Editorial

Senile osteoporosis is associated with disc degeneration

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Osteoporosis and disc degeneration of the spine are common conditions that primarily affect the elderly with significant impact on the quality of life. A number of studies reported that higher lumbar spine bone mineral density (BMD) was associated with disc degeneration (1-5). However, other studies reported that osteoporotic patients have more severe disc degeneration (6,7). Some authors suggest that osteoporosis would possibly delay disc degeneration because of an increase in intradiscal nutrient diffusion and a decreased endplate resistance and decreased intradiscal strain due to the low quality of the bone (8,9). On the other hand, it is also proposed that osteoporosis may be an etiological factor in the development of lumbar disc degeneration with osteoporosis inducing loss of vertebral height, leading to instability, facet arthrosis, and disc degeneration (7). After many years' research, whether osteoporosis promotes or protects disc against degeneration still remains debated in the literature (8-12). Hereby we argue that current evidences suggest senile osteoporosis promotes disc degeneration.

Earlier studies reported osteoporosis protects against lumbar disc degeneration which was assessed by radiograph (1-5). Radiographic signs of disc degeneration typically include the presence and severity of anterior osteophytes, end plate sclerosis, and disc space narrowing. With visual MRI based approaches such as the commonly used Pfirrmann's 5-level grading and the detailed Griffith's 8-level grading, disc space narrowing is again incorporated as an integral sign of disc degeneration (13,14). However, osteoporosis is associated vertebral height loss, particularly vertebral middle height loss, which allows the expansion of the disc vertically (15). Thus the osteoporotic spine may less likely be graded as having disc space narrowing, and thereof, less likely be graded as having disc degeneration.

Another methodological issue is where and how to measure BMD. The prevalence of disc degeneration and facet arthrosis increases with aging, such that by 60 years of age, 60–80% of people have osteophytosis, disc narrowing, and/or facet joint arthrosis (16). Degenerative changes lead to artificially higher DAX-based areal lumbar BMD measurement due to marginal osteophytosis, trabecular thickening, subchondral sclerosis, and facet joint arthrosis (17,18). Narrowed disc spaces can lead to a higher areal BMD reading (19). In the elderly population, lumbar disc spaces are more likely to be narrower when vertebral areal BMD is higher (20). Osteoporosis in its most common form is a systemic disease. In this regard, hip densitometry can be a more reliable and better representative systemic BMD. However, it can also be argued that it is the local lumbar BMD that really matters when investigating the inter-play whether the higher lumbar BMD associated biomechanical alterations may contribute to disc degeneration. Quantitative computerized tomography (QCT)-measured vertebral body trabecular BMD may overcome the areal BMD measurement artefacts caused by spinal degeneration. However, with Griffith's visual 8-level MRI grading, a small study of 48 elderly males with QCT measurement of lumbar spine BMD (L1L2) show a trend of more severe disc degeneration being weakly associated with higher trabecular BMD value (20). Muraki *et al.* (19) analyzed 630 women aged ≥60 years and found that the scores for degenerative spinal diseases were correlated with lumbar spine areal BMD, but not correlated with femoral neck BMD. Pye *et al.* (21) analyzed a sample of 500 men and women and examined osteophytes, disc space narrowing and endplate sclerosis at 4 lumbar discs with radiographs,

showed that lumbar BMD increased with increasing grades for all radiographic features in both sexes. When they adjusted disc space narrowing score for age and body mass index it remained significantly correlated with BMD at the spine, but not at the femoral neck. Similar results have also been reported by Salo *et al.* (22).

With Griffith's visual 8-level MRI grading, in elderly female (73.2±4.1 years, n=196) and male (73.5±4.3 years, n=163) subjects and according to lumbar areal BMD, we initially reported that disc degeneration tended to be less severe in osteoporotic subjects compared to osteopenic subjects and in osteopenic subjects compared to normal bone mineral density subjects (20). However, there was no significant relation between hip BMD and lumbar disc degeneration or disc space narrowing both for males and females (23). Furthermore, when the disc dimension and disc volume were quantitatively measured (15), we demonstrated that lower lumbar BMD (osteopenia and osteoporosis) is associated with a decrease in lumbar disc anterior height and posterior height, as well as a decrease in anterior-posterior diameter; however, the middle height of the discs was increased (therefore the disc biconvexity index was increased). The net result is that lower BMD is associated with a decrease of the disc volume (15). On the other hand, for the vertebral bodies, lower BMD was associated with a decrease of vertebral anterior/middle/posterior height, but not vertebral anterior-posterior diameter; lower BMD was associated with a decrease in vertebral volume, and an increased biconcavity index (15). Our 4-year longitudinal follow-up study with radiographs shows osteopenia and osteoporosis are associated with faster disc area decrease in both thoracic spine and lumbar spine, and both for both males and females (24). We can naturally assume that a decreased disc volume (as measured with MRI) and decreased disc lateral area (as measured with radiograph) are biomarkers of disc degeneration. One point worthy noting is that while elderly females overall had faster radiographic disc area loss during the 4-year follow-up than elderly males, for the subgroups of osteoporotic subjects in this study, elderly men and elderly women had similar extent of disc area loss during the 4-year follow-up period. This observation may tentatively suggest that factors associated with osteoporosis and osteoporosis themselves are important drivers for disc area loss and thus disc degeneration. An increase in central disc height without compensatory increase in disc width may potentially have a destabilizing effect on the spine contributing to altered spinal kinematics.

From a pathophysiological perspective, osteoporosis may negatively impact endplate and thereof contribute to disc degeneration. Although disc degeneration has a multifactorial etiology involving age, mechanical, and genetic factors, a final common pathway of decreased nutrition has been proposed (25). Failure to adequately supply nutrients to the disc cells is a major event in the initiation and progression of disc degeneration (26). Osteoporosis can cause endplate thinning and microfracture, which in turn lead to compromised endplate healing, and add calcification and decrease the vascularization in the endplates adjacent to the degenerated discs, which subsequently exacerbates degeneration of the associated discs (27,28). Experimental studies with aging sand rats show that in the osteoporotic spine, endplate sclerosis occurs with aging, which in turn inhibits nutrition supply to the intervertebral discs (29). In bilateral ovariectomy (OVX) rats, osteoporosis and disc degeneration occurs simultaneously, while the increase of calcification and decrease of vascularization of the endplate further contribute to disc degeneration (30-32). In a rat osteoporosis model induced by a combination of OVX and cervical muscle section, osteoporosis was associated with cartilage endplate lesion and greater disc degeneration in cervical spine (33). In a study using female rhesus monkeys, Zhong *et al.* (34) used intra-vertebral injection of pingyangmycin solution to induce slowly progressive disc degeneration. Endplate vascular channel decrease and shrinking, which were more obvious when combined with OVX, was observed. Zhong *et al.* (34) suggested that osteoporosis could promote endplate calcification and further decrease the vascularization of the endplates adjacent to the degenerated discs. Interestingly, in rat studies, alendronate (an antiresorptive agent to treat osteoporosis) administration initiated pre-OVX or three months post-OVX, or salmon calcitonin (a 32-amino acid linear polypeptide hormone used for postmenopausal osteoporosis treatment) administration initiated pre-OVX, not only alleviate vertebral osteoporosis but also protect disc against degeneration (30-32).

On the other hand, two studies related to this topic involving middle-aged subjects have been published (35,36). In a cadaveric lumbar spine specimen study, Wang *et al.* (35) used micro-CT to measure vertebral BMD, and disc degeneration was assessed by discography which was independent of disc height evaluation. They found that no significant association was found between the BMD of the whole vertebra and adjacent disc degeneration. However, when only the vertebral body was considered

(with the posterior elements, such as lamina, facet joints, spinous process, transverse process, excluded), there was a significant association between greater vertebral body BMD and more severe degeneration in the disc cranial to the vertebra. This association remained after further excluding osteophytes and endplates from the vertebral body BMD measurement. It was suggested that the association between higher vertebral body BMD and severer disc degeneration may be a collaborative effect of general factors and local interaction. However, their samples were from 48 white men aged 21 to 64 years with a mean age of 50. In a twins study, Livshits *et al.* (36) studied a total of 908 subjects from a volunteer-based group of healthy Caucasian women (age range 32–77 years, median age: 53.02 years). The four MRI main traits of disc degeneration were scored: disc signal intensity within the nucleus pulposus, disc height measured in the middle of the disc, lumbar disc extension into the spinal canal and anterior osteophytes. They found that the individuals with more advanced degenerative changes in lumbar spine tended to have higher BMD in spine and hip. The association persists after taking other covariates into account and is true for the all four MRI traits studied. Some common genes, for example, polymorphisms of the vitamin D receptor gene, have double-edged effects that contribute to better BMD in the vertebra but also more degeneration in the intervertebral disk (37-40). In middle-aged subjects, when levels of peak spinal loading and bone strength is high, dense vertebral bone can possibly threaten the adjacent discs by increasing pressure in the disc nucleus (11). However, disc degeneration in an elderly population may behave rather differently compared with discs in middle-aged subjects.

To conclude, current evidences suggest senile osteopenia/osteoporosis in both elderly males and females is associated with disc degeneration. This is probably at least partially mediated by endplate degeneration and the associated decrease in nutrient supply to the disc, as well as the altered biomechanical stress. On the other hand, while more evidence is required, in young-middle aged subjects high physiological BMD may be associated disc degeneration (35,36). Due to the confounding effects that low vertebral BMD is associated with a decrease of vertebral middle height and an increase of disc middle height, and degenerative spines have an artificially elevated lumbar areal BMD measurement, and the difficulties in interpreting MRI results, for assessing the association between BMD and disc degeneration meticulous cares should be taken. We suggest the following points to be carefully considered when designing an *in vivo* study or interpreting results:

(I) Is the study addressing (i) young subjects (such as investigating genetic traits)? (ii) middle aged subjects? or elderly subjects? While in younger subjects disc degeneration is often associated with physical injury in males (41), signs of disc degeneration in the elderly can be associated with natural aging (42).

(II) Men and women should be analyzed separately. Menopause both contribute to rapid loss of bone mass as well as accelerated disc generation, and osteoarthritis (43-51).

(III) QCT-based vertebral trabecular BMD or hip BMD is preferred over areal lumbar BMD (52,53).

(IV) To define disc degeneration *in vivo*, we recommend MRI based methods which provide quantification of biochemical composition of disc tissues, such as T2/T1rho/CEST (54-61). However, it should be noted that accurate measurement of disc tissues can be challenging (57,62-64), and a standard approach for segmenting annuls fibrosis and nucleus pulposus should be considered (64).

(V) Ideally, study subjects sample size should be sufficiently large (57) and lumbar disc levels are analyzed individually, as lumbar discs at different levels are under different biomechanical stress (65). It has been noted that, in elderly subject within a defined period of follow-up, caudal lumbar discs had greater lateral area decrease rate than cephalad lumbar discs (23). Adams and Dolan (66) suggested that there are two types of disc degeneration. "Endplate-driven" disc degeneration involves endplate defects and inwards collapse of the annulus, mostly affecting discs in the upper lumbar and thoracic spine, usually associated with compressive injuries. "Annulus-driven" disc degeneration involves a radial fissure and/or a disc prolapse, mostly affecting discs in the lower lumbar spine, and is associated with repetitive bending and lifting. Lower lumbar discs are subjected to greater loading in bending, and so are more susceptible to degenerative changes (including disc prolapse) which arises from bending injuries to the annulus.

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Footnote

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