

# Can exercise prevent osteoporosis?

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## Abstract

Commonly used definitions of osteoporosis rely upon the measurement of bone mass or bone mineral density and regard the difference between osteopenia and osteoporosis as gradual. An alternative definition has been proposed by Harold Frost, suggesting that osteopenia is the bone's physiological response to disuse. On the contrary, true osteoporoses imply the bone's inability to adapt to the loads imposed on them by their habitual mechanical usage. As a consequence, fractures occur with no or very little trauma in osteoporotic, but not in osteopenic bones. There is now ample evidence that mechanical stimuli can increase strength. Accordingly, exercise, in particular some new forms of it that involve high strain rates, seems to be preventing bone loss and possibly also induces increases in bone mass even at older ages. Hence, exercise may ameliorate osteopenia in the sense of Frost's definition. However, exercise must be feared to facilitate rather than to ameliorate the occurrence of true osteoporoses, e.g., due to microdamage accumulation. This is in sharp contrast to the general 'understanding'.

**Keywords:** Sport, Osteopenia, Aging, Material Fatigue, Bone

## Definition of the problem

Osteoporosis is a condition in which bones fracture without a major trauma. In a strict sense, therefore, osteoporosis can be diagnosed only *a posteriori*, i.e., after the fracture has occurred. This is obviously unsatisfactory for both sides, the patient and the doctor. As an attempt to predict an increased risk of fracture, osteodensitometry has been developed. It is fundamental to bear in mind that this is an attempt to diagnose osteoporosis *a priori*, i.e., before the fracture occurs.

Conventionally, osteoporosis is diagnosed by dual X-ray absorptiometry (DXA). This is problematic because it yields only bone mineral content (BMC) and areal bone mineral density (aBMD), but disregards the structure, geometry and mechanical properties of bone<sup>1</sup>, all of which are known to affect bone strength and to change with age. However, clinicians and many scientists rely upon DXA measurements in succession of a WHO study group<sup>2</sup>, which defined osteo-

porosis as a decrease in bone mass or in bone mineral density, and a deterioration in bone 'structure'. By the same definition, osteopenia is regarded as a precursor of osteoporosis that differs from osteopenia in quantitative, but not in qualitative terms.

An alternative view has been proposed by H.M. Frost, who discerns between physiologic osteopenia and true osteoporoses<sup>a</sup> (see Table 1 and p.302 ff.<sup>3</sup>). Here, osteopenia denotes a state in which bones are weak due to a lack of mechanical usage. Fractures are therefore more likely as a result of trauma, but the bones do resist their habitual loads. Conversely, true osteoporoses are characterised by the inability of bones to adapt their strength to the patient's voluntary mechanical usage. As a consequence, fractures occur with very little trauma or no trauma at all.

The difference between those two definitions is succinct. Whereas the conventional view considers bone mass (and sometimes bone strength) as a predictor of fracture, the Frostian approach distinguishes between primary bone disorders (=osteoporoses) and secondary bone problems (=osteopenia). A similar distinction has successfully been established in the field of paediatric bone disease<sup>4</sup>. The potential superiority of the Frostian concept is not only aca-

The author has no conflict of interest.

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Accepted 25 January 2006

<sup>a</sup> Importantly, H.M. Frost anticipated that there may be different kinds of osteoporoses, each with its own pathogenetic mechanism.

	<b>Conventional view</b>	<b>Harold Frost's view</b>
<b>Based on</b>	WHO study group <sup>2</sup> , Harrison's Principles of Internal Medicine <sup>46</sup> , and many others	Practical experiences as an orthopaedic surgeon <sup>3,47</sup>
<b>Osteopenia</b>	BMD is only slightly lower than average	Bone strength is lower than in other people, but appropriate for the mechanical usage in that individual
<b>Osteoporosis</b>	BMD is significantly lower than average	Bone strength is inappropriate for the mechanical usage; fractures evolve with very little trauma

**Table 1.** Different views and definitions of osteoporosis/osteopenia. Please note that Frost's definition focuses on bone strength (rather than on bone mineral density=BMD), and it sees a qualitative (rather than a quantitative) difference between osteoporosis and osteopenia. The conventional view is currently bound to change, but is still governing medical practice.

demic. Attempting to understand the pathophysiological determinants of brittle bones, it constitutes a rationale to guide our research and therapeutic decisions.

## Exercise and osteoporosis

How do true osteoporoses (in Frost's sense) relate to exercise? The mechanical stimuli that come with the exercise have at least two opposing effects upon bone. As a tissue, strains that exceed a certain threshold induce modelling and thereby adapt the bone's strength to the loads habitually applied<sup>5-9</sup>. This constitutes a negative feedback loop which is commonly referred to as the 'mechanostat'<sup>10</sup>.

On the other hand, bone as a material will be weakened by repetitive strains, which cause microdamage, i.e., material fatigue. As a result, fatigue fractures are not uncommon in recruits, athletes, or in race horses<sup>11,12</sup>. It is thought that remodelling, i.e., the replacement of old material with newly formed bone acts as a repair mechanism in order to prevent microdamage accumulation<sup>13,14</sup>.

It is currently unknown whether the efficacy of that repair mechanism is affected by age. However, it has been argued that material fatigue plays an important role in hip fractures in the elderly<sup>15</sup>, and Frost suspected that the inability to repair microdamage is the hallmark of at least one kind of true osteoporosis (p. 249<sup>3</sup>). If this is so, and if, indeed, this osteoporosis becomes increasingly prevalent with age, then, quite in contrast with the conventional view, exercise would facilitate rather than ameliorate the occurrence of osteoporotic fractures. Other types of true osteoporoses may exist, in which the mechanostat malfunctions for other reasons. Again, it is hard to see how, under such conditions, exercise would be beneficial.

## Exercise and osteopenia

In the following, I will briefly discuss some controversial and recent evidence as to whether specific kinds of exercise are likely to prevent physiological osteopenia in the elderly (for more detailed reviews see<sup>16-20</sup>). Unfortunately, many of these studies are focused on areal bone mineral density

(aBMD). This is error prone, since the bone geometry may change independently of bone mass. It is well documented, for example, that the diameters of long bones get larger as we age<sup>21</sup>. Consider that, during such an enlargement, the bone mineral content remains unchanged. As a consequence, the section modulus and thus bending strength of the bone would increase, but aBMD would decrease.

As the differential effects of exercise and ageing upon bone mass and bone geometry are unknown, increases in aBMD in response to exercise may therefore be judged to reflect a modelling response, but they do not necessarily indicate that bone strength has increased.

In aerobic exercise, the muscles power output is comparatively low, and the developed forces are small. Endurance running was formerly thought to be detrimental to bone health. This notion was based on the observation that, in young to middle aged men, the bone mineral content of the spine and of the hip was 20% lower than in their age peers, and bone turnover was found to be low<sup>22</sup>. However, this ought to be seen as an adaptation of the bones to different loads and body types<sup>23</sup>, rather than a negative effect of running. Indeed, if controlled for stature, male endurance runners appear to have higher aBMD values in the spine, hip and calcaneus than sedentary people<sup>24</sup>.

These benefits of endurance running appear to be effective also at old age, as suggested by a longitudinal study of 54 Master runners aged between 40 and 80 years<sup>25</sup>. During an observational period of almost 5 years, values of aBMD at the spine and the hip were stable, although aerobic fitness declined significantly. Although not understood in detail, the latter alludes to the relative unimportance of aerobic fitness in determining bone strength. Little is known as to other types of aerobic exercise and bone strength at old age.

Although resistive exercise is known to increase bone mass at a young age<sup>26-28</sup>, its efficacy in old age is controversial. Some studies report an increased aBMD of the spine as a response to resistive exercise<sup>29,30</sup>, others found no such effect<sup>31,32</sup>. Even in the studies with a positive outcome, the effects were rather small (1-2%). It seems fair to say, thus, that although effective at a young age, resistive exercise is probably not overtly attractive to increase bone strength in old age.

There is quite some evidence that high impact exercise, such as gymnastics, is particularly beneficial to increase bone mass. In young women, accrual of bone mass was significantly greater in well-trained gymnasts than in either cyclists or swimmers<sup>33</sup>. Interestingly, adding impact to resistive exercise seems to enhance its effects upon bone<sup>34</sup>. With two groups of postmenopausal women performing resistive exercise with the same weight loads, number of sets, number of repetitions etc., those who worked at a greater speed were able to maintain their aBMD at the spine and the hip, whereas the others lost about 2%. This again highlights the importance of strain rate as a signal for adaptive processes in bone.

Recently, vibration exercise has been proposed as a new type of exercise. There are two different 'camps' propagating vibration exercise for the improvement of bone health. One 'camp' suggests that vibrations of low magnitude, but high frequency would elicit an osteogenic response by direct mechanisms in the bone<sup>35</sup>. It is maintained that, because of the small strains involved, these benefits come without the risk of mechanical damage to the bones. The validity of this concept has been shown in sheep, where vibration of the hind limb elicited an increase by 34% in trabecular density in the proximal femur<sup>36,37</sup>. That kind of vibration was able to increase the trabecular bone mineral density in the proximal tibia in children with cerebral palsy who, without treatment, would have lost bone<sup>38</sup>. Furthermore, another recent study suggests benefits of whole body vibration exercise in postmenopausal women<sup>39</sup>. Good compliance provided, the women had a benefit of 2.2% in areal bone mineral density at the femoral neck and of 1.7% at the spine by the treatment.

The second 'camp' considers the important role that muscles seem to play for the accrual and maintenance of bone<sup>40-42</sup>. In consequence, 'high magnitude vibration' exercise is suggested to strengthen the bones by eliciting forceful muscle contractions. In rats, such vibrations were able to partly prevent the ovariectomy-induced bone loss<sup>43</sup>. In postmenopausal women, areal bone mineral density was observed to increase by 1% in response to a six-month program of vibration exercise, paralleled by increases in muscle strength<sup>44</sup>. However, these changes were comparable to a control group, which practised conventional resistive exercise, without vibration. Finally, in the Berlin bed rest study, vibration in combination with resistive exercise was able to almost completely prevent the bone loss induced by bed rest in young healthy males<sup>45</sup>.

## Conclusion

It is currently unclear which kind of exercise is most effective to increase bone strength. Recent evidence suggests that osteogenic responses by exercise can be elicited even at older age. This has been denied in the past. Exercises that involve high strain rates seem to be more effective than others. However, all these benefits are comparatively small. It may therefore be much easier to prevent bone loss throughout life,

potentially by exercising, than to increase it in the second half of life.

Hence, it seems that exercise may ameliorate or even prevent osteopenia as defined by Harold Frost. As explained above, that is most probably not the case for true osteoporoses.

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