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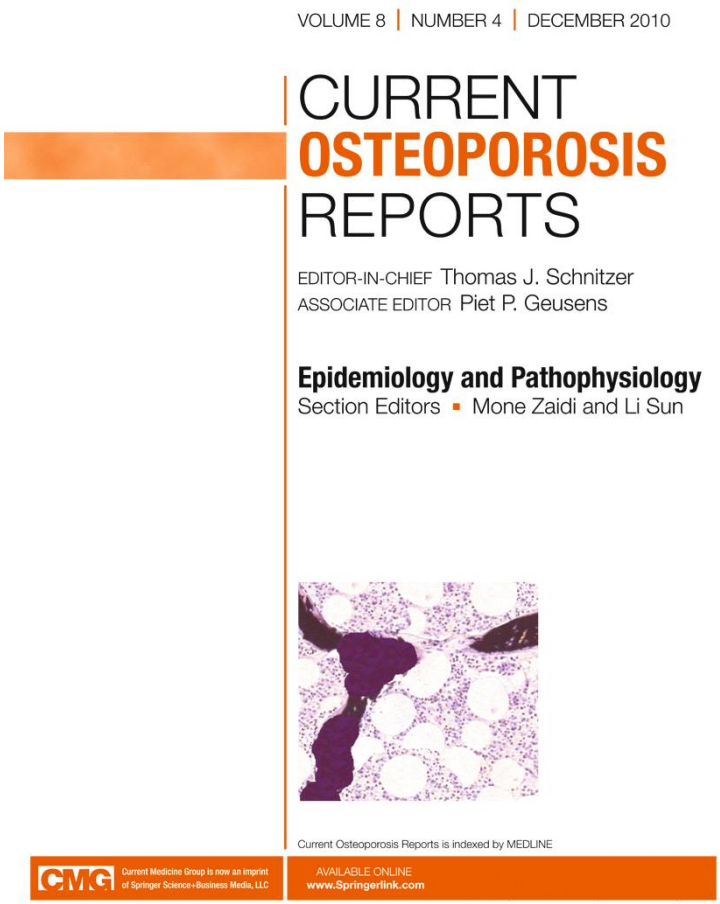

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# Biomechanics of Vertebral Fractures and the Vertebral Fracture Cascade

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**Abstract** Vertebral fractures (VFxs) are the most common osteoporotic fracture, and are a strong risk factor for future fracture. The presence of a VFx greatly increases the risk of sustaining subsequent VFxs—a phenomenon often referred to as the "vertebral fracture cascade." VFxs do not occur uniformly along the spine, but occur more often at the mid-thoracic and thoracolumbar regions than elsewhere. It is likely that both the vertebral fracture cascade and the bimodal distribution of VFx along the spine are attributable to biomechanical factors. VFxs occur when the forces applied to the vertebral body exceed its strength. Loading on the spine is primarily determined by a person's height, weight, muscle forces, and the task or movement performed, but can also be affected by other factors, such as spinal curvature and invertebral disk deterioration. Vertebral strength is determined mainly by bone size, shape, and bone mineral density, and secondarily by bone microarchitecture, collagen characteristics, and microdamage. Better understanding of VFx etiology is hampered by the fact that most VFxs do not come to clinical attention; therefore, the factors and activities that cause VFxs remain ill defined, including possible differences in the etiology of acute fractures versus those of slow onset. Additional research is needed to elucidate the precise mechanical, morphologic, and biological mechanisms that underlie VFx to improve strategies for assessing VFx risk and preventing the vertebral fracture cascade.

**Keywords** Vertebral fracture · Vertebral strength · Spinal loading · Biomechanics · Intervertebral disc degeneration · Spinal curvature · Kyphosis

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## Introduction

Vertebral fractures (VFxs) are the hallmark of osteoporosis, and are associated with height loss, spinal deformity, chronic pain, and reduced quality of life. They are the most common osteoporotic fracture, occurring in 30% to 50% of people over the age of 50 years [1]. VFxs are also critically important because they are a strong predictor of future fracture risk at any site, independent of bone mineral density (BMD) [2, 3]. The risk of sustaining a new VFx is severalfold higher in those who already have a VFx compared to those with no VFx, and increases exponentially with the number and severity of prevalent fractures. Of great concern is the high rate of subsequent VFx following an initial fracture—often referred to as the "vertebral fracture cascade" [4]. As many as 20% of women with a prevalent VFx will suffer a new fracture within 1 year [5]. A fracture occurs when the forces applied to the vertebrae exceed its strength, and therefore factors related to both—skeletal fragility and spinal loading—may play important roles (Fig. 1). Yet, studies of the etiology of VFxs are hindered by the fact that only 25% to 30% of VFxs come to clinical attention, and therefore the conditions, activities, and events that lead to a VFx remain ill defined. Thus, despite the phenomenal personal and public

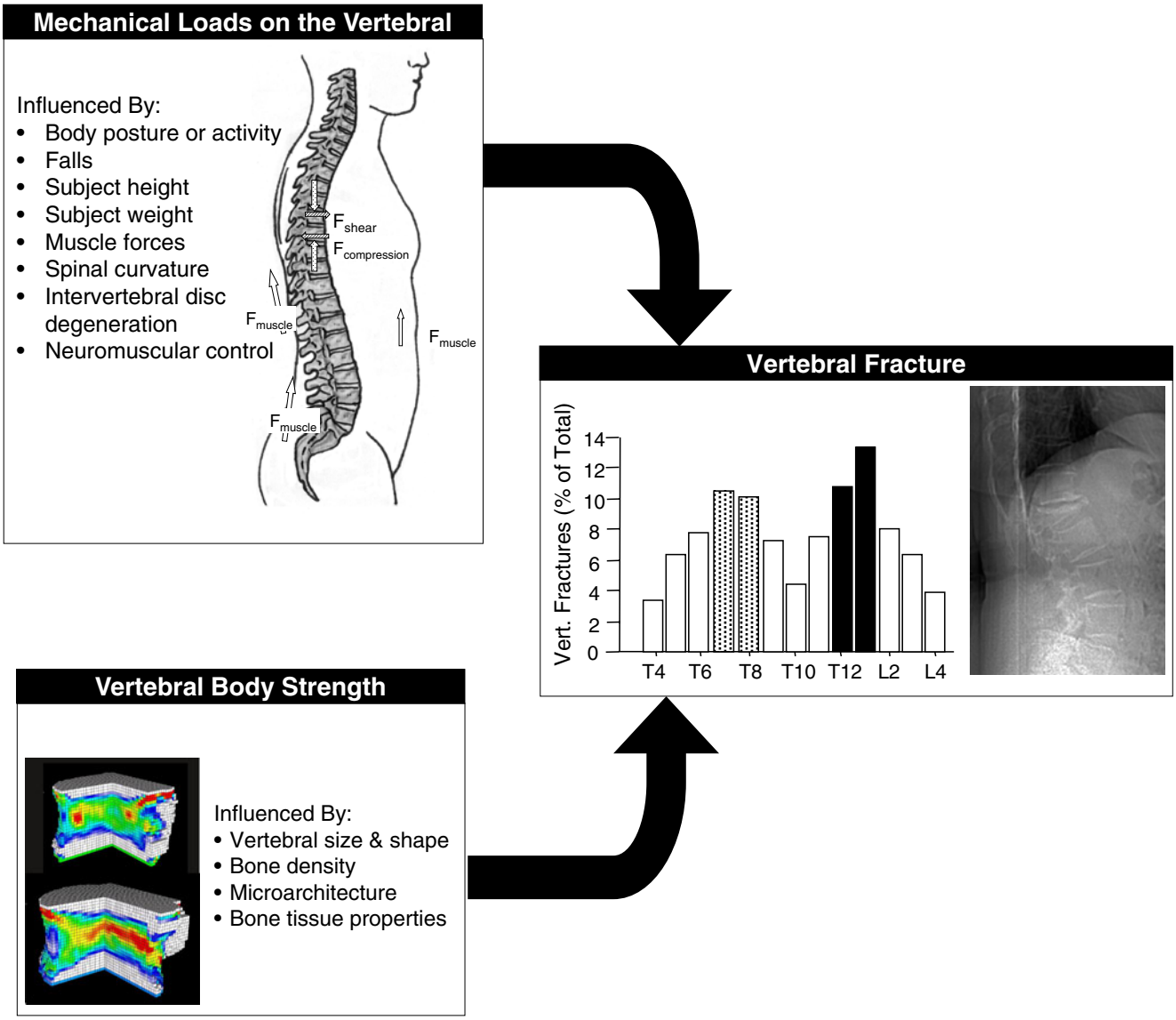


**Fig. 1** This figure highlights the bimodal distribution of vertebral fractures, and the factors that contribute to vertebral fracture etiology. Fractures occur when the loads applied to a vertebral body exceed its strength. The loads applied to the vertebral body are determined by a number of factors, including the specific activity (including falls) and associated body posture; subject height and weight; spinal curvature; neuromuscular function; and intervertebral disc degeneration. Vertebral body strength is determined by vertebral body size, shape, bone density, microarchitecture, and bone tissue properties. F—force

health impact of VFx, the underlying biomechanical mechanisms that result in VFx remain largely unknown. It is possible that a more thorough knowledge of the biomechanics of the spine could lead to a better identification of people who are at risk for VFxs.

## Location of Vertebral Fractures Along the Spine

An important observation is that despite low bone mass being generally thought of as a systemic disorder, VFxs do not occur uniformly along the vertebral column. Rather, they occur more often at the mid-thoracic (T7-T8) and thoracolumbar (T11-L1) regions than elsewhere in the spine [6, 7]. The reasons underlying this bimodal distribution of VFxs are not completely understood, although it has been hypothesized that biomechanical factors due to the variations in the curvature of the spine contribute to the increased VFx incidence in these regions. For example, the maximum thoracic kyphosis occurs around T7-T8; this may result in greater anterior bending moments and increased risk of anterior wedge fractures in this region. In support of this theory, Briggs et al. [8] used a biomechanical model of the spine to demonstrate that

elderly subjects with higher thoracic kyphosis have greater anterior bending moments, as well as increased compressive and shear forces on their vertebral bodies.

At the thoracolumbar junction, the spinal curvature transitions from kyphotic to lordotic, and the rigid thoracic cage gives way to a more mobile lumbar spine. It is possible that the higher incidence of VFxs at T12-L1 is due to increased load-bearing by the vertebral bodies, as the rib cage no longer helps support superincumbent loads at these spinal levels. Moreover, the transition from a relatively rigid thoracic spine/rib cage construct to a more mobile lumbar region may contribute to increased fracture risk, although the mechanism underlying this is not completely understood.

Another factor that may contribute to the uneven distribution of VFxs includes variations in BMD and bone strength along the spine. Vertebral compressive failure loads generally increase from the thoracic to lumbar spine [9]. However, Burklein et al. [10] compared the compressive strength of T6, T10, and L3 vertebrae in 119 cadavers, and reported only modest correlations between the different levels (eg, $r^2$=0.37 for T6 vs L3), suggesting a heterogeneity in bone strength along the spine that may contribute to variations in fracture incidence at different regions of the spine.

## Factor of Risk for Fracture

When engineers design a structure, they consider the forces that the structure is expected to withstand, and compute a "safety factor," which is the ratio of the structure's strength to the loads it must withstand. When this ratio approaches 1.0, the structure is at risk to fail. A similar concept can be used to explore fragility fracture of bones, defining the ratio of loads applied to the bone to its strength as the "factor of risk":

$$\Phi = \frac{Load}{Strength}$$

When the factor of risk (Φ) exceeds 1.0 (ie, the loads applied to a bone exceed its strength), a fracture is predicted to occur. Using this approach, Myers and Wilson [11] showed that at very low BMD values, many routine activities of daily living could cause VFxs. Furthermore, at the lumbar spine, the factor of risk for VFxs during forward bending and lifting increases with age in both sexes, more so in women than men, and mimics the observed incidence of VFx [12]. Melton et al. [13] recently reported that a 1-SD increase in the factor of risk, computed for forward bending and lifting, was associated with a 1.8-fold increased risk of any prevalent vertebral deformity and a 2.9-fold increased risk of a moderate or severe vertebral deformity. In comparison, for a 1-SD decrease in spine areal bone mineral density (aBMD), the risk of any vertebral deformity and moderate or severe deformity was increased 1.3-fold and 1.8-fold, respectively. These findings demonstrate the potential utility of the factor-of-risk approach in assessing VFx risk, and the relative ineffectiveness of using aBMD alone to predict VFx risk.

To further improve this biomechanical approach for predicting VFx risk, it is necessary to identify the activities that cause VFxs, and then to accurately predict both the loads on the spine and the strength of the vertebral body for those loading conditions.

## Activities Associated with Vertebral Fractures

Unlike the strong association between falls and hip fracture, relatively little is known about events associated with VFxs, in part because only one third of VFxs come to medical attention. A retrospective chart review of activities associated with painful VFxs reported that fractures were often associated with moderate trauma such as a car accident, lifting, and falling, but that fully 50% were reported as occurring "spontaneously" [14]. Patel et al. [15] reported that nearly half of acute VFxs were spontaneous (with 64% of these noticed when the patient was getting out of bed), 37% occurred during trivial housework, and 17% were due to moderate or serve injury. In a recent study in men, 73.8% of clinical VFxs were precipitated by no known trauma or by low-energy trauma, including falls in 57.3% [16••]. These latter findings are provocative, and they suggest that in addition to their prominent role in the etiology of non-VFxs, falls may also play a key role in VFxs as well. Yet, there is little known about the association between falls and VFxs (ie, What type of fall is most risky for VFxs? How are forces transmitted through the spine during different types of falls?). A more thorough investigation of the activities associated with VFxs would provide information about the mechanical loads on the spine that lead to VFxs, and would allow for more accurate biomechanical assessment of VFx risk.

## Estimating Loads on the Spine

To compute the factor of risk for VFxs, it is necessary to accurately estimate loads on the spine for various activities. In general, it is difficult to quantify the forces acting on the spine at any given time, since these forces cannot be directly measured. Instead, these forces are typically measured indirectly by quantifying intradiscal pressure [17], or estimated using biomechanical models [18]. Most

biomechanical models of the spine have been developed with the primary goal of exploring the causes of low back pain. These models provide estimates of the loads applied to the lumbar spine during occupational tasks, such as bending and lifting. The simplest models consider only the erector spinae and rectus abdominis muscles in sagittally symmetric flexion and extension tasks. More sophisticated models incorporate multiple muscles, relying on optimization schemes to partition the forces among the various muscle groups. Although all of these models make several simplifying assumptions, predictions of spine forces using these models correlate strongly with intradiscal pressure measurements and electromyogram recordings of muscle activity [19, 20]. Moreover, predictions of spinal compression forces obtained using a variety of techniques show reasonable agreement, lending validity to the overall approach [18].

For assessment of VFx etiology, there are two major limitations in current approaches for prediction of spine loads. First, whereas there are numerous models for the *lumbar* spine, few biomechanical models have been developed to estimate vertebral loading in the *thoracic* spine. Those that are available were designed to study respiratory mechanics, scoliosis, and rib cage deformities, but not loading of the thoracic spine during activities of daily living. Using experimental data on the stiffness of the thoracic spine, rib cage, and sternum, we have recently developed a new quasistatic stiffness-based biomechanical model to calculate loads on the thoracic and lumbar spine during bending or lifting tasks that may prove useful for identifying activities that generate high forces on vertebrae in the thoracic spine [21].

A second limitation in current spine models is that because the majority of biomechanical models are intended to study occupational low back pain, they generally use anthropometric variables relevant to young- to middle-aged men. Estimation of spinal loads is sensitive to subject height, weight, and trunk muscle geometry; therefore, individual differences in these values should be addressed to get accurate predictions of spinal loads. In particular, it has been argued that sex-related differences in trunk muscle geometry should be considered in developing biomechanical models of the torso [22].

## Vertebral Body Strength

Variation in vertebral compressive strength is determined mostly by vertebral size (and therefore bone mass) and bone density, with laboratory studies reporting that aBMD explains 50% to 70% of the variability in vertebral compressive strength [23]. Other features such as microarchitecture, collagen characteristics, microdamage accumulation, mineralization, osteocyte number, and viability may also play a role, although their relative contribution to whole vertebral strength remains ill defined.

Whereas several studies have demonstrated an important role of microarchitecture in determining the mechanical behavior of isolated trabecular bone specimens, the contribution of trabecular and cortical bone microarchitecture to whole vertebral strength is less well understood, and difficult to isolate due to dominant influence of vertebral size, shape, and bone mass. For example, Fields et al. [24•] recently reported that the addition of trabecular microarchitecture improved the correlation with vertebral strength from $r^2$=0.57 for bone mass alone to $r^2$ = 0.85 for bone mass plus microarchitecture. Properties of the cortical shell have also been shown to contribute prominently to whole vertebral biomechanical behavior [25, 26]. Of great interest are recent studies showing that microstructural heterogeneity may contribute to vertebral fragility independently of bone mass [27, 28•]. Many clinical studies also provide supporting evidence for the important role of trabecular microarchitecture in vertebral fragility, showing deteriorated trabecular and cortical microarchitecture (assessed by morphometric analysis of iliac crest biopsies) in patients with VFxs. More recently, noninvasive assessment of trabecular and cortical bone microarchitecture by high-resolution peripheral computed tomography (hr-pQCT) has shown microstructural deficits, particularly in cortical bone, to be associated with increased severity of VFxs in postmenopausal women [29••].

### Noninvasive Measurements of Vertebral Strength

Several methods are available for noninvasively estimating the mechanical strength of a vertebral body, an estimate of its ability to withstand mechanical loading. The general approach used to validate these noninvasive methods is to use human cadaveric spines to determine which noninvasive assessments of vertebral bone density and/or geometry are most strongly correlated with vertebral strength. Generally, these studies show modest to strong correlations ($r^2$~0.30–0.80) between spine aBMD assessed by dual-energy x-ray absorptiometry (DXA) or between QCT-based measures of bone density combined with cross-sectional area, and vertebral compressive strength [23, 30]. However, alternate approaches using QCT-based finite element analysis (FEA) to predict bone strength appear promising. In this technique, a three-dimensional QCT scan is converted into a finite element model on a voxel-by-voxel basis, with assignment of material properties based on the volumetric bone mineral density (vBMD) of each voxel [31]. Laboratory studies suggest that QCT-based FEA is more strongly associated with vertebral compressive strength than QCT measures of bone density and cross-

sectional area [30]. In a seminal study over a decade ago, Faulkner et al. [32] developed patient-specific finite element models from spine QCT scans, and showed that these models more successfully discriminated fracture patients from controls than BMD measurements alone. More recently, Melton et al. [13] reported that decreased vertebral compressive strength, assessed by QCT-FEA, was associated with prevalent VFxs, with odd ratios of 1.4 (95% CI, 1.1–1.8) and 2.9 (95% CI, 1.8–4.8) for mild and severe vertebral deformities, respectively. In comparison, odds ratios for lumbar spine aBMD measurements were 1.2 (95% CI, 0.9–1.6) and 1.8 (95% CI, 1.2–2.6), respectively, for mild and severe vertebral deformities. Moreover, QCT-based FEA has been used in several studies to assess the effect of osteoporosis therapies on vertebral strength, and have demonstrated a greater sensitivity to detecting gains in strength than aBMD measurements [33, 34, 35•, 36]. Most recently, investigators are applying multiscale and nonlinear FEAs to study vertebral failure processes in greater detail [37–40].

## Vertebral Fracture Cascade

As previously stated, prevalent VFx are possibly the strongest predictor of future fracture risk, particularly VFxs, independent of aBMD measurements [2, 3, 41, 42]. However, it is unclear why one VFx predisposes a person to more fractures, although there are a number of factors that may contribute to this phenomenon. It is possible that VFxs are an indicator of overall poor bone strength along with deteriorated trabecular and cortical microstructure that would predispose one to multiple fractures. It is also likely that the presence of a VFx fundamentally alters the mechanical loads experienced by the adjacent vertebral bodies. In fact, estimation of compressive and shear spinal loads using a biomechanical model predicted higher loads in subjects with a single VFx than those with no fracture [43]. Moreover, increased thoracic kyphosis that may result from anterior wedge fractures increases vertebral loads [8]. Altogether, this altered mechanical loading environment may have an important role in contributing to the cascade of VFxs.

## Other Age-Related Changes that May Contribute to Vertebral Fractures

Several morphologic and functional changes to the musculoskeletal system occur with advanced age that may also contribute to changes in the mechanical loading of vertebral bodies, making them more likely to sustain a fracture. These changes include degeneration of intervertebral discs, loss of balance and coordination leading to falls, and decreased muscle quality and strength.

### Intervertebral Disc Degeneration

The intervertebral discs of the spine have an important role in determining the magnitude and distribution of forces that are transmitted to the vertebral bodies. With increased age, intervertebral discs progressively deteriorate, causing them to become more fibrous and less able to distribute compressive stress evenly, and as a consequence some parts of the vertebral body are subjected to high stress concentrations. In particular, severe disc degeneration causes increased load-bearing by the neural arch and posterior elements during upright stance, and decreased loading on the vertebral body, which may lead to progressive loss of bone mass in the anterior vertebral body [44]. As a consequence of this altered load distribution, the anterior vertebral body is stress-shielded during normal erect posture, but severely overloaded when the spine is flexed [45]. This sequence of changes to the spine may be a mechanism by which the anterior region of the vertebral body becomes vulnerable to osteoporotic fracture, why forward bending movements may precipitate VFxs, and why disc degeneration has been reported to be associated with VFxs [46].

### Changes in Neuromuscular Function

Lean muscle mass typically peaks in the mid-20 s, then progressively diminishes throughout life [47]. Muscular strength, conversely, is largely maintained at peak levels until the fifth or sixth decade, after which accelerated losses occur, with strength decreasing 24% to 36% by the age of 70 years [48]. Not only does peak muscle force production diminish with advancing age, but rate of muscle force development and power also decline [49]. These changes in force production by muscles could detrimentally change the loading of the spine, as coordinated antagonistic muscle contraction is key for maintaining the stability of the spine during flexion and extension tasks [50]. A reduction in the intrinsic spine stability may contribute to the poorer balance and postural stability seen with increasing age, and in subjects with osteoporosis. Accordingly, this decreased muscle function may further contribute to falls that lead to fractures.

## Conclusions

VFxs are common and they lead to profound negative effects on quality of life and health status. The mechanisms underlying VFxs are multifactorial, and include factors

related to vertebral strength and to the forces applied to the vertebral column. Better understanding of VFx etiology is hampered by the fact that a minority come to clinical attention, and therefore the factors and activities that cause VFxs remain ill defined, including possible differences in the etiology of acute fractures versus those of slow onset. Finally, many factors may contribute to the vertebral fracture cascade, and additional work to elucidate the precise mechanical, morphologic, and biological mechanisms that underlie this phenomenon is needed to improve strategies for preventing VFxs.

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- •• Of major importance


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