Fragile bones of elite cyclists: to treat or not to treat?

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VIEWPOINT

Fragile bones of elite cyclists: to treat or not to treat?

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Accumulating evidence suggests that most elite cyclists have lower bone mineral density (BMD) values when compared with their nonelite counterparts (1) or sedentary young males (2, 3). This raises the question whether these ostensibly healthy athletes have a higher acute bone fracture risk and a higher risk of osteoporosis and associated comorbidities later in life. Although treatment of low BMD seems warranted in elite cyclists, the benefits of treatment for health and performance in this population remain to be established. In this viewpoint, we describe the etiology and consequences of impaired bone health in elite cyclists and discuss the need for interventions to optimize bone health in this unique population.

IMPAIRED BONE HEALTH IN ELITE CYCLISTS: WHAT ARE THE CAUSES?

The cause of impaired bone health in elite cyclists is likely multifactorial. Lack of mechanical loading of the skeleton is an important factor contributing to impaired bone health in elite cyclists (4). Elite cyclists perform extremely high volumes of exercise training and competition (20–30 h/wk; 500–1,000 km/wk), spending a large part of their days on a bike. As the recovery periods are largely spent in a seated or supine position, these cyclists generally obtain insufficient robust osteogenic stimuli throughout daily life.

Low energy availability (LEA) and low body mass are also implicated in the compromised bone health of elite cyclists. Indeed, male and female elite cyclists have been identified as a population at risk for LEA (5, 6), which may eventually lead to the relative energy deficiency in sport (RED-S) syndrome. LEA can be partly attributed to extremely high energy demands for long periods, which may even exceed 30 MJ/day during multistage races (7). Energy intake may also be purposely low when aiming to reduce body mass to enhance the power-to-mass ratio (8). Furthermore, LEA has a major impact on the endocrine system, affecting key hormones that regulate bone metabolism (9).

Another factor that may be involved in low BMD in elite cyclists is dermal calcium loss through sweating, which can be as high as ~150 mg/h (10). In response to dermal calcium losses, the parathyroid gland will release the parathyroid hormone (PTH), which activates demineralization of bone tissue to prevent or attenuate a decline in serum calcium levels. Chronic activation of this mechanism may contribute to low BMD in elite cyclists (11), although the impact of dermal calcium loss in calcium homeostasis has also been challenged recently (12).

It can also be speculated that chronic exercise stress is implicated in impaired bone health in elite cyclists. Although research on this topic is lacking, there is some evidence to suggest that chronic inflammation (13) and elevated cortisol levels (14) are related to bone loss, albeit in nonathletes.

It can be argued that the use of glucocorticoids, as a treatment for musculoskeletal injuries, asthma, and exercise-induced bronchoconstriction, may also contribute to low BMD. However, it should be noted that the use of systemic glucocorticoids seems rare in modern elite cycling, which is also evidenced by a steady decline in "adverse analytical findings" due to glucocorticoid use over the past 2 decades (15). Although inhaled glucocorticoids may be used by some elite cyclists for the treatment of asthma or exercise-induced bronchoconstriction (16), their systemic bioavailability (17) and impact on BMD (18, 19) seem rather limited. Taken together, we believe that the potential contribution of glucocorticoids to the decreased BMD in the current generation of elite cyclists is likely to be negligible.

IMPAIRED BONE HEALTH IN ELITE CYCLISTS: WHAT ARE THE CONSEQUENCES?

Short-term consequences of low BMD in athletes include an increased risk of stress fractures and traumatic bone fractures (5). Stress fractures, however, seem very uncommon among elite cyclists due to the minimal bone stress during cycling. Traumatic bone fractures, on the other hand, are highly prevalent among elite cyclists due to the considerable risk of crashes during training and competition. In this regard, Haeberle and coworkers (20) showed that fractures as a result of crashes were the most common reason for withdrawal during the Tour de France between 2010 and 2017.

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Moreover, half of the cyclists with fractures underwent surgery (20), emphasizing the importance of this problem. Crashes, however, are inherent to cycling races, and it remains to be established whether stronger bones reduce the risk of bone fractures due to crashes.

An important long-term consequence of low peak bone mass in elite cyclists could be an increased risk of bone fractures later in life. It has been proposed that a high peak bone mass during early adulthood is the single most important factor for the prevention of osteoporosis with aging (21). An increase in peak bone mass of 10% has been estimated to delay the onset of osteoporosis by 13 years (22), thereby emphasizing the necessity for healthy bones in young adulthood. However, the progression and/or regression of impaired bone status during and after the cyclists' active career remain(s) to be established, and no (anecdotal) evidence is available that indicates a higher prevalence of bone fractures in retired elite cyclists.

The implications of poor bone health for performance should be considered as well. RED-S syndrome, which is often associated with low BMD, has been linked to impaired exercise performance (5). However, when low BMD occurs without other features of RED-S syndrome, there is no direct evidence to assume that cycling performance will be affected. Nevertheless, given the function of bone in hematopoiesis, and the emerging evidence regarding bone-muscle cross talk (23), it should be realized that the importance of healthy bones may extend well beyond bone fracture risk alone.

IMPAIRED BONE HEALTH IN ELITE CYCLISTS: CONSIDERATIONS FOR TREATMENT

Although oral bisphosphonates are effective in increasing BMD and reducing the risk of bone fractures in men with osteoporosis (24), we feel that pharmacological treatment should be the last line of defense, especially in young athletes. The impact of exercise and nutritional interventions to increase BMD has been reported extensively, particularly for older adults and postmenopausal women (25, 26). To our knowledge, no exercise and/or nutritional interventions aimed at increasing BMD have been documented in elite cyclists. Possible interventions should result in clinically relevant increments in BMD, without interfering with training targets and cycling performance.

Resistance exercise training and impact training (e.g., jumping or bounding) are generally prescribed as the more effective exercise strategies to increase BMD (27). Although resistance exercise training may support cycling performance, many elite cyclists are afraid of potential negative effects of resistance-type exercise training on body mass and cycling performance (8). Impact training is likely more effective than resistance exercise training (28) and may interfere less with the adaptation to endurance training (29). In support, daily short bouts of high-impact jumping exercise have been shown to increase BMD (30), making this a possible intervention to integrate into an elite cyclist's training program. It is unknown, however, if such a low-dose osteogenic stimulus outweighs the deleterious effects of elite cycling on bone health.

Energy availability, calcium, vitamin D, and protein are among the major nutritional factors that should be considered (31). Careful assessment of nutritional intake and regular blood testing (for vitamin D) are needed to assess whether cyclists have an inadequate energy and calcium intake and/or vitamin D status. An adequate calcium intake is needed for bone mineralization, with adequate serum 25-hydroxyvitamin D levels promoting the absorption of calcium from the gut. Deficiencies should be addressed, whereas supplementation above intake recommendations seems to provide little (32) or no (33) benefit for bone health. Being the most abundant protein in the bone matrix, collagen could be an interesting target for novel nutritional strategies as well. Indeed, 12 mo of daily supplementation with collagen has been shown to positively affect BMD and markers of bone metabolism in postmenopausal women (34), whereas a combination of gelatin supplementation with jumping exercise has been shown to increase the (bone) collagen synthesis marker N-terminal propeptide of type I collagen (P1NP) in young males (35).

It is clear that both exercise and nutrition have the potential to increase BMD in elite cyclists, but more work is needed to establish their efficacy and effectiveness in this specific population.

IMPAIRED BONE HEALTH IN ELITE CYCLISTS: TO TREAT OR NOT TO TREAT?

The answer to the question whether low BMD in elite cyclists should be treated may not be as clear-cut as initially thought. It is concerning that elite cyclists have a low bone mass at an age where peak bone mass is normally achieved. However, the potential short- and long-term consequences of impaired bone health in terms of health and performance are unclear in this specific population. Although BMD can generally be increased by exercise and/or nutritional interventions, the feasibility, effectiveness, and potential side effects of such interventions remain to be established in this population. The ultimate piece of evidence would reveal the relationship between bone health and the incidence of traumatic bone fractures during and after the active career of elite cyclists. Until more evidence becomes available, all elite cyclists and their supporting staff should at least be aware of this issue and carefully consider the available treatment options for low BMD.

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AUTHOR CONTRIBUTIONS

L.H. and J.W.v.D conceived and designed research; L.H. and J.W.v.D. drafted manuscript; L.H., P.K., M.H., R.K., A.E.J., L.J.v. and J.W.v.D. edited and revised manuscript; L.H., P.K., M.H., R.K., A.E.J., L.J.v. and J.W.v.D. approved final version of manuscript.

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