (preferred distance 800 – 3000m). Sprint training is considered more important in middle-distance runners, as the outcome of a race is more dependent on the sprint qualities of the runner. Thus, it is likely that they perform a considerable portion of their training at a speed that would stimulate bone formation, however, this was not examined as the low number of subjects in this study prevented subgroup analyses.

6.4. BMI and FMM

An interesting observation in the present study is that cyclists had significantly greater BMI, compared to runners. Furthermore, cyclists had greater FFM, however this was not statistically significant. No association was found between BMI and BMD or FFM and BMD. Height and weight, which are the two components of BMI, has been shown to be positively correlated with BMD in previous studies (Carter, Bouxsein & Marcus, 1992). Greater BMI creates greater mechanical strain on the skeleton during physical activity. This contributes to osteoblastic activity and increased bone modelling. More recent studies have suggested that FFM play a more pivotal role in predicting BMD, compared to BMI, and that FM is associated with lower BMD (Specker, Wey & Smith, 2010). FFM was greater in cyclists and they had lower FM, compared to runners. FFM is associated with increased internal strain during muscle contraction, which has shown to elicit bone formation (Babtista et al., 2012). As no association between BMD and BMI or BMD and FFM was observed, it is likely that type of sport trumps the possible positive affect associated with these factors.

6.5. Lumbar spine was the most affected site

The prevalence of low BMD in the present study was site specific. In particular, the lumbar spine was exposed. Seven cyclists had low BMD in the lumbar spine and four had low BMD in the DP femur. By contrast, only one rider had low total body BMD. Previous research has shown that both the lumbar spine and DP femur are risk areas of low BMD in cyclists (Nagle & Brooks, 2011; Beshgetoor et al., 2000; Rector et al., 2008).

There is currently limited research explaining why the spine seems to be especially exposed in cyclists. On theory is the sitting position during training and racing. Cyclists are partially

supported by the gluteus and the hands, which result in minimal strain on the spine during training. By contrast, the internal strain put on the DP femur during muscle contraction has been hypothesized in previous research to elicit bone remodeling process, thus, preserving bone mass (Barry & Kohrt, 2007; Olmedillas, 2011).

Another plausible explanation to why the spine would be most exposed, is the type of bone associated with the different sites. Trabecular bone, which is mainly found in the spine has a higher metabolism compared to cortical bone, which is the main constituent of the skeleton, including DP femur. Thus, it could be that trabecular bone responds to loading and unloading earlier than cortical bone. Trabecular bone has been shown to start declining earlier than cortical bone in young adults, especially in inactive individuals (Specker et al., 2010). Although elite cyclists can hardly be considered inactive, cycling might not put hard enough strain on the spine to elicit the bone remodeling process.

Not all previous studies have shown that the lumbar spine is the most affected site. Campion et al., (2010) showed that low BMD was most prevalent in the femoral neck (DP femur). Similarly, Sherk et al., (2014), showed that the loss of BMD was more substantial in the femoral neck, compared to lumbar spine over the course of 1 year of training and competition in female elite cyclists. Future research is needed to determine why the lumbar spine and DP femur seems to be especially exposed in cyclists. Nutritional and hormonal components are likely to play an important role.

6.6. Recovery phases

For all professional athletes, restitution is a key component of optimal performance. In professional cycling, the importance of recovery is well known and sometimes taken to extreme levels. Chris Carmichael, former professional cyclist and personal coach to Lance Armstrong is quoted on an adage known in the cycling milieu; “Why stand when you can sit, why sit when you can lie down?” (Chris Carmichael, 2016). In the book “The Secret Race”, former professional cyclist Tylear Hamilton tells how Bernard Hinault (5 times Tour de France winner) hated stairs so much that he had his soigneurs carry him up the stairs to his hotel room, to avoid unnecessary energy use (Coyle & Hamilton, 2012). Regardless of the truth to this rumour, it does provide an insight into the cultural beliefs and attitudes in the sport. The extreme focus on rest and recovery could remind of bedrest studies. Garland et al., (1992) followed 25 acutely injured spinal cord patients (males under 40 years) for 16 months. The patients lost 22 % of their total bone mass during the first three months. Although not directly comparable, it is reasonable to assume that professional cyclists spend most their time resting when they are not training. Thus, if the skeleton does not get mechanical stimulus from cycling, it is unlikely that it happens at all.

6.7. Calcium and Vitamin D

There was no relationship between calcium intake an BMD in the present study. Both runners and cyclists were found to have an adequate calcium intake (cyclists = 1324mg, runners = 1503mg). This is consistent with previous research conducted on Norwegian athletes that demonstrated that both male and female athletes had an adequate calcium intake (Helle, Bjerkan, Holm & Trygg, 2008). Previous research in cyclists has not been able to demonstrate a significant relationship between calcium intake and BMD (Beshgetoor et al., 2000; Barry & Kohrt, 2008; Medelli et al., 2009).

By contrast, Vitamin D deficiency is a common problem in endurance athletes, and has been linked to stress-fractures, muscle weakness, osteoporosis, depression and fatigue (Lappe et al., 2008). Vitamin D status was not examined in the present study, however, previous research has shown that a large percent (37 – 79 percent) of elite athletes do not meet the general recommendations of Vitamin D intake (Willis et al., 2008; Helle et al., 2008). Both endurance running and cycling are considered out-door sports, which is associated with higher levels of Vitamin D. However, based on high prevalence of sub-optimal Vitamin D status from previous research, it is likely that some of the athletes in the present study suffer from Vitamin D deficiency.

6.8. The female athlete triad and RED-S

The prevalence of the female athlete triad was not examined extensively in the present study. However, the prevalence of secondary amenorrhea was examined. Sixty-one percent of the female athletes reported prevalence of secondary amenorrhea. Sub-group analysis produced no significant relationship between secondary amenorrhea and prevalence of low BMD in the present study. This is surprising as previous research has shown secondary amenorrhea to be an important predictor of low BMD in athletes (Larsen & Hansen, 1998; Haenggi, Casez, Birkhaeuser, Lippuner, Jaeger et al., 1995).

Results from previous studies regarding the prevalence of the female triad vary depending on the criteria being used. Torstveit & Sundgot-Borgen (2005) reported that 4.3 percent of athletes suffer from all three components of the triad. Conversely, when assessing only one component, the prevalence is much higher. Reports regarding clinical eating disorder in young female athletes vary from 16-47 percent (Beals, Brey & Gonyou, 1999; Sundgot-

Borgen & Torstveit, 2004). Furthermore, incidences of low BMD have been reported to be 20 – 62 percent (Khan et al., 2002).

Energy deficits, eating disorders and low BMD is not limited to female athletes. Sundgot-Borgen & Torstveit (2004) reported that 8 % of Norwegian male athletes suffered from eating disorders. Moreover, there are reports of 63 % of professional male cyclists being diagnosed with osteopenia (Smathers, Bemben, M.G & Bemben, D.A., 2009). The high percentage is in alignment with the findings of the present study. Due to alarming reports regarding the potential triad problematic, an alternative terminology, RED-S has been proposed, in part to acknowledge male athletes. The syndrome involves energy deficit as the main component (Mountjoy et al., 2014).

Energy deficit is associated with low bone mass in endurance athletes (DeSouza et al., 2008). The most important performance measurement in cycling is power-to-weight ratio, or watt per kilogram. Thus, lower weight is thought to increase cycling performance, if the power is sustained. Tyler Hamilton reported in his book, that it was much easier to reduce weight than increase power (Coyle & Hamilton, 2012). This has been shown in research as well, where there are reports of cyclists trying to enhance their power-to-weight ratio at the expense of energy intake (Zanker & Cooke, 2003; Nichols, Palmer & Levy, 2003). Furthermore, due to prolonged exercise, cyclists may be at risk of having sub-optimal energy intake during training.

Weight loss has been associated with substantial bone loss in previous research. Salamone et al., (1999) followed 236 healthy premenopausal women over 18 months during a weight loss

program. The females who lost weight lost bone mass twice as fast as those who remained weight stable. Thus, staying in an energy deficit state for long periods may lead to increased bone resorption in athletes.

6.9. Hormonal disturbances associated with RED-S

Energy deficits are associated with numerous hormonal disturbances. Recent research in professional cyclists have shown that basal testosterone levels decreased by 15 percent during 12 weeks of intensive endurance training. The same relationship was demonstrated during Tour of Spain where a decrease in testosterone levels was observed after the three weeks of racing (Lucia et al., 2001). Similarly, runners have displayed low levels of testosterone during prolonged exercise (Wheeler, Singh, Pierce, Epling & Cumming, 1991; Hackney, 1989). Wheeler et al., showed that testosterone in serum was decreased over 6 months of prolonged running. Testosterone is imperative for normal growth in males, however, the decrease of testosterone during prolonged and hard exercise and its relationship with bone health has not yet been established (Smathers et al., 2009).

Cortisol has been shown to increase in male cyclists and runners during exercise (Hoogeveen & Zonderland, 1996; Houmard et al., 1990). Small amounts of cortisol are necessary for normal bone development. However, large amounts of cortisol stimulate the expression of RANKL and decreases the expression of OPG, which causes bone resorption (Canalis & Delany, 2002). Moreover, estrogen, which is an important regulator of bone mass in both females and males, has been shown to decrease as well (Vaananen & Hakonen, 1996). Estrogen depletion is commonly seen with energy depletion in athletes (DeSouza et al. 2008). In males, studies have shown that estrogen helps maintaining trabecular bone in the spine

(Morishima, Grumbach, Simpson, Fisher & Qin, 1995). In women, estrogen plays an even more important role, and estrogen depletion is the main cause of the accelerated bone loss observed in post-menopausal females.

The hormonal changes discussed above are typical for both runners and cyclists. Hormonal status was not investigated in the present study. Thus, it is impossible to conclude whether hormonal depletion was present or not, or if there were significant differences between the groups.

6.10. Caffeine

There is currently little research conducted on caffeine and its effect on bone health in endurance athletes. Previous research has shown that excessive caffeine intake (> 300mg) could be harmful for the skeleton (Rapuri, Gallagher, Kinayamu & Ryschon, 2001; Cooper et al., 1992.) In professional cycling, there are reports of excessive use of caffeine pills in combination with analgesic drugs to enhance performance, which could potentially be harmful for the skeleton (Procycling, 2012). Interestingly, a recent study conducted on rodents showed that orally Ibuprofen treatment, decreased bone loss from high repetitive, high force loading (Jain et al., 2014). Whether this is comparable to humans performing high repetitive, low force loading is unknown. In any case, athletes with low bone mass should be made aware of the possible risk related to excessive use of caffeine pills and analgesics.

6.11. Methodological concerns

The present study is a cross-sectional study. This implies that both exposure (independent variables) and outcome (BMD) were measured at one point in time. This makes it impossible to determine causality. Furthermore, it does not allow to follow fluctuations in BMD throughout the year.

6.11.1 The sample and sample size

The runners were recruited by convenient sampling. Runners from the major clubs in- and around Oslo was approach, as well as the runners from the national and recruit team. The recruit team consist of runners currently most likely to become a part of the national team. Anyone who met the inclusion criteria were enrolled in the study. Convenient sample has its advantages in being a fast, inexpensive and the athletes enrolled were motivated to participate. However, it is important to be aware that these subjects may share certain unknown characteristics. Thus, it is not possible to draw general conclusions for the whole population. By contrast, the cyclists were all a part of a continental team or the national team, and they underwent a yearly DEXA examination. Thus, the DEXA scan was mandatory for all athletes. All cyclists except for 3 agreed to participate, which makes the studied sample more generalizable compared to runners. However, this creates a systematic bias between the two groups, as one group had better methodological standard than the other.

The present study involved 40 athletes consisting of 11 female runners, 10 female runners, 12 male cyclists, 7 female cyclists. This gave the study limited statistical power. In particular, we were unable to perform sub-group analyses due to the small sample size. Thus, it is possible that some associations between independent variables and BMD were overlooked. In this

study, we did not identify any runners with low BMD. However, it would be erroneous to conclude that elite runners are not at risk of having low BMD, only that their risk appears to be far less than among cyclists. A study including larger number of subjects would be able to estimate the actual difference in risk between groups more accurately.

A strength of the present study is the high level of the athletes. All cyclists in the present study were competing at a professional level at the time of the measurement. Thus, the sample of cyclists in the present study is highly homogenous. It is likely that they share similar characteristics of professional cyclists in other countries, making the results of the present study generalizable to the entire population. Furthermore, 12 of the 22 runners included were either on the national team or the recruit team. The remaining runners were competing in elite clubs in Oslo. Moreover, 16 of 22 runners reported to have finished top 3 in national competitions during their carrier. Although elite athletes are more challenging to recruit then lower-level/recreational athletes, we chose to limit our inclusion to a homogenous group of high level athletes, at the expense of a larger sample size and statistical limitations.

6.11.2. BMD measurement

All but two DEXA scans were performed on the same equipment (Lunar Prodigy). The remaining two were measured on Hologic. The results were cross calibrated as recommended, however, the standardized equation used for cross calibration is dated and may not be as applicable on new machines using new technology. Recent research which compared Hologic to Lunar Prodigy showed that there was only a difference of 1 % in the DP femur between the two manufactures. However, the difference was significant at the lumbar spine (Fan, Lu, Genant, Fuerst & Shepherd, 2010). Thus, it could be that the two DEXA scans obtain by

Hologic are not directly comparable the scans conducted on Lunar Prodigy machines, despite cross-calibration.

The DEXA scans were obtained by using several technicians. In the runners group, all but three scans were conducted by the same technician. However, DEXA measurements of cyclists were conducted by several technicians-, at different institutes. This may introduce a systematic bias in the results, as positioning of the subject is critical for an accurate DEXA measurement, as well as the technician's analysis of the result (Lewiecki & Lane, 2008). Thus, the scans are most likely to be more consistent and reliable in the runners group, which had fewer technicians, compared to the cyclist group.

The timing of DEXA measurement is of importance in athletes. As pervious research, has demonstrated, BMD declines during the cycling season (Shrek et al., 2014; Barry & Kohrt, 2008). In the present study, 35 of 40 scans were obtained during the period February – April. Thus, it could be that the remaining 5 interfered with the result. Furthermore, 4 scans in the present study were more than one year old. Thus, it is possible that the information acquired by the questionnaire in these subjects does not accurately reflect the measured BMD.

6.11.3. The questionnaire

As mentioned, the questionnaire was not administered at the same time as the DEXA scan for all patients, which represents a limitation. Furthermore, a general limitation of questionnaires is recall bias. The questionnaire in the present study required athletes to remember training and health factors from the last year, as well as what they had eaten and drunk during the past

week. Two of 40 athletes did not respond to the questionnaire, which could have interfered with statistical limitations.

A part of the questionnaire was a standardized short calcium questionnaire (CaQ) (Macdonald et al., 2014). This has been validated in osteoporotic patients. However, it was not validated in Norwegian language and was therefore administered in its original English format. Thus, it is possible that the result could suffer from language bias. However, Norway was top 4 in a recent international ranking of English skills (EF English Proficiency Index, 2017), which makes language bias less likely. Furthermore, the questionnaire has not been validated in athletes. Athletes gain a substantial amount of their calcium intake through sports drinks, bars and gels which were items not included in the questionnaire. Nevertheless, all athletes in the present study reported an adequate calcium intake, despite this limitation. Thus, it is highly likely that the athletes of the present study had an adequate calcium intake.

Several other factors, which could influence bone health are not evaluated in the present study. This includes hormonal factors, Vitamin status, timing of calcium intake and caffeine intake. Future studies need to address these factors to fully understand how cycling affects BMD.

6.12. Practical implications

The findings in the present study underlines what was already known, that cyclists are at risk of developing low BMD. The novelty is however, that riders report to have been performing heavy resistance training and still display low bone mass. However, due to the study design, it

is not possible to assess whether the strength training performed has had a positive effect on the skeleton.

Previous studies have documented incidence of low BMD in adolescent cyclists comparable to the findings in adults (Olmedillas et al., 2011; Duncan et al., 2002). This is of particular importance as BMD is highly responsive to mechanical loading during puberty. A previous systematic review demonstrated that the skeleton seems to be most sensitive to skeletal loading between at age 11.5 – 13.5 in girls and 13 – 15 in boys (MacKelvie, Khan, McKay, & Sanborn, 2002). During this period 26 % of total BMD is gained. This is equal to the amount lost after menopause. Cycling could impair the possibility of achieving and optimal PBM (Olmedillas et al., 2011). This could have long term consequences for skeletal health in adulthood. Thus, children competing in cycling should perform varied training. With respect to long- distance and marathon running, there are very few children who compete in this sports. Thus, it is unlikely that they are affected by the same problem.

The observed low BMD in cyclist's cause grounds for interventions. However, this is challenging as high-level athletes are unlikely to be positive to adding a training to their already full schedule. In particular, if there are no obvious performance benefits. The risk of fracture due to falling is high in cycling (Cerynik, Roshon, Abzug, Harding & Tom, 2009). Research in frail elderly females have shown that a fall to the side increases the risk of fracture by 5.7 times, compared to falls in any other direction (Greenspan et al., 1998). Although frail women and professional cyclists are hardly comparable, they do share some common characteristics with high prevalence of low BMD in the spine and hip region. Falls to the side is very common in cycling due to slippery surface. Thus, making athletes see the

possible benefit of reduced number of days injured, might make the recruitment procedure easier.

Previous research has demonstrated that as little as 10 min three times/week is enough to significantly improve bone health in young children. This is probably a more responsive group than the athletes in the present study. However, many of these athletes are only at the beginning of their twenties and the skeleton is still adaptable to mechanical loading. Martin & Burr (1989) showed that high strain together with high strain rate is more effective for maximal adaptive bone response. Uneven strain distribution seems to be more important for osteogenesis than strain repetition or strain magnitudes. Rubin & Laynon (1984) showed that 36 consecutive loading cycles was sufficient to maximise osteogenesis. Further loading did not seem to elicit the bone modelling process further. Thus, 30 minutes of jumping exercise, might be all that is required to improve bone health in cyclists.

7.0. Conclusion

Elite Norwegian cyclists had lower BMD compared to runners, and a large portion were classified as having low BMD as per ASCM criteria, despite that cyclists reported to perform heavy resistance training. Interventions to increase BMD in cyclists are necessary.

8.0. References

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Appendixes

1. Written informed consent (in Norwegian)
2. Approval from the Regional Medical Ethics committee (REK) (in Norwegian)
3. Questionnaire (in English and Norwegian)

Forespørsel om deltakelse i forskningsprosjekt

Beinhelsestatus i norsk utholdenhetsidrett

Dette skrivet er rettet til alle potensielle forsøkspersoner. Jeg ber om din deltakelse i prosjektet; *«Beinhelsestatus hos norske kvinnelige- og mannlige elitesyklister og langdistanseutøvere»*, så fremt du oppfyller kriteriene for deltakelse. Du må være i alderen 18-35 år, mann, du skal ha drevet med landeveissykling på elitenivå (norgescup eller internasjonalt) sammenhengende de siste 4 årene. Du skal i denne perioden ikke ha konkurrert i vektbærende idretter (eks. ballsport, løp). Du skal ikke ha røkt, eller hatt sykdom (cøliaki, ulcerøs kolitt og Crohns sykdom, nyresykdommer, revmatiske sykdommer, beinmargssykdommer) som potensielt kan påvirke skjelettets beinmineraltetthet. Du skal heller ikke ha kjent familiehistorikk med tidlig (før fylte 50 år) benskjørhet (osteoporose).

Bakgrunn og hensikt

Osteoporose eller beinskjørhet har blitt et alvorlig helseproblem i dagens samfunn. I Norge antas det at 50 prosent av alle kvinner og 25 prosent av alle menn i 50 års alderen vil rammes av et osteoporosebrudd en eller annen gang senere i livet. Oppnåelse av en høy Peek Bone Mass (PBM) i ung alder har vist seg å være en viktig faktor for beinmasse senere i livet. Osteoporose er forbundet med inaktivitet. Det er funnet en klar sammenheng mellom vektbærende trening og en høy beinmineraltetthet eller Bone Mineral Density (BMD). Det samme positive forholdet har ikke blitt demonstrert i ikke-vektbærende idretter, slik som sykling og svømming. Flere studier har sett på sammenhengen mellom sykling og BMD. Funnene er motstridende, men viser en trend mot lavere BMD hos både kvinnelige- og mannlige elitesyklister sammenliknet med kontrollgrupper eller utøvere som konkurrerer i vektbærende idretter.

Hensikten med denne studien er å undersøke om norske kvinnelige- og mannlige elitesyklister har en suboptimal beinstatus, sammenliknet med kvinnelige- og mannlige langdistanseløpere.

Hva innebærer prosjektet?

På testdagen vil det bli gjennomført en DXA undersøkelse. Du vil i tillegg få tilsendt et spørreskjema på mail som du kan svare på elektronisk. Spørreskjemaet vil ikke oppta mer enn 20 minutter av din tid.

DXA (Dual-energy X-ray absorptiometry), Lunar iDXA

Dual-energy X-ray absorptiometry (DXA) er et spesialkonstruert røntgenapparat hvor beinmassen i kroppen måles. Selve undersøkelsen er helt smertefri og tar ca. 10 minutter. Undersøkelsen gjennomføres fastende. Som forsøksperson er du iført undertøy under selve undersøkelsen.

Egenkomponert spørreskjema vedrørende trening, kosthold og idrettsrelaterte skader

Et egenkomponert spørreskjema vedrørende trening og kosthold vil bli benyttet for å kartlegge nivå, treningsmengde, enkelte kostholds variabler og idrettsrelaterte skader.

Spørreskjema CaQ

For å kartlegge om du har et tilstrekkelig kalsiuminntak blir et kort spørreskjema (CaQ) benyttet. Skjemaet inneholder 21 av de mest vanlige kalsiumrike matvarene i et normalt kosthold.

Mulige fordeler og ulemper ved å delta som forsøksperson

Studien krever at du besvarer noen spørreskjemaer elektronisk. Dette vil oppta noe av din tid og oppmerksomhet.

Som deltager får du en god evaluering av din nåværende beinhelsestatus og kroppssammensetning. Testresultatene vil bli tolket av lege tilknyttet ditt lag, eller klinisk ernæringsfysiolog tilknyttet Olympiatoppen, før resultatene blir videreformidlet til hver enkelt utøver. Ved funn av lav fettprosent eller beinmineraltetthet vil utøverne få anbefaling av ernæringsfysiolog om hvor de kan følges opp videre.

Frivillig deltakelse

Det er frivillig å delta i prosjektet. Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Du kan når som helst, uten å oppgi noen grunn trekke ditt samtykke. Dette vil ikke få konsekvenser for ditt videre forhold til Norges Idrettshøgskole. Dersom du trekker deg fra prosjektet, kan du kreve å få slettet innsamlede prøver og opplysninger, med mindre opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner.

Hva skjer med informasjonen om deg?

Informasjonen som registreres om deg skal kun brukes slik som beskrevet i hensikten med studien. Du har rett til innsyn i hvilke opplysninger som er registrert om deg og rett til å få korrigert eventuelle feil i de opplysningene som er registrert.

Alle opplysningene vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjennende opplysninger. En kode knytter deg til dine opplysninger gjennom en navneliste.

Prosjektleder har ansvar for den daglige driften av forskningsprosjektet og at opplysninger om deg blir behandlet på en sikker måte. Informasjon om deg vil bli anonymisert eller slettet senest fem år etter prosjektslutt.

Samtykke

Hvis du har lest informasjonsskrivet og ønsker å være med som forsøksperson i prosjektet, ber vi deg undertegne «Samtykke om deltakelse» og returnere dette til kontaktpersonen oppgitt nedenfor. Du bekrefter samtidig at du har fått kopi av og lest denne informasjonen.

Det er frivillig å delta og du kan når som helst trekke deg fra prosjektet uten videre begrunnelse og uten at det vil få konsekvenser for ditt forhold til NIH. Alle data vil, som nevnt ovenfor, bli avidentifisert før de blir lagt inn i en database, og senere anonymisert.

Skulle du ønske og tilbakekalle samtykket om deltakelsen i studien, kan du kreve at innsamlende helse- og personopplysninger blir slettet eller utlevert.

Forsikring

Som forsøksperson vil du være forsikret gjennom NIH som er selvassurandør i kraft av å være en statlig, vitenskapelig høyskole.

Publisering

Resultatene vil i første omgang publiseres som en masteroppgave. Oppgaven vil være tilgjengelig på NIH sine nettsider. Det er mulig at resultatene senere vil bli publisert i en vitenskapelig artikkel.

Dersom du ønsker flere opplysninger kan du ta kontakt med Oddbjørn Klomsten Andersen på tlf: 93 26 45 44/oddbjorn_andersen@outlook.com

Med vennlig hilsen

Oddbjørn Klomsten Andersen

Samtykket om deltakelse

Jeg har gjort meg kjent med innholdet i infoskrivet «navn på prosjekt/oppgave» og ønsker å delta som forsøksperson i prosjektet.

Navn: ……

E-post: ……..

Tlf: ……

Dato: …./…. 2014 Sted……

…….

(signatur)

REK
REGIONALE KOMITEER FOR MEDISINSK OG HELSEFAGLIG FORSKNINGSETIKK

Region:	Saksbehandler:	Telefon:	Vår dato:	Vår referanse:
REK sør-øst	Mariann Glenna Davidsen	22845526	20.12.2016	2016/1976 REK sør-øst B
			Deres dato: 01.11.2016	Deres referanse:

Vår referanse må oppgis ved alle henvendelser

Trine Stensrud
Norges idrettshøgskole

2016/1976 Beinhelsestatus hos norske elite syklister og langdistanseløpere

Forskningsansvarlig: Norges idrettshøgskole **Prosjektleder:**
Trine Stensrud

Vi viser til søknad om forhåndsgodkjenning av ovennevnte forskningsprosjekt. Søknaden ble behandlet av Regional komité for medisinsk og helsefaglig forskningsetikk (REK sør-øst) i møtet 30.11.2016.
Vurderingen er gjort med hjemmel i helseforskningsloven (hfl.) § 10, jf. forskningsetikkloven § 4.

Prosjektleders prosjektbeskrivelse

"Syklister på toppnivå gjennomfører store treningsmengder i løpet av et år, men treningen er i all hovedsak ikke vektbærende. Det kan medføre redusert beinmasse og økt risiko for tidlig utvikling av osteoporose som kjennetegnes ved lav beinmineraltetthet (BMD) og økt risiko for beinbrudd. Hensikten med studien er å undersøke om norske elitesyklister har en suboptimal beinhelsestatus sammenlignet med norske elitelangdistanseløpere (begge kjønn). I tillegg vil vi undersøke om spesielle ryttertyper er mer utsatt enn andre for å utvikle suboptimale BMD verdier. Kroppssammensetning og BMD uttrykt som gCal/cm2 målt med "gullstandardmetoden"; Dual-energy X-ray absorptiometry (DXA) vil bli sammenlignet mellom syklister og langdistanseløpere med tilnærmet lik treningsmengde og kroppsvekt i en tverrsnittsstudie. Kalsiuminntak, treningshistorikk,restitusjonsforhold og menstruasjonshistorikk mm registreres ved spørreskjemaer for å studere mulige assosiasjoner mellom potensielle årsaksfaktorer."

Komiteens vurdering

Dette er en masteroppgave hvor det skal gjøres en tverrsnittsstudie av beinhelsestatus hos en gruppe elitesyklister som driver ikke-vektbærende trening, hvorpå denne gruppen skal sammenlignes med beinhelsestatus hos langdistanseløpere som driver massiv vektbærende trening. Det vises til at vektbærende trening er antatt å styrke beinmassen, mens sykling ikke gjør det. Det er i dette prosjektet ønskelig å se om elitesyklistene har sub-optimal beinhelsestatus sammenlignet med løperne.

Det skal gjøres en klinisk undersøkelse av deltagerne i form av "dual energy x-ray absobtimetry scan", en røntgenscanning av deltagerens beinmasse som varer i 10 minutter. Deltakerne må da være fastende. I tillegg skal deltagerne svare på spørreskjema om trening, kosthold, sykdomshistorikk, skadehistorikk, vekt, høyde m.m.

Det skal rekrutteres ca. 20 deltagere fra hvert idrettsfelt, både kvinner og menn. Deltakerne vil være mellom 18-29 år og har trent på elitenivå i minimum 4 år. Alle deltakerne fra de aktuelle lagene vil bli spurt via sportslige ledere/ medisinske ansvarlige. Forskerne tar kontakt med disse i forkant og spør om de ønsker delta.

Besøksadresse: Gullhaugveien 1-3, 0484 Oslo
Telefon: 22845511
E-post: post@helseforskning.etikkom.no
Web: http://helseforskning.etikkom.no/
All post og e-post som inngår i saksbehandlingen, bes adressert til REK sør-øst og ikke til enkelte personer
Kindly address all mail and e-mails to the Regional Ethics Committee, REK sør-øst, not to individual staff

Komiteen oppfatter deltakergruppen som en begrenset og veldig lett identifiserbar gruppe, og det forutsettes derfor at man tar hensyn til risiko for blant annet bakveisidentifisering ved publisering.

Komiteen mener videre at informasjon- og samtykkeskrivet må modereres noe under «*Mulige fordeler og ulemper ved å delta som forsøksperson»*. Blant annet kan det virke kunstig forlokkende å friste med tilgang på «*ulike kostbare tester*», «*innblikk i hvordan forsknings bedrives»*, *«hjelp til å tolke resultater»* og lignende.

Ut fra dette setter komiteen følgende vilkår for prosjektet:
-Informasjonsskrivet revideres under «*Mulige fordeler og ulemper ved å delta som forsøksperson*». Revidert informasjonsskrivet sendes komiteen til orientering.

Komiteen vil bemerke at prosjektets sluttdato i søknadsskjema er satt til 15.01.202. Denne prosjektperioden ansees som for langvarig sett hen til prosjektets omfang. Komiteen gir derfor tillatelse til at prosjektet kan pågå frem til 15.01.2019. Det kan ved behov søkes om forlengelse dersom prosjektet ikke er sluttført innen ovennevnte sluttdato.

Vedtak

Komiteen godkjenner prosjektet i henhold til helseforskningsloven § 9 og § 33.

Godkjenningen er gitt under forutsetning av at prosjektet gjennomføres slik det er beskrevet i søknaden og etter at vilkår referert ovenfor er oppfylt.

Tillatelsen gjelder til 15.01.2019 Av dokumentasjonshensyn skal opplysningene likevel bevares inntil 15.01.2032. Opplysningene skal lagres avidentifisert, dvs. atskilt i en nøkkel- og en opplysningsfil. Opplysningene skal deretter slettes eller anonymiseres, senest innen et halvt år fra denne dato.

Forskningsprosjektets data skal oppbevares forsvarlig, se personopplysningsforskriften kapittel 2, og Helsedirektoratets veileder "*Personvern og informasjonssikkerhet i forskningsprosjekter innenfor helse- og omsorgssektoren"*

Sluttmelding og søknad om prosjektendring

Prosjektleder skal sende sluttmelding til REK sør-øst på eget skjema, jf. hfl. § 12. Prosjektleder skal sende søknad om prosjektendring til REK sør-øst dersom det skal gjøres vesentlige endringer i forhold til de opplysninger som er gitt i søknaden, jf. hfl. § 11.

Klageadgang

Du kan klage på komiteens vedtak, jf. forvaltningslovens § 28 flg. Klagen sendes til REK sør-øst B. Klagefristen er tre uker fra du mottar dette brevet. Dersom vedtaket opprettholdes av REK sør-øst B, sendes klagen videre til Den nasjonale forskningsetiske komité for medisin og helsefag for endelig vurdering. Med vennlig hilsen

Grete Dyb professor, dr.
med. leder REK sør-øst B

Mariann Glenna Davidsen
rådgiver

Kopi til:

- Norges idrettshøgskole ved øverste administrative ledelse

NIH NORGES IDRETTSHØGSKOLE

Beinhelse i norsk utholdenhetsidrett

For å bedre kunne tolke resultatene fra DXA undersøkelsen håper jeg du har tid til å svare på et kort spørreskjema vedrørende trening, idrettsrelaterte skader og kosthold.

For å kunne gjennomføre dette trenger jeg ditt samtykke. Deltakelse er frivillig og du kan trekke deg når du måtte ønske det uten noen form for begrunnelse. Det er kun autorisert personell knyttet til prosjektet som har adgang til navnelisten. Jeg ber også om å kunne benytte dataene som samles inn til forskningsformål. Alle data vil bli anonymisert og resultatene vil bli presentert på gruppenivå og på en slik måte at det ikke vil være mulig å identifisere deg når disse publiseres. Når studien er avsluttet og alle data er analysert, vil informasjonen bli slettet.

Takk for at du deltar i studien Beinhelse i norsk utholdenhetsidrett.

Med vennlig hilsen

Oddbjørn Klomsten Andersen

1) * Samtykke

- ☐ Jeg har lest og forstått informasjonen over og gir mitt samtykke til å samle inn informasjon om min treningshistorikk, idrettsrelaterte skader og kosthold.
- ☐ og vitenskapelig publisering av anonymiserte data

2) * Er du mann eller kvinne?

- ○ Mann
- ○ Kvinne

3) * Hvor gammel er du?

år

4) * Hvor høy er du?

cm

5) * Hva er din nåværende vekt?

kg

6) * Hva anser du som din konkurransevekt/"matchvekt"?

kg

7) * Nåværende idrettsgren?

- ○ Syklist
- ○ Løper

8) * Er du idrettsutøver på heltid?

- ○ Ja
- ○ Nei

9) Hvis nei: Hva driver du med ved siden av idretten?

- ☐ Heltidsjobb
- ☐ Deltidsjobb
- ☐ Studier/skolegang
- ☐ Annet

10) * På hvilket nivå konkurrerer du?

- ○ Klubb
- ○ Landslag, junior
- ○ Landslag, rekrutt
- ○ Landslag, senior
- ○ Profesjonelt

11) * Hva er din beste plassering i Norgesmesterskap (NM)/enkeltkonkurranse i Norgescup?

- ○ 1-3 plass
- ○ 4-6 plass
- ○ 7-10 plass
- ○ 11. plass eller dårligere
- ○ Ikke deltatt i NM eller norgescup
- ○ Husker ikke

12) * Hva er din beste plassering i VM, OL eller enkeltstående løp i verdenscupen?

- ○ 1-3 plass
- ○ 4-6 plass
- ○ 7-10 plass
- ○ 11. plass eller dårligere
- ○ Ikke deltatt i VM, OL eller verdenscup
- ○ Husker ikke

13) * Har du målt maksimalt oksygenopptak (VO2maks) siste 12 måneder?

- ○ Ja
- ○ Nei

14) Hva er ditt høyeste maksimale oksygenopptak (VO2maks) siste 12 måneder?

ml/kg/min

l/min

15) * På hvilket nivå konkurrerer du?

- ○ Klubb
- ○ UCI Continental Teams
- ○ UCI Professional Continental Teams
- ○ UCI WorldTeams
- ○ UCI Women's Team

16) * Hva er din beste plassering i Norgesmesterskap (NM)/enkeltkonkurranse i Norgescup?

- ○ 1-3 plass
- ○ 4-6 plass
- ○ 7-10 plass
- ○ 11. plass eller dårligere
- ○ Ikke deltatt i NM eller norgescup
- ○ Husker ikke

Enkelt etapper i etapperitt telles også her.

17) Hva er din beste plassering i et UCI løp?

- ○ 1-3. plass
- ○ 4-6. plass
- ○ 7-10. plass
- ○ 11. plass eller dårligere
- ○ Har ikke deltatt i UCI ritt
- ○ Husker ikke

18) * Hva er din beste plassering i VM, OL eller enkeltstående UCI WorldTour ritt?

- ○ 1-3 plass
- ○ 4-6 plass
- ○ 7-10 plass
- ○ 11. plass eller dårligere
- ○ Ikke deltatt i VM, OL eller UCI WorldTour ritt
- ○ Husker ikke

19) * Har du målt maksimalt oksygenopptak (VO2maks) siste 12 måneder?

- ○ Ja
- ○ Nei

20) Hva er ditt høyeste maksimale oksygenopptak (VO2maks) siste 12 måneder?

ml/kg/min

l/min

21) * Hvor mange år har du satset landeveissykling?

år

22) * Hvor gammel var du da du begynte å satse landeveissykling?

år

NB! Terrengsykling klassifiseres som andre idretter.

23) * Har du satset i andre idretter?

- ○ Ja
- ○ Nei

24) Hvilke andre idretter har du satset i?

- □ Terrengsykling
- □ Løp
- □ Langrenn
- □ Svømming
- □ Ballidrett
- □ Skøyter
- □ Triatlon
- □ Orientering
- □ Annet

NB! Om du har satset i flere idretter tidligere. Spesifiser hvor lenge du satset i de respektive idrettene.

25) Hvor mange år satset du i andre idretter?

år

NB! Om du har satset i flere idretter tidligere. Spesifiser hvor lenge det er siden du satset i de respektive idrettene.

26) Hvor mange år er det siden du satset i andre idretter?

år

27) * Hvilken ryttertype anser du deg selv som?

- Klatrer
- Sprinter
- Allround

Om du har ført treningsdagbok i denne perioden oppfordres det til å benytte den for å få mest mulig nøyaktig data.

28) * Hvor mange treningstimer hadde du i fjor (1. jan 2016 - 31. des 2016)?

timer

Om du har ført treningsdagbok i denne perioden oppfordres det til å benytte den for å få mest mulig nøyaktig data.

29) * Hvor mange mil sykler du i en gjennomsnittlig uke? Ta utgangspunkt i forrige sesong.

mil

Her menes systematisk trening med minimum 2 økter i uken. Tung styrketrening klassifiseres som styrketrening med motstand hvor du maksimalt klarer 15 repetisjoner før utmattelse.

30) * Har du trent tung styrketrening på beina over en periode på minimum 2 måneder de siste 4 årene?

- Ja, denne sesongen
- Ja, for mindre enn 2 år siden
- Ja, for 2 - 4 år siden
- Ja, for mer enn 4 år siden
- Nei

31) * Er de foregående svarene angående treningshistorikk basert på nøyaktige data fra treningdagbok?

- Ja
- Nei

32) * Hvor mange år har du satset løping?

år

33) * Hvor gammel var du da du begynte å satse løping?

år

34) * Har du satset i andre idretter?

- Ja
- Nei

35) * Hvilke andre idretter har du satset i?

- ☐ Landeveissykling
- ☐ Terrengsykling
- ☐ Ballidrett
- ☐ Langrenn
- ☐ Svømming
- ☐ Skøyter
- ☐ Triathlon
- ☐ Orientering
- ☐ Annet

NB! Om du har satset i flere idretter tidligere. Spesifiser hvor lenge du satset i de respektive idrettene.

36) Hvor mange år satset du i andre idretter?

år

NB! Om du har satset i flere idretter tidligere. Spesifiser hvor lenge det er siden du satset i de respektive idrettene.

37) Hvor mange år er det siden du satset i andre idretter?

år

Om du har ført treningsdagbok i denne perioden oppfordres det til å benytte den for å få mest mulig nøyaktig data.

38) * Hvor mange kilometer løper du i en gjennomsnittlig uke? Ta utgangspunkt i forrige sesong..

km

De neste spørsmålene vil omhandle treningshistorikk det forrige året. Om du har ført treningsdagbok i denne perioden oppfordres det til å benytte den for å få mest mulig nøyaktig data.

39) * Hvor mange treningstimer hadde du i fjor (1. jan 2016 - 31. des 2016)?

timer

Her menes systematisk trening med minimum 2 økter i uken. Tung styrketrening klassifiseres som styrketrening med motstand hvor du maksimalt klarer 15 repetisjoner før utmattelse.

40) * Har du trent tung styrketrening på beina over en periode på minimum 2 måneder de siste 4 årene?

- ○ Ja, denne sesongen
- ○ Ja, for mindre enn 2 år siden
- ○ Ja, for 2 - 4 år siden
- ○ Ja, for mer enn 4 år siden
- ○ Nei

41) * Er de foregående svarene angående treningshistorikk basert på nøyaktige data fra treningdagbok?

- ○ Ja
- ○ Nei

42) * Hvilke distanser konkurrerte du i forrige sesong (2016)?

- ☐ Under 400 m
- ☐ 400 - 800 m
- ☐ 800 - 1500 m
- ☐ 1500 - 3000 m
- ☐ 3000 - 10 000
- ☐ > 10 000

43) * Hvilken løpsdistanse er din sterkeste?

- ○ distanse under 800 m
- ○ 800 - 1500 m
- ○ 1500 - 3000 m
- ○ 3000 - 10 000 m
- ○ > 10 000 m

Med sprintdistanser menes 400 meter eller kortere

44) * Har du konkurrert i sprintdistanser tidligere?

- ○ Ja
- ○ Nei

45) Hvilke sprintdistanser konkurrerte du i?

46) Hvor lenge er det siden du konkurrerte i sprintdistanser?

- ○ under 2 år siden
- ○ 2-4 år siden
- ○ 4-6 år siden
- ○ 6-8 år siden
- ○ mer enn 8 år siden

47) Hvor lenge konkurrerte du i sprint distanser?

- ○ under 1 år
- ○ 1-2 år
- ○ 2-3 år
- ○ 3-4 år
- ○ mer enn 4 år

Med stillesittende menes all aktivitet som foregår i en sittende eller liggende posisjon. Dette innebærer sittestillende arbeid, forelesninger, TV og PC tid, søvn om dagen osv.

48) * Hvor mange timer i løpet av et døgn tilbringer du stillesittende (eksklusive nattesøvn)?

- ○ mindre enn 2 timer
- ○ 2 - 4 timer
- ○ 4 - 6 timer
- ○ 6 - 8 timer
- ○ 8 - 10 timer
- ○ > 10 timer

49) * Bedriver du ukentlig tung fysisk aktivitet/hardt fysisk arbeid utenom trening? Bedriver du tung fysisk aktivitet/hardt fysisk arbeid utenom trening? Eksempler kan være fjellturer, klipping av gress, snekring osv.

- ○ Nei
- ○ Ja, mindre enn 2 timer ukentlig
- ○ Ja, 2 - 4 timer ukentlig
- ○ Ja, 4 - 6 timer ukentlig
- ○ Ja, mer enn 6 timer ukentlig

50) Beskriv den harde fysiske aktiviteten du gjør utenom treningen?

Idrettsskader

A) Akutte skader

Med **akutt frakturer** menes brudd som har oppstått som følge av en enkelt hendelse. Dette skiller seg fra **stressfrakturer** som oppstår som følge av ensidig og gjentatte belastninger.

51) * Har du hatt akutt frakturer (beinbrudd) i løpet av din idrettskarriere?

- ○ Ja
- ○ Nei

52) Hvor mange akutte frakturer (beinbrudd) har du hatt i løpet av din idrettskarriere?

frakturer

53) Hvilke områder har du hatt akutte frakturer?

54) Antall akutte frakturer siste året

frakturer

NB! Dette spørsmålet gjelder **ALLE TYPER** akutte skader. Eks. beinbrudd, strekk, kuttskader, skader relatert til velt, overtråkk osv.

55) Hvor mange dager i løpet av det siste året har du måtte stå over trening/konkurranse på grunn av en akutt skade?

dager

B) Stressfrakturer og andre belastningsskader

Med stressfrakturer menes frakturer som skyldes belasting over tid.

56) * Har du hatt stressfrakturer (tretthetsbrudd) i løpet av din idrettskarriere?

- ○ Ja
- ○ Nei

57) Hvor mange stressfrakturer har du hatt i løpet av din idrettskarriere?

frakturer

58) Hvilke områder har du hatt stressfrakturer?

59) Antall stressfrakturer siste året

frakturer

60) * Har du hatt andre betydelige belastningskader det siste året som har ført til nedsatt prestasjonsevne eller et treningsopphold på mer enn 3 uker?

- ○ Ja
- ○ Nei

61) Beskriv skadene

62) * Hvor mange dager i løpet av det siste året har du måtte stå over trening/konkurranse på grunn av stressfrakturer eller andre belastningsskader?

dager

63) * Hvor mange dager i løpet av det siste året har du hatt nedsatt prestasjonsevne som følge av stressfrakturer eller andre belastningsskader?

- 0-7 dager
- 7-28 dager
- Mer enn 28 dager

C) Sykdom

Med sykdom menes vanlige sykdommer slik som forkjølelse, bihulebetennelse, influensa ect. Det er ikke snakk om kroniske sykdommer.

64) Har du vært syk det siste året?

- Ja
- Nei

65) Hvor mange ganger det siste året har du vært syk?

ganger

66) Hvor mange dager i løpet av det siste året har du måtte stå over trening på grunn av sykdom?

dager

67) Hvor mange dager i løpet av det siste året har du hatt redusert prestasjonsevne på trening/konkurranse som følge av sykdom?

- 0-7 dager
- 7-28 dager
- Mer enn 28 dager

D) Overtrening

68) Har du vært overtrent slik at det har påvirket din idrettsprestasjon i minimum 3 uker i løpet av din idrettskarriere?

- Ja
- Nei

69) Hvor mange dager i løpet av det siste året har du måtte avstå fra trening som følge av overtrening?

dager

70) Hvor mange dager i løpet av det siste året har du gjennomført treningen med nedsatt prestasjonsevne som følge av overtrening?

- 0-7 dager
- 7-28 dager
- mer enn 28 dager

E) Menstruasjon

71) Har du normal menstruasjon?

- Ja
- Nei
- Vet ikke

72) Hvis ja, har du regelmessig menstruasjon (hver 28. - 32. dag)

- Ja, som regel
- Nei, som regel ikke

73) Hvis nei eller vet ikke, hvor lenge er det siden du sist hadde menstruasjon?

- 2-3 måneder
- 4-5 måneder
- 6 måneder eller mer
- Jeg er gravid og har derfor ikke menstruasjon
- Jeg går på minipiller og har derfor ikke mensturasjon

74) Har din menstruasjon uteblitt helt i 3 måneder eller lengre uten at det skyldes graviditet eller minipiller?

- Nei, det har aldri skjedd
- Ja det har skjedd tidligere
- Ja, jeg opplever det nå

Kalsiuminntak

De neste spørsmålene vil dreie seg om kalsium inntak i hverdagen. Dette spørreskjemaet er et standardisert og validert spørreskjema og av den grunn vil spørsmålene og instruksen til spørsmålene bli gitt på engelsk.

To work out how much calcium you are getting on average in a day:

1. Write down the number of times **in a typical week** that you eat or drink the items lited below.

75) * How many glasses of milk do you drink in a typical week (1 glass = 2 dl)?

glasses

76) * How many cups of tea with milk do you drink in a typical week?

cups

77) * How many cups of coffee with milk do you drink in a typical week?

cups

78) * How many cups of milky drinks (cafe latte, milkshakes, chocolate milk) do you drink in a typical week?

cups

79) * How many portions of milk with cereal/porridge do you eat in a typical week?

portions

80) * How many slices of white or brown bread do you eat in a typical week?

slices

81) * How many slices of wholemeal bread do you eat in a typical week?

slices

82) * How many portions of hard cheese (i.e. norvegia) do you eat in a typical week?

portions

83) * How many portions of cottage cheese do you eat in a typical week?

portions

84) * How many biscuits do you eat in a typical week?

biscuits

85) * How many portions of cake do you eat in a typical week?

portions

86) * How many portions of milk pudding (i.e. ice cream, yoghurt) do you eat in a typical week?

portions

87) * How many portions of green vegetables do you eat in a typical week?

portions

88) * How many portions of sardines or pilchards (i.e. makrell i tomat, sardiner på boks) do you eat in a typical week?

portions

89) * How many portions of fish do you eat in a typical week?

portions

90) * How many portions of soft cheese (i.e. mozzarella) do you eat in a typical week?

portions

91) * How many portions of muesli do you eat in a typical week?

portions

92) * How many pancakes or waffles do you eat in a typical week?

pancakes/waffles

93) * How many oranges do you eat in a typical week?

oranges

94) * How many eggs do you eat in a typical week?

eggs

95) * How many portions of bean curd/tofu do you eat in a typical week?

portions

96) * How many portions of cheese sauce based dish (i.e. macaroni cheese) do you eat in a typical week?

portions

97) * How many portions of dried fruit or nuts do you eat in a typical week?

portions

98) * How many portions of hummus do you eat in a typical week?

portions

Kalsium inntak under trening

99) * Er du bevisst på å få i deg nok salter under trening?

- ○ Ja, alltid
- ○ Ja, ofte
- ○ Ja, av og til
- ○ Nei, sjelden eller aldri

100) * Drikker du kalsiumholdig drikke under trening?

- Ja, alltid
- Ja, ofte
- Ja, av og til
- Nei, sjelden eller aldri
- Vet ikke

101) * Spiser du kalsiumholdig mat under trening?

- Ja, alltid
- Ja, ofte
- Ja, av og til
- Nei, sjelden eller aldri
- Vet ikke

102) Tar du kalsiumtilskudd i hverdagen?

- Ja, hver dag
- Ja, en eller flere ganger i uken
- Ja, en eller flere ganger i måneden
- Sjeldnere eller aldri

Vitamin D

103) * Hvor ofte tar du tran eller omega 3 tilskudd i perioden September-April?

- 5 - 7 ganger i uken
- 3 - 4 ganger i uken
- 1 - 2 ganger i uken
- 1 - 3 ganger i måneden
- Aldri eller sjeldnere enn en gang i måneden

En porsjon = 5 gram

104) * Hvor mange porsjoner med margarin eller meierismør bruker du hver dag?

porsjoner

Meieriprodukter tilsatt vitamin D:
Ekstra lett melk
Biola drikker
Styrk drikker
Sprett+yoghurt
Lakktoseredusert lettmelk
1 porsjon = 1,5 dl eller 1 beger youghurt

105) * Hvor mange porsjoner meieriprodukter tilsatt vitamin D bruker du hver dag?

porsjoner

Fet fisk = Laks, makrell, ørret, kveite eller røye

106) * Hvor mange porsjoner med fet fisk spiser du i løpet av en uke?

porsjoner

Fiskepålegg av fete fiskeslag = makrell i tomat, gravlaks, røkelaks, peppermakrell, sild o.l.

107) * Hvor mange porsjoner med fiskepålegg av fete fiskeslag spiser du i løpet av en uke?

porsjoner

108) * Hvor mange egg spiser du i løpet av en uke?

egg

Sydligere strøk er her definert som Sør Europa eller andre steder med tilsvarende temperaturer om vinteren.

109) * Hvor mange reisedøgn til sydligere strøk har du i gjennomsnitt i månedene september - april?

reisedøgn

110) * Bruker du solkrem med solfaktor når du er eksponert for sterk sol om sommeren i Norge eller når du er i sydligere strøk på samlinger?

- ○ Ja, solfaktor 10 eller lavere
- ○ Ja, solfaktor 10 - 20
- ○ Ja, solfaktor 20 - 30
- ○ Sterkere enn 30
- ○ Nei