Osteoporosis and Poor Bone Health in Cyclists: A Review

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How to navigate this page

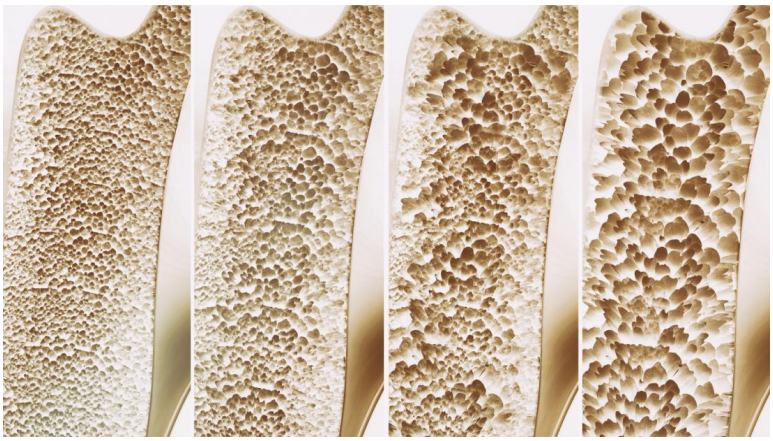
There are two different ways I've written the material. Both address the main issues investigated but are addressed to different audiences. First is "for the cyclist." This section is directed towards people with limited anatomy and physiology backgrounds. It aims to keep it big picture while introducing details that may help understanding more complex topics. More extensive citations and descriptions of individual studies can be found in the second section, "for the clinician." This section is for those who may be currently in school or practicing in the health field. It goes much more in depth into the mechanisms and literature behind bone health in cyclists. Sources and a data table of relevant literature follow both. Enjoy, and don't be afraid to reach out with questions or just start a conversation (cyclingmvmt@gmail.com).

For the Cyclist

Introduction

Cycling is a vastly popular activity. Endurance cycling has many health benefits while simultaneously reducing impact forces found in running or team sports. Although high impact forces in excess can lead to injury, appropriate amounts of stress on your bones are imperative to overall skeletal health. As such, cyclists are considered at risk for bone disorders such as osteopenia and osteoporosis due to chronic de-loading of their skeletal system.

Osteoporosis is a bone disorder characterized by decreased bone mass or bone mineral density (BMD). The picture below depicts the progression of bone loss going from left to right, or healthy to unhealthy. Osteopenia is simply a milder version of osteoporosis, and if discovered can serve as a warning sign.



Progression of bone loss – *Medical News Today*

Although the exact cause for osteoporosis is yet to be discovered, the cellular explanation has been well documented. Bones are a form of connective tissue. They protect the inner body and provide structure to the body. The bone itself is constantly changing as a result of worker cells forming and taking away bone. In several years your thighbone, for example, may contain only a small percentage of cells currently present. In this way, bone is alive and always changing.

In normal bone, there is a balance between bone formation and resorption (taking away). In osteoporosis, however, the balance favors the breakdown of bone, effectively decreasing BMD. This loss reduces the skeletal system's ability to resist stress, making it particularly vulnerable to fractures.

Cyclists and Decreased BMD

Cyclists have been shown in multiple studies to have decreased BMD compared to a variety of populations. Out of the 17 studies included in the appended table, 15 found low BMD in cyclists in some respect. These studies included a wide demographic including male, female, elite, competitive, recreational, masters, and adolescent cyclists. The participants were shown to have decreased BMD compared to runners and triathletes as well as inactive controls. Cycling, in some capacity, clearly has a detrimental effect on bone.

The hip and lumbar spine (low back) are most commonly affected, two sites often subject to fragility fractures in osteoporotic populations. One study found a staggering 16% difference in elite cyclists' lumbar BMD compared to controls in addition to a 15% difference in one part of the hip. It is argued that competitive cyclists experience approximately 1.0% decrease in lumbar BMD during one year of competition. This BMD wasn't recovered in the off season. Multiply this decrease over several years, and the result could be dramatic deficiencies. For example, a 7-year study on masters cyclists found that they were much more likely to progress from osteopenia to osteoporosis compared to sedentary individuals (31.6% vs. 5.6%).

That being said, one study on master track cyclists found greater bone strength in their arms and shins. And cyclists who participated in multiple disciplines such as mountain biking or cyclocross had higher site-specific BMD. Lastly, one study by Baker et al. found no differences in BMD over a competitive season of cycling, but found that higher competitive levels (Cat1 vs Cat4) and older age were correlated with lower BMD. High competitive level, older age and low BMD were consistent throughout the literature.

Common Risk Factors

As stated in the introduction, the exact cause for osteoporosis hasn't been discovered in the general population. Multiple risk factors, however, have been identified. Although all are relevant, the final four on the following list will be explored in greater detail as they relate to cycling specifically.

- Low BMI
- Family history
- Regular smoking
- Alcohol consumption
- Oral glucocorticoid use
- Rheumatoid arthritis
- Type 1 diabetes mellitus
- Hyperthyroidism
- Chronic malnutrition
- Premature menopause (<45 years)
- Malabsorption
- Chronic liver disease
- *Current inactivity*
- *Age*
- *Decreased calcium intake*
- *Female*

Activity Considerations

We've established that bone is constantly improving its microstructure, and just like a muscle being worked in a gym, a bone will get stronger when appropriate stress is placed on it. This is called Wolff's Law: "bone tissue forms and is remodeled in response to the mechanical forces that it experiences." Stress can occur from impact forces such as jumping, or due to muscles pulling on their attachments on bone. For example, high impact athletes such as gymnasts or weightlifters have been shown to have higher BMD compared to matched controls (age/weight/gender etc.), presumably because their bones have adapted to the stresses experienced. Swimmers, and other low impact athletes, on the other hand, have been shown to have lower BMD.

Harold Frost expanded upon Wolff's law by introducing the mechanostat theory. This theory proposes that a bone must reach a certain threshold of strain before bone growth can occur. Studies have shown that low intensity cycling only reaches 20-30% of the strain needed to cause positive change. This suggests that cycling has limited osteogenic (bone growth) properties, resulting in lower BMD.

This concept makes sense when considering low BMD in cyclists' spines. The lumbar spine is largely de-weighted due to their horizontal posture, eliminating gravitational compression in the upper body. Wolff's Law and the Mechanostat Theory would also explain why track cyclists and cyclocross athletes have improved BMD. These disciplines undergo much higher muscular stresses due to the explosive nature of the sports. In addition, cyclocross incorporates weight-bearing activities such as jumping barriers or running up steep slopes. Considering these factors, the stresses applied may reach threshold for positive bone growth.

One of the more applicable pieces of evidence is that masters cyclists who include regular weight training or running (2x/wk), lose significantly less BMD, as found in a study by Nichols et al. In a study by Lagendonck et al, non-cycling adults who participate in weight bearing activity have significantly greater BMD than those who stop physical activity after adolescence.

Although the argument that weight supported activity reduces BMD is attractive, it fails to explain why cyclists have lower BMD compared to healthy but relatively inactive controls. As such, other factors must play into why cyclists are at a higher risk for osteoporosis and osteopenia.

Age

Osteoporosis is a disorder commonly associated with older adults. The studies included in the appended table indicate that older age was correlated with lower BMD, as expected. That being said, masters cyclists were losing BMD at a much higher rate than their age-matched peers, indicating that cycling accentuates this process, making older cyclists an even higher risk population.

It is also argued that initiating appropriate bone growth at a younger age could improve outcomes later on in life. BMD peaks around the age of 25-30 and decreases starting at age 35. The higher the peak, the more it takes to decrease into osteoporotic values. Adolescent cyclists were demonstrated to have lower BMD than their healthy active pears. Bone loss during these developmental periods could lower peak BMD, initiating a more rapid onset of bone disorders such as osteoporosis later in life. It has been shown, however, that the major factor in bone health for older adults is continuing weight-bearing activity later in life.

Nutrition and Energy Availability

Tradition tells us drinking milk makes our bones strong, and it's a justified claim. Appropriate calcium intake has been described as a major factor when considering bone health. The rational is based on the complex role bone has in maintaining appropriate levels of calcium in the blood. Whenever calcium is low in the blood, the body taps into calcium stores in the bone. This effectively weakens the structure. This is happening constantly, and so long as calcium is appropriately resoted via diet, there will be no net loss of bone density. It's proposed, however, that when calcium isn't adequately replaced, the bone will never re-fortify. Think of going to an ATM to get money (the ATM is bone), but the bank never refills it. Over time the ATM will lose all of its money, rendering it ineffective. Bone is the same way.

Some studies suggest that cyclists lose calcium through sweating, therefore making it important to replenish stores. That being said, no study investigating calcium levels in cyclists correlated low calcium diets to low BMD, suggesting that increasing dietary calcium may have little effect on bone health in cyclists. These studies, however, weren't controlled very well and should be taken with a grain of salt.

Vitamin D has also been shown to improve calcium absorption in the gut, maximizing dietary intake. Although vitamin D supplementation hasn't been correlated with improved BMD in cyclists, the combination of vitamin D and calcium supplementation in middle-aged and older adults resulted in a 15% decrease in total fractures (30% at the hip), as demonstrated through a meta-analysis performed by the International Osteoporosis Foundation.

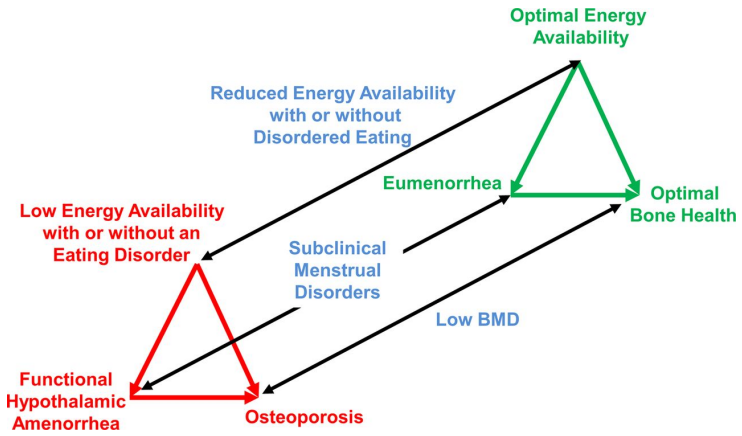
Several studies on cyclists, however, correlated low caloric intake with low BMD. It is argued that a deficit of 30 kcal * kg * FFM^{-1}*day^{-1} or greater leads to detriments in bone health, mainly due to metabolic and hormonal stresses. Endurance athletes, specifically runners and cyclists, are known to reduce weight with the intention of improving performance, therefore placing themselves in dangerously high caloric deficits. It's important to lose weight safely and carefully watch your energy availability to prevent a host of issues in addition to poor bone health.

Gender

A disproportional 70% of all fractures affect women over 65 years old, and one in every six white woman will have a hip fracture during her lifetime. Differences in gender typically come down to hormonal changes at menopause, but isn't limited to older women.

One study showed that pre-menopausal cyclists' BMD declined at a similar rate compared to men. No study focused on post-menopausal cyclists and therefore conclusions can't be made. That being said, during menopause, women have a rapid decrease in estrogen (sex hormone), an important component of regulating bone resorption. As a result, risk of fragility fractures increases at menopause and continues to increase gradually until the age of 75.

Although hormonal changes are notable in women at older ages, hormonal imbalance can occur at any age. Most profound is the female athlete triad, "a disorder best described as the relationship between low energy availability… menstrual dysfunction and decreased bone mineral density". This is demonstrated as a spectrum (not an absolute), depicted by the figure below. Energy deficiency may be a result of an eating disorder such as anorexia and bulimia. This presents with a much more complicated physcosocial element. Although these components don't have to present in a particular order, energy availability is often the precursor to altered menstrual status and poor bone health. Low caloric deficits disrupt many of the important sex hormones. As discussed earlier, the alteration of hormonal balance has a direct relationship to bone health. This reinforces the importance of proper caloric and nutritional intake, especially in females who are exerting themselves at a high level, such as cyclists.



Conclusion and Recommendations

It's been clearly demonstrated that bone health should be a concern for competitive cyclists who are spending a large amount of time training for their sport. It seems that higher competitive levels, larger training volumes, and lower caloric intake are major factors when considering BMD. Cyclists are at particular risk of low BMD in the hip and spine, common locations for fragility fractures in older adults.

Cycling involves high velocity crashes and therefore subsequent trauma to the skeletal system is not uncommon. Although fractures can occur in healthy bone, lower BMD can increase the severity of the trauma. As such, current lifestyle factors may determine bone health later on in life.

There are two easily controlled aspects in training that any cyclist can include to improve bone health. Firstly, include weight training or running/jogging into your training routine two times per week. Weight training is typically unappealing to cyclists due to the mass gains and lack of training specificity. That being said, multiple studies have demonstrated performance gain with weight training, suggesting the ability to hit two birds with one stone.

Secondly it's important to control dietary factors such as caloric, calcium, and vitamin D intake. The evidence behind calcium and vitamin D intake in cyclists is mixed, however supplementation has been shown to decrease risk of fractures. A s result it wouldn't hurt to stick to the daily recommended values for calcium (>1000mg) and vitamin D (>400mg). Other risk factors within a cyclists control include limiting alcohol consumption and smoking.

Limitations

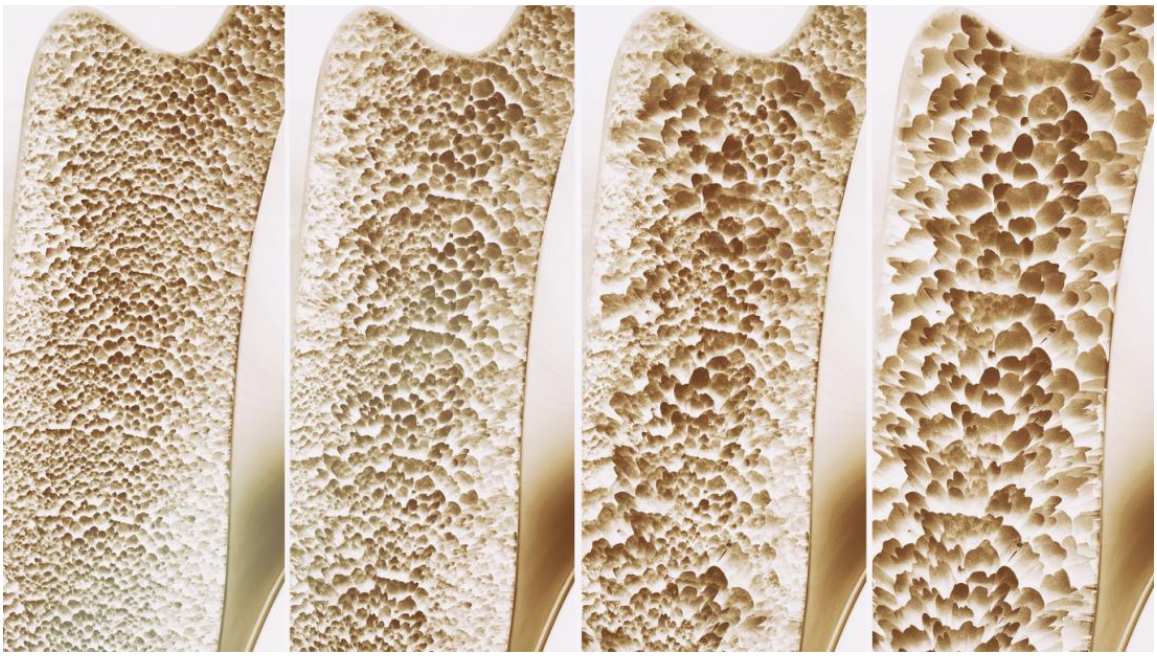
Certainly there are limitations to this review. Firstly, although many studies have come out since the turn of the millennium, the quality of these studies are lacking. Low populations, cross-sectional design, and poorly controlled variables limit the ability to extrapolate causation. Secondly, it isn't within my scope to recommend detailed dietary plans, nor appropriate medicinal interventions. As such, it's important that you consult with your PCP about bone health if you relate to any of the risk factors. Lastly, my own level of understanding regarding the depth of much of the material is a limitation in itself. Although I do believe I've learned a lot, each study leads to another, initiating an endless cycle of detail difficult to master. That being said, I expect to continue advancing my understanding on the topic and will update this page as I go along.

For the Clinician

Introduction

Cycling is a vastly popular activity. Endurance cycling has many health benefits while simultaneously reducing impact forces found in running or team sports. Although high impact forces in excess can lead to injury, appropriate amounts of stress on your bones are imperative to overall skeletal health. As such, cyclists are at risk for bone disorders such as osteopenia and osteoporosis due to chronic de-loading of their skeletal system, and have been shown to be the case in multiple studies.

Osteoporosis is a "skeletal disorder characterized by compromised bone strength [consequently increasing] risk of fracture" [1]. This is a result of altered osteogenic and osteoclastic balance favoring deterioration of trabecular and cortical bone. Loss of bone mineral density (BMD) reduces the skeletal system's ability to resist stress making it particularly vulnerable to fractures. Someone is considered osteoporotic when their site specific BMD, as measured by dual energy x-ray absorptiometry (DEXA), is greater than 2.5 standard deviations (SD) off of healthy young values and osteopenic when between 1 and 2.5 SD away [1]. In the picture below you can see a spectrum of bone samples, giving a physical representation of these disorders.



Progression of bone loss – *Medical News Today*

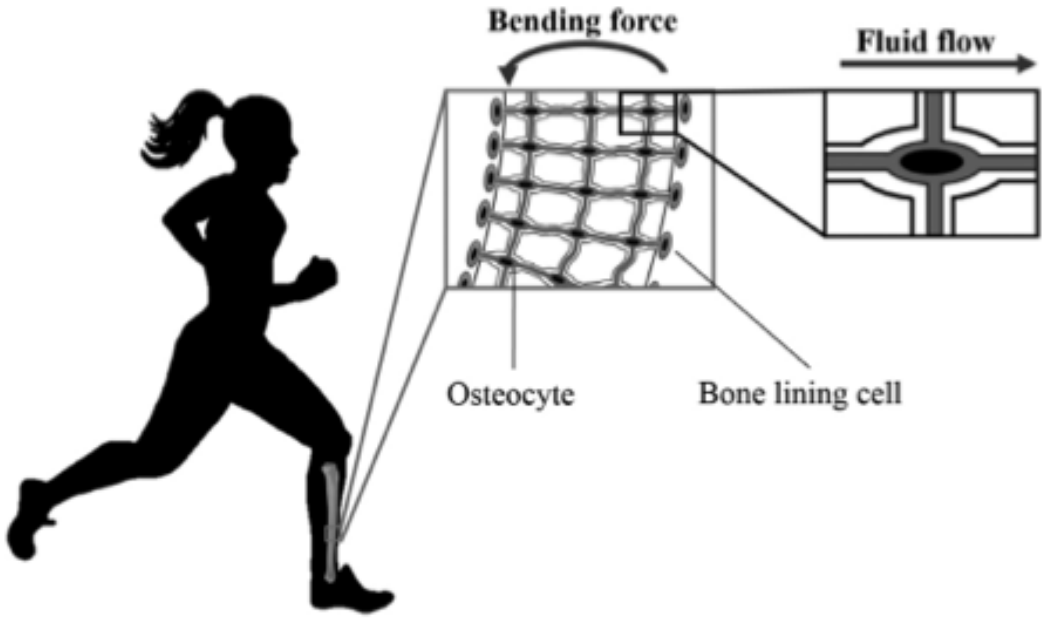
The histopathology of bone loss is rather complex but can be simplified to three cells: osteoclasts, osteoblasts, and osteocytes. These three cells together dictate whether bone is built or resorbed [2].

Osteoclasts are responsible for resorption of bone. They are derived from hematopoietic stem cells and resemble monocytes and macrophages found elsewhere in the body [2]. Osteoclasts attract hydrochloric acid and secrete acid phosphatase, dissolving bone and collagen respectively [3]. Osteoclastic activity is important in removing unnecessarily laid collagen and helps regulate

blood calcium levels [3]. Osteoclasts are integral to regulating bone deposition, therefore providing an integral function in bone structure.

Osteoblasts are the builders of bone and are derived from mesenchymal stem cells [2]. They produce helical collagen networks within osteons and allow calcium and phosphate ions to mineralize these preliminary scaffolds [3].

Osteocytes are fascinating cells. They are found in caniculi and lacunae and respond to changes in compartmental fluid pressure. As demonstrated in the diagram below, weight-bearing activity causes deformation of bone, resulting in fluid displacement and stimulation of osteocytes [4]. It has also been suggested that osteocytes respond to deformation of its own cell [5]. The details of mechanotranduction are still under investigation, but the outcomes are well studied [6].



How bone strain and fluid flow determine osteocyte behavior – [4]

Upon stimulation, the osteocytes initiate a cascade of responses including opening Ca^{2+} channels, increasing intracellular protein Kinase C and stimulating anabolic regulators of bone growth such as nitric oxide and prostaglandin E2 [4]. These prompt osteoblasts to lay down bone where stress is being applied [3]. As a result, compression and stimulation of osteocytes is key to bone growth, leading us to the concept that activity type, particularly weight bearing conditions, is a large determinant of bone health. In this sense, bone is constantly changing as a result to the stresses applied. Bone is a live and dynamic tissue.

Cyclists and Decreased BMD

Cyclists have been shown in multiple studies to have decreased BMD compared to a variety of populations. Out of the 17 studies exploring BMD in cycling (described in table 1), 4 studies found decreased BMD over time [8,11,12,13], 6 found decreased BMD in cyclists compared to controls [14,15,16,17,18,19], 3 found decreased BMD in cyclists compared to runners [10,20,21], and 1 found deceased BMD in cyclists compared to both runners and controls [22]. Of the studies that compared elite vs trained vs recreational cyclists [23, 24], the higher competing levels and

training volumes were correlated with lower BMDs. Likewise, older age was also correlated with lower BMD [17,12]. Clearly cycling in some capacity has a detrimental effect on bone.

These low BMD patterns are expressed the greatest in hip and lumbar regions of cyclists, two sites often subject to fragility fractures in osteoporotic populations [1]. Campion et al found a staggering 16% difference in elite cyclist's lumbar BMD compared to controls in addition to a 15% difference in trochanteric values [14]. Several studies were only able to find deficiencies in the lumbar spine while all other sites were considered normal [8,19]. It is argued that competitive cyclists experience ~ 1.0% decrease in lumbar BMD over just one year that isn't recovered in the off-season [11,13]. This is similar to the decreases experienced by post-menopausal women (~1.5%) [13]. Add that for multiple years and you have a dramatic decrease in BMD. A 7-year longitudinal study comparing BMD of competitive male masters and age matched non-athletes, for example, found that 31.6% of the cyclists progressed from osteopenic to osteporotic whereas only 5.6% of non-athletes did [12].

That being said, one study on track cyclists found 9-13% and 8-13% improvements in RPol (bone strength measure) at the tibia and radius respectively, compared to matched controls [26]. This was attributed to higher forces for shorter durations and improved imaging techniques (pQCT) [26]. That being said, there were no significant differences in BMD, arguably a less functional measure compared to RPol. Likewise, there has not been a study to date using pQCT to investigate bone health at the hip and lumbar spine of cyclists, an increasingly important site-specific correlation. Lastly, one study didn't find any significant changes in BMD of competitive cyclists (Cat 1 and C4) over a summer of racing [23].

Common Risk Factors

No one cause has been pinpointed for osteoporosis in the general population, however, multiple risk factors have been identified (demonstrated in the list below). Although all are relevant, the last four will be explored in greater detail as they relate to cycling specifically.

- Low BMI [1]
- Family history [2]
- Regular smoking
- Alcohol consumption
- Oral glucocorticoid use
- Rheumatoid arthritis
- Type 1 diabetes mellitus
- Hyperthyroidism
- Chronic malnutrition
- Premature menopause (<45 years)
- Malabsorption
- Chronic liver disease
- Age
- Female
- Current inactivity [7]
- Decreased calcium intake [2]

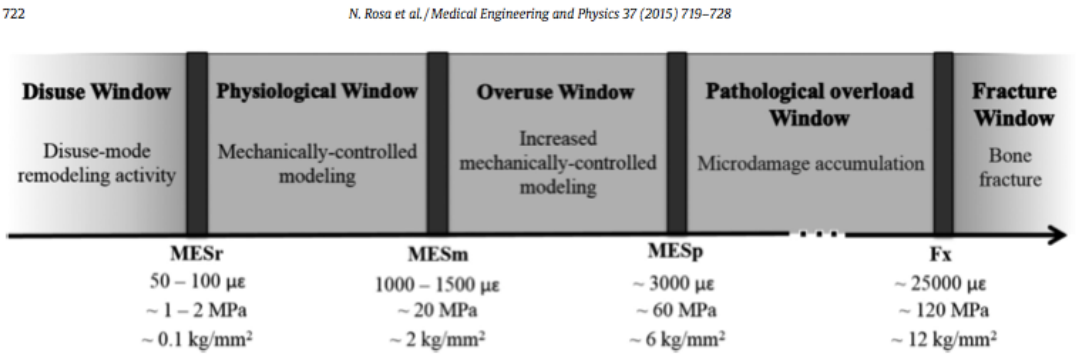
Activity Considerations

It has been established that bone is constantly improving its microstructure based on stresses applied. Bone growth, as a result of mechanical load, relies on its frequency, strain rate, amplitude, duration, and rest periods between loads [6]. The mechanical stimulus can come from muscular contractions or external forces such as gravity (weight bearing) or impact [4]. In the 19th century Julius Wolff was one of the first scientists to present this relationship, deriving Wolff's Law: "bone tissue forms and is remodeled in response to the mechanical forces that it experiences" [5]. In 1960 Harold Frost refined Wolff's law to include the idea that bone growth is due to elastic deformation and responds based on specific strain values, introducing the

mechanostat theory [6]. This theory has been the basis for osteocyte behavior and is continually improved with further research.

Frost proposed that bone would respond in different ways depending on the minimal effective strain (MES), suggesting different zones of osteogenic or clastic activity, as demonstrated in the figure below [4]. According to this theory, to increase bone density the mechanical strain of an activity should fall within 1000-1500με, and anything below that will not be osteogenic [4].

Subsequent studies have demonstrated that cycling (low intensities) falls around 300με, jogging around 900με, and a forward jump around 1600με [4]. These studies were performed in the frequency range of 1-60hz, however, newer evidence suggest that higher frequencies at lower strain values can induce comparable results to those in the overuse window [4]. This is the basis for vibration therapy. That being said, it strengthens the hypothesis that low intensity cycling is much less stressful to the skeletal system than many other weight bearing activities, leading to potential detriments in bone health.



Windows of bone activity based on strain values – [4]

Based on Wolff's law and the mechanostat theory, activity dictates bone growth. A study on pre-adolescent female gymnasts, a high impact sport, found higher BMDs in the gymnasts compared to age matched controls [9]. Unsurprisingly, on the other end of the spectrum, swimmers have been demonstrated to have much lower site specific BMDs compared to running athletes and controls [10].The repetitive low force movements along with a weight-supported environment in cycling may not provide appropriate stimulus to induce bone growth.

This concept especially takes strength when considering low BMD in cyclists' spines. The lumbar spine is largely de-weighted due to their horizontal posture, eliminating gravitational compression in the upper body. Higher muscular forces at the hip could potentially reach threshold, but at lower intensities, this wasn't seen as the case [4]. Likewise, Wolff's Law and the Mechanostat Theory would explain why track and cyclocross athletes have improved BMD. These disciplines undergo much higher muscular stress due to the explosive nature of the sports. In addition, cyclocross has been shown to have a higher osteogenic index than other disciplines, explained by weight bearing skills such as jumping barriers and running up steep grades [25].

A more applicable piece of evidence regarding activity specificity and bone growth is that master who included regular weight training or running (2+x/wk), lost significantly less BMD over a 7-year period [12]. This suggests osteogenic activities (running, jumping, weight lifting) utility in maintaining bone health in cycling specific populations. Similarly, non-cycling older adults who

continued weight bearing activities had significantly greater BMD than those that stopped physical activity [7]. Triathletes have been shown to have no change in BMD over a 24 week competitive season [27] and have higher site specific BMD than controls (adolescent females) when assessed at one point in time [10]. This is attributed to the inclusion of running in their training.

Although the argument that weight supported activity reduces BMD is attractive, it fails to explain why cyclists had lower BMD compared to healthy but relatively inactive controls. As such, other factors must play into why cyclists are at a higher risk for osteoporosis and osteopenia.

Age

Osteoporosis is a disorder commonly associated with older adults [1]. The studies presented thus far indicate that cycling may induce decline in bone similar to that seen during the natural aging process. One study compared competitive masters and young cyclists to age matched controls [17]. Four out of the 27 masters cyclists were osteoporotic while none of the other groups were. Likewise, masters cyclists had approximately 10% lower BMD at the hip and spine compared to their age-matched controls [17]. As stated before, a 7-year longitudinal study comparing BMD of competitive male masters and age matched non-athletes found that 31.6% of the cyclists progressed from osteopenic to osteporotic whereas only 5.6% of non-athletes did [12]. This gives rise to the concern for older adults participating in cycling, especially at the competitive level.

It is also argued that initiating appropriate bone growth at a younger age could improve outcomes later on in life. BMD peaks around the age of 25-30 and decreases from there [7]. Duncan et al found that adolescent female cyclists had 11% less leg BMD compared to runners [10] while Gonzalez-Aguero et al concluded that adolescent endurance-trained cyclists had lower cortical and trabecular BMD and BMC at some sites at the tibia and radius compared to age matched controls [15]. Olmedillas et all similarly found a 17% decrease in BMD in adolescent cyclists compared to age-matched controls [18]. Bone loss during these developmental periods could lower peak BMD, initiating a more rapid onset of bone disorders such as osteoporosis later in life.

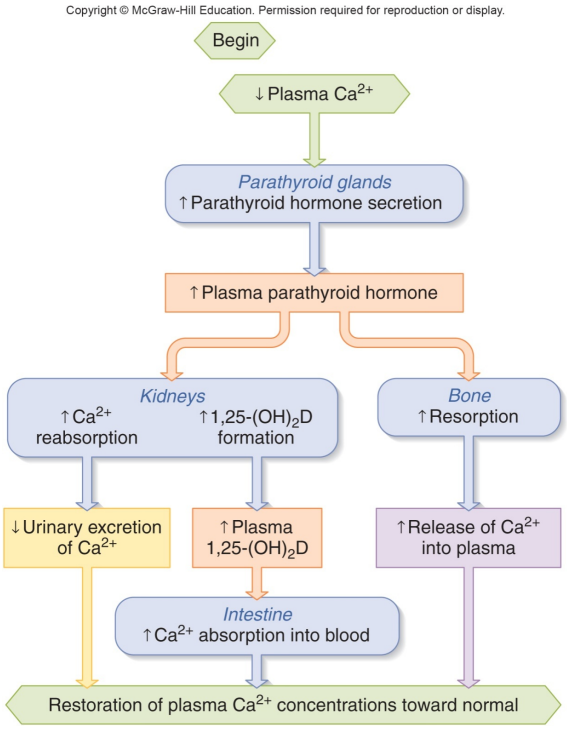
In a study on non-cycling older adults, participants who undertook high amounts of physical activity both as a kid and currently, compared to those who only exercise currently have similar BMDs [7]. This suggests that exercise later in life may be the most important temporal factor when considering BMD over the lifetime. Long-term longitudinal studies, however, would be needed to make these kinds of conclusions in cyclists.

Nutrition and energy availability

Tradition tells us drinking milk makes our bones strong, and it is a justified claim. Appropriate calcium intake has been described as a major factor when considering bone health [2]. The rational is based on the complex role bone has in maintaining appropriate levels of Ca^{2+} in the blood.

99% of your Ca^{2+} is stored in your bones while only 1% is found in your blood. This 1% is the only pool of Ca^{2+} available for a broad spectrum of cellular processes, namely for the muscular and nervous system. Serum levels of Ca^{2+} are tightly controlled and any deviation from normal initiates a rapid response [3]. If there is a decrease in serum Ca^{2+}, the parathyroid gland secretes parathyroid hormone (PTH) [28]. PTH stimulates several mechanisms to increase Ca^{2+} availability, namely stimulating osteoclastic activity. The osteoclasts metabolize bone and

repurpose Ca^{2+} from the skeleton into the blood [29]. In chronic cases of low serum Ca^{2+} levels, the bone is constantly being demineralized and therefore weakened.



Parathyroid hormone's role in plasma calcium regulation – [28]

Low serum Ca^{2+} can be due to low dietary intake of Ca^{2+} rich foods in combination with dermal losses due to sweating [29]. One study suggested that a cyclist could lose 69 mg of Ca^{2+} per hour at 70% of ventalatory threshold (based on power). That equates to about 310mg loss of Ca^{2+} per day depending on duration and intensity of the exercise [30].

In order to limit these losses several studies investigated acute hormonal responses to ingestion of a Ca^{2+} supplement before and during an exercise bout [29, 32]. One study determined that ingestion of Ca^{2+} before and during exercise decreased PTH and bone resportion [32], while the other only found a decrease in PTH [29]. Both these studies however only look within the window immediately after exercise, and don't reflect long-term changes in BMD as a result of calcium ingestion.

No study investigating Ca^{2+} intake in cyclists correlated low Ca^{2+} diets to low BMD [11,14,16,19,31]. Several studies even found that cyclists tended to have higher Ca^{2+} intake compared to controls, but still had lower site specific BMD [16,19]. That being said, one study found that a high fat diet was correlated with increased BMD. Dairy was a major source of fat for many of the cyclists, and although Ca^{2+} was not explicitly tracked, this could have influenced BMD in these cyclists [36]. Similarly, a study on highly trained cyclists found non-statistically significant improvements in BMD with increased Ca^{2+} consumption [16]. All this considered, there is limited evidence to support higher Ca^{2+} intake in order to improve BMD in cyclists.

Another critical player in regulating calcium is vitamin D. As you can see in the figure above, vitamin D assists in calcium absorption in the GI tract [28]. Parathyroid hormone has a key role in

converting vitamin D into its active form, 1.25-(OH_2D [28]. Vitamin D has also been shown to enhance osteoblast activity by assisting in the calcification process [2]. That being said, male cyclists who were given vitamin D and calcium supplements over a 5-month period still saw decreases in BMD [31]. Likewise, serum levels of vitamin D have not been correlated with low BMD in the general population [1]. There is a large body of evidence to support vitamin D intake to prevent osteoporotic fractures [41], however, none look at cyclists specifically.

One longitudinal study of competitive cyclists found that insufficient caloric intake was correlated with low BMD [23]. Similarly, a small study on competitive male and female cyclists found lower BMD in athletes who limited their carbohydrate intake [33]. The second study was particularly small with only 10 participants (6 male and 4 female) [33].

A study on 29 healthy menstruating women found that even small deficits in caloric intake altered osteogenisis. At more extreme caloric deficits, serum N-terminal (NTX) levels were drastically increased, indicating high bone resorption activity [38]. This study only spanned several sessions, but it's hypothesis supports the notion that a deficit of 30 kcal * kg * FFM^{-1}*day^{-1} or greater leads to detriments in bone health [33]. Endurance athletes, specifically runners and cyclists are known to reduce weight with the intention of improving performance, therefore placing themselves in high caloric deficits that may lead to detrimental effects on BMD.

Sex

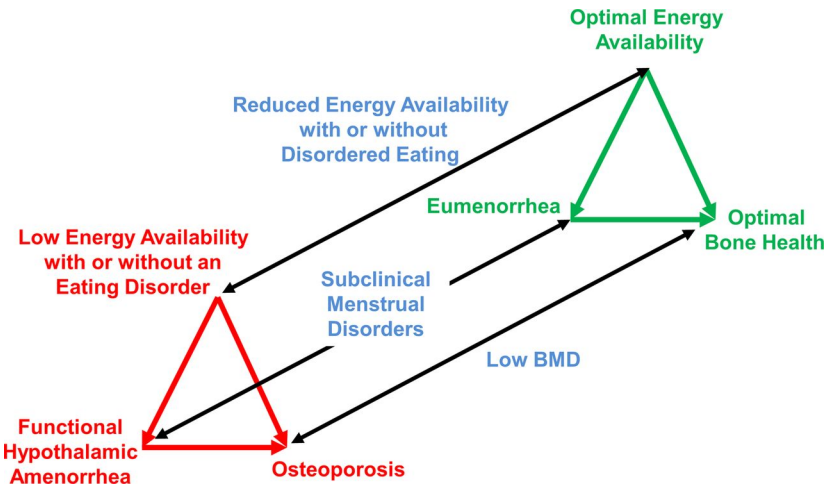
A disproportional 70% of all fractures affect women over 65 years old [1], and one in every six white woman will have a hip fracture during her lifetime [37]. Differences in gender typically come down to hormonal changes at menopause [1], but isn't limited to older women.

None of the studies included in the table compare women and men directly, however several focus on female populations [10,13]. Sherk et al, designed a 1 year longitudinal study with female cyclists (n=14) in the age range of 21 to 41 years. The study found a decrease in hip BMD and BMC (-1.4% and -2.1% respectively), subtrochanteric BMD and BMC (-2.1% and -3.3%), and lumbar spine BMC (-1.1%). The study also found a weak correlation between hormonal contraceptives and low hip and lumbar BMD/BMC [13]. The decreases over a year of training (-1.4%) were similar to a longitudinal study of men cyclists over the same period of time (-1.5%) [11]. The women included in the study, however, were all pre-menopausal, and therefore don't represent post-menopausal women undergoing greater hormonal stresses.

During menopause, women have a rapid decrease in estrogen levels, an important hormonal component of maintaining BMD [1]. Osteoprogeterin (OPG) is a natural antagonist of osteoclast formation through the RANKL pathway. Estrogen promotes OPG activity and therefore osteoclast behavior increases during this time period [2]. As a result, risk of fragility fractures increases at menopause and continues to increase gradually until the age of 75 [1].

Although hormonal changes are notable in women at older ages, hormonal imbalance can occur at any age. Most profound is the female athlete triad, "a disorder best described as the relationship between low energy availability… menstrual dysfunction and decreased bone mineral density" [39]. Energy deficiency may be a result of an eating disorder such as anorexia and bulimia. This presents with a much more complicated physcosocial aspect [40]. This is demonstrated as a spectrum (not an absolute), depicted by the figure below [40]. Although these components don't have to present in a particular order, energy availability is often the precursor to menstrual status and bone health [39]. Low caloric deficits disrupt gonadotropin-releasing hormone (GnRH), luteinizing hormone (LH), and estrogen [39]. As discussed earlier, the alteration of hormonal

balance has a direct relationship to bone health. This reinforces the importance of proper caloric and nutritional intake, especially in people who are exerting themselves at a high level, such as cyclists.



Female athlete triad spectrum – [40]

Conclusion and recommendations

It's been clearly demonstrated that bone health should be a concern for competitive cyclists who spend a large amount of time training for their sport. It seems that higher competitive levels, larger training volumes, and lower caloric intake are major factors when considering BMD. Cyclists are at particular risk of low BMD in the hip and spine, common locations for fragility fractures in older adults.

High velocity crashes are a risk in cycling and therefore subsequent trauma to the skeletal system is not uncommon. Although fractures can occur in healthy bone, lower BMD can increase the severity of the trauma. Likewise, bone health later in life could be altered based on current activity selection. It's important to know that discovery of osteoporosis is typically a result of a fracture, at which point the person is at a much higher risk of a subsequent event [1]. Master cyclists and post-menopausal women in particular should be aware of these typical bone progressions and be screened as such.

There are two easily controlled aspects in training that any cyclist can include to improve bone health. Firstly, include weight training or running/jogging into your training routine two times per week and secondly balance caloric intake with energy expenditure. Weight training is typically unappealing to cyclists due to the hypertrophic mass gains and lack of training specificity. That being said, multiple studies have demonstrated performance gain with weight training [34,35], suggesting the ability to reach multiple goals through the intervention.

Contrary to popular belief, Ca^{2+} did not play a large factor in BMD with cyclists. That being said, Ca^{2+} was rarely controlled in these studies, relying heavily on subjective feedback from subjects. One study found non-statistically significant improvements in BMD with increased daily Ca^{2+} intake. Similarly vitamin D had no apparent positive results in cyclists [31]. That being said, a meta-analysis including 30,970 participants found that vitamin D plus calcium supplementation

resulted in a 15% total fracture reduction (30% at the hip) in middle aged to older adults [41]. As such it would be safe to include the daily-recommended values for Ca^{2+} (>1000mg) and vitamin D (>400mg) in your diet [41]. Similarly, making sure the body has enough energy to undertake high volume training could be a major factor in improving BMD and hormonal balance. Other risk factors within a cyclist's control include limiting alcohol consumption and smoking.

Limitations

Certainly there are limitations to this review. Firstly, although many studies have come out since the turn of the millennium, the quality of these studies are lacking. Low populations, cross-sectional design, and poorly controlled variables limit the ability to extrapolate causation. The studies that do use longitudinal designs [23,11,8,12,13], however, all suggest that over time cyclists lose BMD. Time spans ranged from a competitive season [23] to seven years [Nichols]. Studies that included quantitative computed tomography [15,13,26] gave a more holistic view of bone composition as compared to dual energy x-ray absorptiometry, as DEX can only estimate BMD based on 2D imaging. Results from pQCT studies were mixed but heterogeneity of cycling discipline limits comparisons. Secondly, this review was by no means systematic to the standards of published material. There was no systematic appraisal of literature sited nor an *in depth* analysis of available data. Lastly, it isn't within my scope to recommend detailed dietary plans nor appropriate medicinal interventions. As such, it's important that you consult with your PCP about bone health if you relate to any of the risk factors it may be beneficial to get a screening. Likewise, my own level of understanding regarding the depth of much of the material is a limitation in itself. Although I do believe I've learned a lot, each study leads to another, initiating an endless cycle of detail difficult to master. That being said, I expect to continue advancing my understanding on the topic and will update this page as I go along.

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