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REVIEW

Biomechanics of the aging spine

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Abstract The human spine is composed of highly specific tissues and structures, which together provide the extensive range of motion and considerable load carrying capacity required for the physical activities of daily life. Alterations to the form and composition of the individual structures of the spine with increasing age can increase the risk of injury and can have a profound influence on the quality of life. Cancellous bone forms the structural framework of the vertebral body. Individual trabeculae are oriented along the paths of principal forces and play a crucial role in the transfer of the predominantly compressive forces along the spine. Age-related changes to the cancellous core of the vertebra includes a loss of bone mineral density, as well as morphological changes including trabecular thinning, increased intratrabecular spacing, and loss of connectivity between trabeculae. Material and morphological changes may lead to an increased risk of vertebral fracture. The vertebral endplate serves the dual role of containing the adjacent disc and evenly distributing applied loads to the underlying cancellous bone and the cortex of the vertebra. With aging, thinning of the endplate, and loss of bone mineral density increases the risk of endplate fracture. Ossification of the endplate may have consequences for the nutritional supply and hydration of the intervertebral disc. The healthy intervertebral disc provides mobility to the spine and transfers load via hydrostatic pressurization of the hydrated nucleus pulposus. Changes to the tissue properties of the disc, including dehydration and reorganization of the nucleus and stiffening of the annulus fibrosus, markedly alter the mechanics of load transfer in the spine. There is no direct correlation between degenerative changes to the disc and to the adjacent vertebral bodies. Furthermore, advancing age is not the sole factor in the degeneration of the spine. Further study is crucial for understanding the unique biomechanical function of the aging spine.

Keywords Aging spine · Biomechanics · Osteoporosis · Vertebral endplate · Disc degeneration

Introduction

The vertebral column is built from alternating bony vertebrae, interconnected with fibrocartilagenous discs and diarthrodial facet joints. In total 33 vertebrae (7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 4 coccygeal) all conform to a basic plan. With the exception of the atlas and axis, all vertebra are made of an anterior approximately cylindrical vertebral body and an arch composed of paired pedicles and laminae, the latter joined posteriorly forming the spinous process. The arch on either side also features a transverse process, as well as superior and inferior articular processes forming corresponding synovial joints (called

facets) between adjacent vertebrae. The spinous and transverse processes serve as lever arms for many muscles running over single or multiple spinal levels. Only limited movements are possible between adjacent vertebrae, but the sum of these movements amounts to considerable spinal mobility in all major planes. Differences in mobility between regions (cervical, thoracic, lumbar) are due to the splinting effect of the rib cage, differences in shape and size of the articular, and spinous processes.

At birth the spine is generally dorsal convex (kyphotic), but during the first year with the assumption of an upright posture (lifting head, sitting up) the cervical and lumbar regions develop a lordotic shape. The bipedal human erect posture necessitates a tilt of the sacrum between the pelvic bones, increased lumbosacral angulation, and adjustments in size of individual vertebrae and discs. The increasing size of the vertebral bodies from cranial to caudal corresponds to the increasing weights and stresses imposed by successive segments. The sacral (and coccygeal) vertebrae are fused, forming a solid, wedge shaped base, transmitting the axial load of the spinal column over the paired pelvic bones and hip joints into the lower extremities.

The erect posture greatly increases the load carried by the lower spinal joints, and despite millions of years of evolutionary adaptations imperfections seemingly continue to exist, predisposing this region to strains and lower back pain. About three-quarters of axial spinal load is carried by the anterior column. Vertebral bodies, endplates, and intervertebral discs are the principal structures of the anterior column. We describe its elements more in detail below.

The vertebral body

The architecture of a vertebral body is comprised of highly porous trabecular bone, but also of a fairly dense and solid shell. The shell is very thin throughout, on average only 0.4 mm [34]. It is virtually indistinguishable from the trabecular core but rather is a denser arrangement of trabecular elements forming solid and compact bone (histologically different from cortical bone). Finite element analysis estimates the contribution of the shell to the overall load carrying capacity to be less than 15% [23, 35].

Regional variation in bone architecture also exist within any given vertebral body. The regions adjacent to the endplate feature more dense, rodlike trabecular structures. The regions far from the endplate, on the other hand, are less dense, with platelike shaped trabeculae. Mechanical properties tested in normal vertebrae for distinct regions of trabecular bone samples attribute higher strength, stiffness and bone mineral density (BMD) to central trabecular regions [18, 19, 20]. Variability in mechanical properties can be interpreted as adaptive to the environment, in this case to higher vertical stresses transmitted by the central region adjacent to the nucleus pulposus, as opposed to the peripheral region adjacent to the annulus fibrosus. Keller et al. [20] have demonstrated for degenerated intervertebral discs a change in adjacent trabecular mechanical properties, suggesting a more uniform load distribution across the endplate in degenerated spines.

The apparent bone density varies widely (0.05 g/cm^3 to 0.30 g/cm^3) between individuals, between levels, but also as a function of age. Starting in the fourth decade of life, elderly men can easily lose up to 30% and elderly women up to 50% of bone density [22]. Routine estimates of the apparent bone density are obtained using dual energy X-ray absorptiometry (DEXA). Although BMD or bone mineral content (BMC) are not volumetric parameters for bone, they still have proven to be useful predictors for ultimate vertebral strength, since the ultimate vertebral strength is dependent on both the vertebral geometry and the trabecular failure strength. To compare failure strength for vertebral samples from different spinal regions or from different individuals it is best to express the failure strength as a material property, normalized for the endplate's cross-sectional area [4], or expressed as compressive failure stress. The stress at failure for a lumbar vertrebral body is found to range from 1.0 to 5.0 MPa. This measure, however, does not differentiate between trabecular and compact elements of the vertebral body.

Keller [17] established from in vitro testing of isolated trabecular bone samples a relationship between apparent bone density and compressive failure strength. The exponential function [compressive strength=(97.8×apparent bone density)$^{2.30}$] identifies trabecular bone with low apparent density (<0.10 g/cm^3) to feature an ultimate compressive strength of less than 0.2 MPa, which puts this bone at risk to fracture already at axial loads seen in routine and low level daily activity. Resch et al. [30] have shown using quantitative computer tomography that men with 0.11 g/cm^3 apparent bone density have a 25% vertebral fracture risk, whereas individuals with 0.05 g/cm^3 bone density have a 99% vertebral fracture risk.

Osteoporosis is a disease that weakens the structural strength of bone to an extent that normal daily activity can exceed the vertebra's ability for carrying this load, resulting in vertebral fractures. The incidence of fragility fractures doubled within the last decade. It is predominant in women, with an osteoporotic fracture prevalence at age 50 years and above of over 40% ("Bone and joint decade," WHO 2003: http://www.boneandjointdecade.org/background/default.html). Clinically osteoporosis is characterized using DEXA measurements (BMD or BMC) of the lumbar spine that are 2.5 SD or more below the average value for a 30-year-old gender-matched individual [24]. In women the risk for vertebral fractures rises 2.2-fold for every 1 SD loss in BMD or BMC [5].

Decreased structural strength is not only the result of reduced apparent bone density, but also of profound changes in the architecture and the bone remodeling and/or repair rate, resulting in faster damage accumulation for continuous cyclic loading. The increase in bone fragility

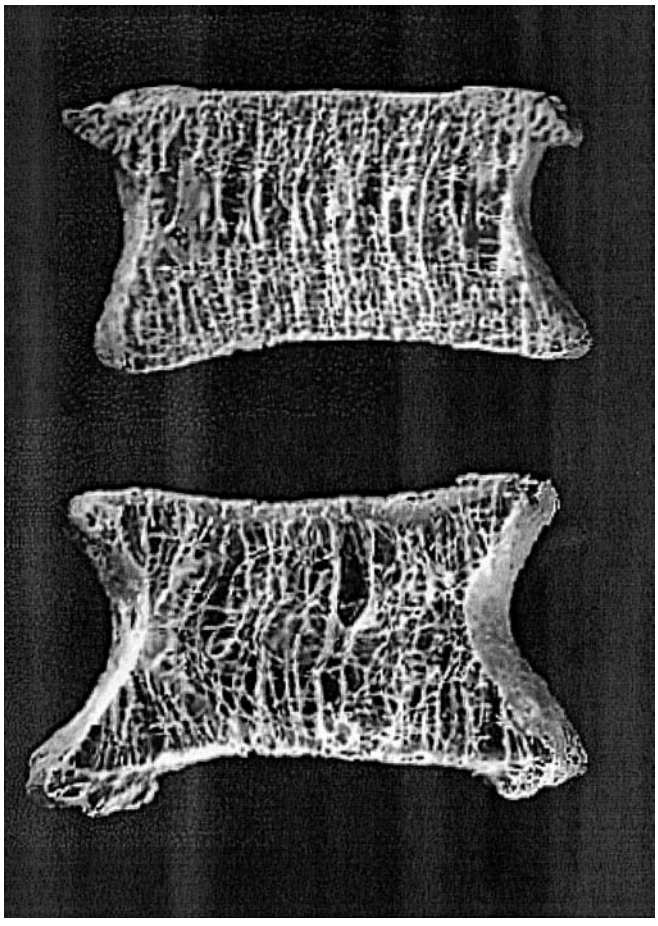

Fig. 1 Normal (*top*) and osteoporotic (*bottom*) vertebral bodies. Decreased structural strength is not only the result of reduced apparent bone density but also changes in the architecture of the trabecular bone. The increase in bone fragility is due to replacement of platelike close trabecular structures with more open, rodlike structures. The more porous cancellous bone appearance is the result of reduced horizontal cross-linking struts

is due to replacement of platelike close trabecular structures with more open, rodlike structures. The more porous cancellous bone appearance is the result of reduced horizontal cross-linking struts, further reducing the buckling strength of vertically oriented trabeculae (Fig. 1).

The typical osteoprotic vertebral fracture leads to a height reduction of the anterior vertebral body, often leaving the posterior vertebral wall intact. This wedge-shaped deformity usually leads to a local increase in kyphosis, and with multiple adjacent vertebral fractures to a progressive kyphotic deformity with postural disfigurement. Multiple vertebral fractures are very common, since the fracture risk of neighboring levels have shown to have a fivefold increased fracture risk compared to normal vertebrae [16].

The vertebral endplate

The vertebral endplate forms a structural boundary between the intervertebral disc and the cancellous core of the vertebral body. Comprised of a thin layer of semiporous subchondral bone, approximately 0.5 mm thick, with an overlying cartilage layer of similar thickness, the principal functions of the endplate are to prevent extrusion of the disc into the porous vertebral body, and to evenly distribute load to the vertebral body. With its dense cartilage layer, the endplate also serves as a semipermeable interface, which allows the transfer of water and solutes but prevents the loss of large proteoglycan molecules from the disc. Finally, the dense subchondral bone of the endplate provides secure anchorage for the collagen network of the intervertebral disc.

The thickness of the endplate varies, with thicker bone found under the annulus than adjacent to the nucleus. The superior endplate is generally thinner than the inferior endplate. A positive correlation between the thickness of the endplate and the proteoglycan content of the disc has been shown, especially for the central endplate under the nucleus. This may be the result of a remodeling process whereby the endplate responds to a greater hydrostatic pressure in discs with higher proteoglycan content [32]. Therefore it is possible that the changes associated with aging and disc degeneration could result in a weakening of the adjacent endplate.

The local material properties of the endplate demonstrate a significant spatial dependence. Grant et al. [9] have shown that the strength and stiffness of the endplate are highest posterolaterally and lowest in the center of the endplate (Fig. 2). Sacral and inferior lumbar endplates are

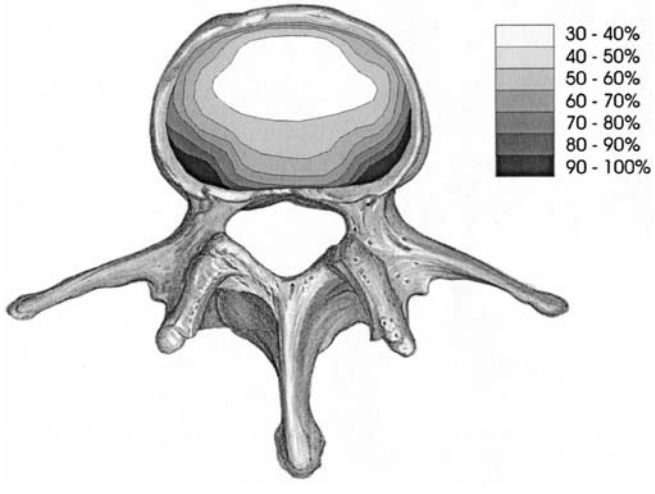

Fig. 2 Spatial distribution of endplate material properties, normalized to maximum values measured. Endplate strength is greatest towards the posterolateral and lowest at the center of the endplate. Regional variation in endplate properties is more pronounced with decreasing bone mineral density. (Adapted from [9])

stronger than superior lumbar endplates, which may indicate an increased fracture risk in the aging spine for the superior endplate. The importance of the endplate for load transfer and the overall structural integrity of the vertebra has been highlighted in laboratory experiments which have shown a significant reduction in the local structural properties of the vertebral body following partial endplate removal [27]. Similar experiments have provided support for the hypothesis that the strength of the central endplate region decreases with increasing disc degeneration due to remodeling, and that logically the overall strength of the endplate decreases with decreasing BMD [10]. With decreasing BMD, the regional variation in material properties becomes more pronounced, which likely plays a significant role in the initiation of the endplate fractures which are a characteristic of the aging spine.

Of particular relevance for the aging spine, the morphology of fatigue fractures of lumbar motion segments has been investigated in laboratory experiments [11, 12]. Under repetitive cyclic loading designed to simulate vigorous physical activity, the weakest part of the vertebral body was shown to be the endplate; failure often occurred after only several hundred cycles. Two main types of fatigue failure occurred, both involving the endplate and the adjacent subchondral cancellous bone of the vertebral body. Fracture morphology was weakly correlated with disc degeneration grade. The development of Schmorl's nodes – the local extrusion of disc material through the endplate – was most often seen with normal intervertebral discs. Central endplate fractures were associated with moderately degenerated discs. In some cases crush or burst fractures were observed. These occurred always on the first loading cycle and were seen in specimens with low BMD.

Deformity of the vertebral body with aging is closely related to BMD loss, i.e., osteoporosis. With aging, increased concavity of the vertebral endplate is seen together with a loss of BMD [36]. The typical loss of stature, often attributed to disc thinning, is more likely a consequence of a fairly normal disc migrating into this concavity. Endplate fracture is significant in the initiation of vertebral body collapse, but is difficult to diagnose from conventional morphometric assessment of spinal osteoporosis; up to 80% of all endplate fractures are missed by conventional diagnostic radiography [21]. However, Schmorl's nodes, which generally evolve from significant traumatic events, are easily recognized on magnetic resonance imaging (MRI) as either a characteristic extrusion of disc material, or as a localized edema in the vertebral body adjacent to the fracture [39].

In contrast to the thinning of the endplate and increased fracture risk often observed with aging, endplate sclerosis with aging has also been reported and can be so substantial as to bias normal measurements of vertebral body bone density [29]. Ossification of the overlying cartilaginous layer has been observed with aging [31]. Localized calcification directly influences the permeability of the endplate, and it has been shown that this may lead to a potential reduction in the volume of fluid exchanged to the disc during daily activity, resulting in a disruption of the nutritional supply to the disc and possible dehydration of the disc [3, 8]. Degenerative changes to the disc are extremely important factors determining the function of the elderly spine, as is outlined below.

The intervertebral disc

The intervertebral disc provides mobility to the spine. Positioned between the bony vertebrae, the disc allows complex motion without the mechanical disadvantages of the opposing articular surfaces of a diarthrodial joint. The disc derives its function from its unique structure, whereby the amorphous, gel-like nucleus pulposus is surrounded by the highly oriented annulus fibrosus. In the healthy disc, a hydrostatic pressure is developed within the nucleus, which is contained by the strong lamellae of the annulus, and loads are thereby evenly distributed across the underlying vertebrae.

The degenerative changes to the vertebral disc which are often observed with aging have been well described in the review by Vernon-Roberts [38]. Macroscopic changes to the disc include the appearance of horizontal splits and clefts midway between the center of the disc and the cartilage endplates, which extend posteriorly and posterolaterally and can eventually lead to fissures through the annulus. Microscopic fragmentation of annulus fibers has been observed, leading to a degeneration of individual fibers. Vertebral rim lesions, annular tears at the corners of the vertebral body separating the annulus from the bony attachment, are commonly present after the age of 50 years. Concentric cracks and cavities and radiating ruptures of the annulus are often present. At the disc boundaries cartilage endplate fissure formation, horizontal cleft formation, death of chondrocytes, vascular penetration, and Schmorl's nodes are observed. Disc thinning occurs due to loss of water content, conversion of the nucleus tissue to a highly organized collagenous tissue, gradual ossification of the endplate and protrusion of disc tissue. While the cartilage endplate and annulus are normally sufficiently strong to contain the nucleus, even under great stress, degeneration of the disc can lead to potential weak points in the subchondral bone and in the posterior and posterolateral segments of the annulus, which are thinner and less firmly attached to the vertebra.

Age- and degeneration-related changes to disc tissue material properties have been extensively evaluated. Based on measurements of the viscoelastic properties of the human nucleus pulposus, Iatridis et al. [14] concluded that changes to the mechanical properties suggest a shift from a "fluid like" behavior to a more "solid like" behavior with degeneration. Due to its crucial role in the containment of the nucleus, changes to the properties of the an-

nulus fibrosus have also been the subject of several studies. An increase in the elastic modulus with progressive degeneration has been shown, likely the results of an increase in tissue density due to water loss. This suggests a shift in the load carriage mechanism of the disc with increasing degeneration from fluid pressurization to elastic deformation of the annulus fibrosus [15]. Although dramatic changes in annulus fibrosus morphology and composition have been documented with aging and degeneration, the tensile mechanical properties of the annulus are not substantially affected by degeneration. A far more important factor for the tensile properties, especially in the radial direction, is the position within the annulus, and this relationship does not change substantially with age or degeneration [1, 7]. Significant changes to the ligamentous structures of the spine with aging have been reported. For example, the elastic modulus of the main substance of the anterior longitudinal ligament increases twofold, while the modulus of the ligament insertion decreases threefold, between 20 and 80 years of age, and the strength of the bone ligament junction decreases twofold with aging [25].

The fluid content of the disc is important for determining its mechanical response. Hydration depends on the proteoglycan content of the disc and also on the balance between external load and the internal swelling pressure of the disc. The influence of age, spinal level, composition and degeneration on disc swelling pressure has been measured for human discs [37]. The natural swelling pressure for human discs was found to be approximately 0.1–0.2 MPa. Proteoglycan content decreased with age, and was lowest at L5–S1, but no substantial change in collagen content was found. Therefore the relationship between equilibrium hydration and swelling pressure could be predicted based on proteoglycan and collagen content, while age and degree of degeneration were not significant factors.

Aging and disc degeneration have a profound effect on the mechanism of load transfer through the disc. Using the technique of "stress-profilometry," it has been shown that age-related changes to the disc composition result in a shift of load from the nucleus to the annulus [2]. A reduction by approximately 50% of the central hydrostatic region of the disc was observed, and a corresponding 30% reduction in pressure for degenerate discs (Fig. 3). The width of the functional annulus increased by 80% and the height of the compressive stress peak in the annulus by 160% with degeneration. While age and degeneration were closely related, the state of degeneration had the most profound influence on the measured stress distributions. Therefore structural changes in the annulus and endplate with aging may lead to a transfer of load from the nucleus to the posterior annulus, which may cause pain and also lead to annular rupture.

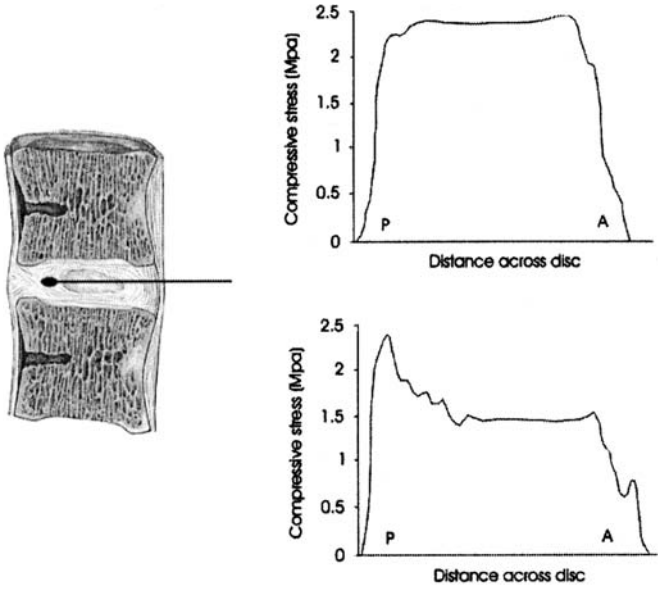

Fig. 3 Typical stress profiles for grade-1 disc (*top*) and for a grade 4 disc (*bottom*). In the healthy disc, a hydrostatic pressure is developed in the nucleus, as indicated by the plateau in the stress plot. For the degenerate disc, nuclear pressure is lower, and stress peaks in the annulus fibrosus are observed. *A* Anterior; *P* posterior. (Adapted from [2])

Combined effects of disc degeneration and osteoporosis

The correlation between degenerative changes to the vertebra and the disc remains an open question. Endplate fracture or vertebral body deformity is not necessarily associated with disc degeneration. While disc thinning may be implied from observed stature changes, disc morphometry is altered to accommodate changes to the vertebral body shape by extrusion into the concave endplate, but indicators of degeneration (i.e., MRI signal intensity) are not altered subsequent to throacolumbar spine fractures [26]. Based on MRI imaging and DEXA measurements, a negative correlation between vertebral BMD and intervertebral disc degeneration has been shown [13]. Dai [6] has suggested that, for patients with severe osteoporosis, vertebral bodies adjacent to discs with decreased height or signs of degeneration are less likely to be deformed. In an in vitro study of the influence of disc degeneration on the mechanism of vertebral burst fractures, Shirado et al. [33] demonstrated that disruption of the middle end plate was found only in specimens with normal disc quality. In specimens with severe disc degeneration and osteoporosis, no burst fractures were observed. Further analysis of their test results led to the conclusion that stresses were concentrated towards the center of the vertebral endplate due to hydrostatic pressurization of a normal nucleus pulposus.

The possible mechanical interactions due to disc degeneration and concurrent osteoporotic changes to the vertebrae have been extensively studied using detailed computer

simulations of whole spine segments [28]. These analyses have predicted that osteoporosis alone has a substantial influence on the overall stiffness of a spine segment, resulting in a 35–40% reduction in stiffness. Correspondingly, the magnitude of internal vertebral strains for a nominal load level were predicted to increase with the progression of osteoporosis. However, the spatial patterns of strain distribution within the vertebral bodies were similar for the normal and osteoporotic vertebra. Conversely, the simulation of disc degeneration has predicted a substantial load shift from the nucleus towards the annulus, as previously demonstrated in stress-profilometry measurements [2]. While vertebral strain magnitudes for the degenerate disc were similar, there was a marked change in strain distribution, which was an opposite effect to that observed for osteoporosis. Therefore a degenerate disc may moderate the detrimental effects of extreme osteoporosis and it could be hypothesized that the increased fracture risk of an osteoporotic spine segment may be slightly counterbalanced by the material consequences of disc degeneration. This is in agreement with the findings by Shirado et al. [33] and Dai et al. [6], reporting that vertebral bodies next to degenerated discs were less likely to be deformed or fractured for patients with spinal osteoporosis.

Conclusion

The human spine is a highly evolved structure capable of an extensive range of motion and with considerable load carrying capacity. Age-related changes to the form and composition of the individual structures of the spine may increase the risk of injury and limit quality of life for elderly patients. Cancellous bone forms the structural framework of the vertebral body. With aging a loss of BMD, as well as morphological changes including trabecular thinning, increased intratrabecular spacing, and loss of connectivity between trabeculae, may lead to an increased risk of vertebral fracture. The vertebral endplate serves the dual role of containing the adjacent disc and evenly distributing applied loads to the vertebra. Thinning of the endplate and loss of bone density increases the risk of endplate fracture. The intervertebral disc provides mobility to the spine, and transfers load via hydrostatic pressurization of the hydrated nucleus pulposus. Changes to the tissue properties of the disc, including dehydration and reorganization of the nucleus and stiffening of the annulus fibrosus, markedly alter the mechanics of load transfer in the spine. However, advancing age is not the sole factor in the degeneration of the spine and further study is required to understand the mechanisms of degeneration and the unique biomechanical function of the aging spine.

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