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Short Review

Resistance Training is Recommended for those at Risk for Osteoporosis and Alzheimer's Disease

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Abstract

The goal of this brief review is to highlight the relationship between osteoporosis and Alzheimer's Disease (AD), using weight-lifting exercises as key to preventing, or at least, forestalling both. At the molecular level, both diseases share certain etiologies, such as β-amyloid and hyperphosphorylated tau protein. At the behavioral level, consistent resistance training maintains bone density and wards off cognitive decline, which may start with clinical depression, but can ultimately degenerate into AD. There is, therefore, strong argument for beginning weight training early in life, at adolescence, and maintaining it consistently throughout, especially for those who are genetically predisposed for either or both diseases.

Keywords: Osteoporosis; Alzheimer's disease; Depression; Weight training; Resistance training

Incidence of osteoporosis with age

Data from the following 12 industrialized countries were evaluated: USA, Canada, UK, Germany, France, Italy, Spain, Australia, Japan, Sweden, Finland and Denmark. Data for hip fractures showed that women had the highest incidence in Sweden, Finland and Denmark, ranging between 4.7 to 5.8 per every 10,000 women 50-59 years of age; 15.5 to 16.8 in women 60-69 years of age; and 115 to 130 in women over 70 years of age. In this same age range, women in eight of the countries the rates were much lower – 69.7 to 89.9 per 10,000 women, but were lowest in Spain at only 58.7 [1].

Men had similar, but lower, incidence rates as women: Sweden, Finland and Denmark had the highest rates at 7.9 to 11.9 in men 60-69 years of age; from 70-80 years of age, the rates increased to 24.9 to 36.7; then, for men 80 years old or more, rates increased to 93.2 to 119.6, which was twice as high as those in the UK, Germany and Italy, which ranged from 54.9 to 58.8 [1].

Incidence of Alzheimer's Disease (AD) with age

As with osteoporosis, the incidence of AD also accompanies age. During the age of mid 60s, rates tend to triple before age 64, double before age 75 and decline significantly to 1.5 times at around 85 years of age, meaning that the increase in incidence rates slows down as age increases, although the incidence rates themselves show no signs of declining [2]. Nino et al. [3] found similar trends: ages 60-69, incidence was 60.6%; for ages 70-79, incidence was 30.6%, for ages 80-89, incidence was 8.4%; and for age 90 or more, incidence was 0.4%, all culminating in the median age of 67 [3]. Likewise, AD also tends to be gender-specific, with women and men showing a 58% and 42% frequency, respectively, of AD [3].

Weight-lifting (resistance) exercise and osteoporosis

Chief among the non-pharmacological interventions is physical activity, particularly any weight-bearing exercise that stresses the muscles, bones and joints. In addition to building bone mineral density, such exercise, especially as a preventive measure [4,5], helps prevent the occurrence of falling, maintaining balance, posture and mobility [6,7]. Indeed, without such exercise, other interventions, such as hormonal and calcium supplements alone cannot maintain bone mass [8]. Despite the fact that it may indeed make intuitive sense that weight-bearing exercise would have a positive effect in the prevention, or at least, mitigation against osteoporosis, the data in the literature are neither so definitive nor robust, if it has any effect at all.

Weight-lifting exercise may decrease the risk of osteoporosis in younger women by increasing bone mineral density and minimizes the loss of bone density in postmenopausal women [9,10]. There is evidence that such strength training has either a positive effect on bone mineral density in young athletic [11], premenopausal [12] and postmenopausal [13-16] women or no effect at all in premenopausal [17] and postmenopausal [18,19] women. Earlier, Layne and Nelson [20] reported the same pattern of discrepancies in a short review. Lack of standardized protocols, length, type and intensity of exercise, the age and type of human subjects, as well as differences in how bone density is measured may account for many of the results both earlier [20] and more recently (see above).

Educating patients about osteoporosis prevention

In light of the preceding section, there is at least the indication that preventing osteoporosis should begin when the person is relatively young, even as young as college-age women [11]. As with most diseases, the best treatment is prevention [9]. A healthy lifestyle, making smart choices throughout life, includes regular exercise [9], as well as the judicious use of medications, such as glucocorticoids, taken for other medical problems, but that can exacerbate or increase the risk of osteoporosis [21,22]. Because their physicians prescribe these medications and have access to their medical history, the former [23,24], specifically,

orthopaedic surgeons, can play an active role in educating and, therefore, treating, or even preventing osteoporosis [25]. Thus, if glucocorticoid-like drugs are required to treat some other medical problem (respiratory, gastrointestinal, etc.), some other prophylaxis calcium supplement (e.g., calcitriol) should also be prescribed and concomitant bone mineral density scans is routinely performed to monitor osteoporosis risk [21].

Relationship between osteoporosis and Alzheimer's Disease (AD)

Although the literature indicates a glaring inconsistency among studies in whether resistance exercise is indeed beneficial for preventing osteoporosis (see above), and among studies that do report a positive relationship between bone mineral density/strength and osteoporosis, the gains in bone strength are relatively modest, increasing by about 2-3% [8] or even less (e.g., 1.5%-1.6% [14]; 1.54% [15]; 1.4% [16]). In fact, these same investigators even found negative effects on bone mineral density, depending on the specific bone or joint measured. Thus, there may be little motivation to exercise with weights, particularly if the patient has no interest in such activities.

One type of such patient that may find little-to-no motivation to exercise is those suffering from some type of mood disorder, such as clinical depression. There is ample evidence that depression is a strong predictor for AD [26,27], as well as other peripheral diseases [26], such as osteoporosis. Indeed, it makes intuitive sense that someone who is so depressed to the point that no form of treatment has significantly helped (yet), they will tend not to exercise, making their depression worse, further increasing their risk for AD later (or maybe even earlier) and osteoporosis. But exercise is not only beneficial for helping to forestall or even prevent osteoporosis (see above). Over the past 2-3 decades, there is mounting evidence that exercise is also beneficial in enhancing cognition [7,28-31], preventing its decline and preventing development of dementia [28,30,32-37] and AD [38-44] and boosting mood [45]. Specifically, weight-bearing, rather than aerobic, exercise would be more beneficial for the elderly [46].

At the molecular level, there is likewise mounting evidence that many of the biochemical hallmarks of AD are also present in osteoporosis, which may help explain their co-morbidity [47-52]. One of the molecular hallmarks of depression, indeed, that upon which antidepressant medications are based, is that the levels of synaptic circulating neurotransmitters (norepinephrine, serotonin, dopamine) are lower than in non-depressed individuals [53]. Because depression is a risk factor for AD [26,27], it has been reasonable to hypothesize that both diseases share many molecular causes, such as decreased serotonergic neurotransmission [54]. Recently, in a mouse model of AD, with hyperphosphorylated tau (a putative hallmark of AD), there was a 70% decrease in tryptophan hydroxylase-positive neurons in the dorsal raphe, where much of the serotonin is produced and from where structural integrity of the skeleton is regulated; such decrease in serotonin therefore, is highly correlated with the observed decrease in bone mineral density in these mice [54]. In addition, because the nervous and skeletal systems are derived from the same germ layer, it is not surprising that during their development, they are regulated by the same molecules. Thus, high levels of dickkopf-related protein-1 are a risk factor for AD [50]. And expression of osteocytic β-amyloid peptides (another putative hallmark of AD), which increased osteoclastic activity, was highly and negatively correlated with bone mineral density [55]. Unexpectedly, however, frequency of the Apo E4 allele (a genetic risk factor for AD) has not been found to correlate with bone mineral density; but this finding may be due to racial polymorphisms of their test subjects [56]; specifically, this lack of correlation between Apo E4 genotype and bone mineral density was conducted in Chinese subjects, whose Apo E4 prevalence was only 7%, whereas in, say, Caucasians, it is nearly 15% [56].

Previously, Koscak [46] indicated that resistance exercise may be more beneficial than other forms in preventing or forestalling AD-associated cognitive decline. Likewise, Beherer [7] and Koscak [46] and several others (see above, [13-19]), indicate that resistance exercise may be a viable alternative for people for whom aerobic forms of exercise may be medically inappropriate. The exercise program, therefore, should be tailored specifically to a person's medical history, current health and strength/fragility.

Resistance training, osteoporosis and Alzheimer's Disease

Extensive search of the literature for studies aimed at discovering the effects of resistance training on both osteoporosis and AD came up surprisingly very short, revealing only one study [57]. These investigators [57] found that in patients with AD and osteoporosis in the femoral neck, handgrip strength was significantly lower than in healthy elderly controls (19.4 *vs.* 37 pounds of force). One explanation for the extreme paucity of studies examining all three variables (resistance training/strength, osteoporosis and AD) simultaneously is that the cognitive impairment of AD may prevent the participant from fully understanding the (verbal) instructions of the experimenter, thereby providing a confounding variable: It is therefore not possible to know if the lack of grip strength is because of less than full understanding of the experimenter's instructions or if it is truly the result of some physiological deficit, such as compromised motor control, because of the AD. There is, therefore, a tremendous need to perform similar studies with other populations. Although the study by Ayhan [57] used a moderate-sized samples (n=56 healthy elderly controls; n=75 AD with osteoporosis), additional studies should be performed with other measures of strength training, exercise protocols and bone density locations, despite the confounding problem alluded to above.

Conclusions

In this short review, we have provided much evidence that to prevent or at least forestall osteoporosis and AD, physical exercise is key, and the earlier in life that exercise becomes a lifestyle choice and daily habit, the greater reserve capacity is accumulated, and the more healthy the individual will be during the later years. Although any form of exercise, provided that it is of sufficient duration, intensity and frequency (regularity), can have a significant impact in forestalling AD, it is resistance training (weight lifting) that plays a greater role in forestalling osteoporosis. Thus, building and maintaining bone strength throughout life also means building neural strength and enhancing synaptic connections and plasticity.

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