

## Review Article

# Lumbar Intervertebral Disc Injuries in Low Velocity Rear End Vehicular Collisions: The Current Evidence

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**Abstract**

Rear-end collisions are the most frequently occurring type of collision in the United States, accounting for approximately 29% of all collisions. Of these collisions, many are so-called "low velocity" or "minimal impact" type collisions, characterized by low impact speeds (less than 9-10 mph) and limited observable damage to the rear-ended vehicle. Lumbar intervertebral disc injuries are sometimes claimed by injured parties as a result of such collisions. This paper addresses lumbar biomechanics and relevant topics, such as degeneration and potential injury mechanisms, with a focus of how such knowledge relates to the occurrence of lumbar intervertebral disc injuries as a result of low velocity rear-end impacts. We conclude that the evidence for the occurrence of lumbar disc injuries in such collisions is not compelling, reflecting limited impact forces, limited lumbar range of motion, and the general lack of injury mechanisms being present. However, it is acknowledged there are significant data gaps. Important questions needing further inquiry include the experimental validation of the concept that acute disc injuries can occur at all under such conditions and the role of degenerative processes.

**ABBREVIATIONS**

**BMI:** Body Mass Index; **ADL:** Activities of Daily Living; **MIST:** Minimal Impact Soft Tissue; **FSU:** Functional Spinal Unit

**INTRODUCTION**

Lumbar disc pathologies such as herniation, protrusion and bulge are one of the most common reasons for low back pain; however, such conditions can often be asymptomatic [1-3]. Congenital, degenerative, infectious and traumatic events are some of the common causes of disc disease. Acute injury events, such as vehicular collisions, have also been referred to as a possible contributor; however, the speed threshold and the dynamics at which the collision can be harmful are controversial topics. Evidence suggests the claim of a disc injury may be litigation-driven (i.e., financially driven), where the symptoms seem to clear up rapidly after the legal process concludes [4]. The question if an acute event or natural degeneration causes lumbar disc injuries is still unknown. One possible exception to this debate is blunt trauma like in sports injuries or fall

events. In these events, there is a direct contact with an object/subject, where high impact forces are experienced, as opposed to the presence of a penetrating object. On the other hand, the causal relationship between a supposed acute injury and a pure acceleration-deceleration event (in the absence of any blunt trauma) is at the center of the controversy.

A common potential acute injury event suggested by some investigators is the possibility of a lumbar disc injury sustained in vehicular collisions. In high velocity collisions, specifically, lumbar injuries are commonly a combination of bone and soft-tissue injury [5] with lumbar disc injuries reported [6]. Seat-belt usage, body mass index (BMI) and age have shown to play an important role in the mechanism of these injuries [7]. However, the incidence of lumbar disc injury in the so-called low velocity rear-end collisions, also sometimes referred to as "MIST" (Minimal Impact Soft Tissue) events, is a controversial topic, in both the medical and litigation areas. Much of the claimed supporting evidence is either anecdotal or scattered in non-peer reviewed sources. In medical treatment, a causal relationship

between a lumbar disc injury diagnosis and a recent vehicular collision is often the result of “diagnosis by history” (i.e. the treatment provider is relying strictly on the history as reported by the injured party). Alternatively, the causal relationship results from a “diagnosis of exclusion” (i.e. there is no obvious injurious event to reference other than the recent collision). Secondary monetary gains often exaggerate or muddle the medical issue as patients, physicians and attorneys may financially benefit from insurance settlements. Also, physicians who provide treatment under a financial arrangement, such as a Letter of Protection (LOP), now could have a vested interest in the outcome of the case; hence they may not be unbiased in their testimony. The public widely assumes, based on the medical causation approach (diagnosis by history and exclusion), an underlying assumption that pain or abnormalities after an event must originate from the event and does not derive from a pre-existing condition. Two theories related to the “acute event causes injury” exist. The first postulates that an acute event such as a vehicular collision can establish lumbar disc injury in otherwise healthy subjects. The second hypothesizes that the subject already has some stage of disc pathology, but is otherwise asymptomatic, and the acute event is the final step in the transition from asymptomatic to symptomatic; many refer to this as the “egg shell theory”. Advocates to the second theory note that such condition is more likely in older subjects, i.e., and they expect that degenerative changes would be present. Regardless of the injury theory, many involved parties claim these injuries after low impact rear end collisions, but the feasibility of this event is ambiguous. This investigation reviews background information and scientific evidence available in the area of lumbar intervertebral disc injuries related to low velocity rear-end collisions. Literature related to the question as to whether an acute lumbar disc injury can occur at all is discussed, as well as the importance of exploring any data gap.

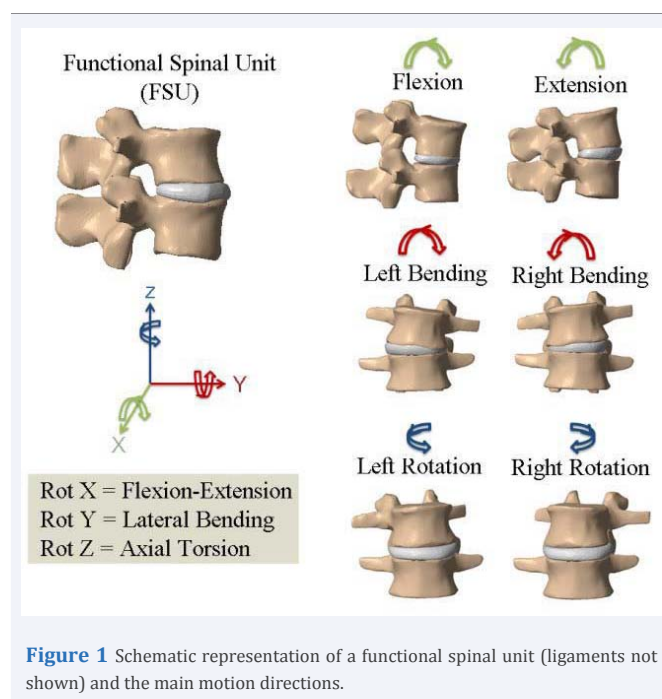
## BACKGROUND

### Lumbar basic anatomy and biomechanics

The vertebral column consists of the cervical, thoracic, lumbar, and sacral and coccyx regions of the spine. The male average length of the entire column is approximately 71 cm, of which the lumbar region is responsible for about 18 cm [8]. The lumbar spine receives special clinical attention since it is known to experience greater degenerative changes [9] and it is also known as the main source of back pain. One of the most important biomechanical characteristics of the lumbar spine is that the trunk exposes it to the greatest compressive load. In the upright postures, this compressive load could be approximately 400 N due to gravity alone; however, this force can increase exponentially with muscle interaction from bending and torsion, exceeding 4000 N [10]. Flexion-extension, lateral bending, axial rotation, as well as their

Combination, are the main motions experienced by the spine. It is a common practice to evaluate its biomechanical behavior through isolated cadaveric Functional Spinal Units (FSUs) exposed to various anatomical loads and/or range of motion in different directions. A FSU, which consists of two adjacent vertebrae, the intervertebral disc and ligaments, is considered the smallest spinal segment capable of demonstrating biomechanical

properties (Figure 1). Ranges of motion for the different FSUs of the lumbar spine have been reported in the literature (Table 1), showing that lumbar segments, specifically, exhibit greatest flexibility during flexion, followed by extension/lateral bending and axial rotation. Biological and mechanical changes occurring in the intervertebral disc, specifically, are believed to be one of the common causes of low back pain. The disc, which is constituted by the nucleus pulposus, annulus fibrosus and cartilaginous endplate (Figure 2), is mainly designed to absorb and distribute the loads experienced by the spine and its shape has been shown to play an important role in the disc biomechanics. Table 2 presents information on lumbar disc cross-sectional area and average disc thickness from the work of Lin and colleagues [14]. Lower lumbar discs are suggested to be weaker in torsion than upper levels with the same cross-sectional area since they are more oval in shape; however, lower levels generally have greater area, which tends to compensate the loss of strength in healthy spines [15]. There have been limited reports of lumbar intervertebral disc mechanical properties in the literature. Several investigations have determined maximum compression strength: 610-1002 lbs [16], 1,109 lbs [17], and 448-2,128 lbs [18]. Adams and Hutton [19] found that inelastic stretching occurred in lumbar discs



**Figure 1** Schematic representation of a functional spinal unit (ligaments not shown) and the main motion directions.

**Table 1:** Representative values for lumbar range of motion (in degrees) of functional units and during activities of daily living (ADLs).

Motion	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1	Total	ADLs*
Flexion	6 - 8	7 - 10	7 - 12	8 - 13	7 - 9	35 - 52	
Extension	4 - 5	3 - 5	1 - 6	2 - 7	5 - 6	15 - 29	9 (3-49)
Lateral Bending	3 - 6	3 - 6	5 - 6	4 - 5	1 - 2	16 - 25	6 (2-11)
Axial Rotation	1 - 4	1 - 3	1 - 3	1 - 3	1 - 3	5 - 16	5 (2-7)

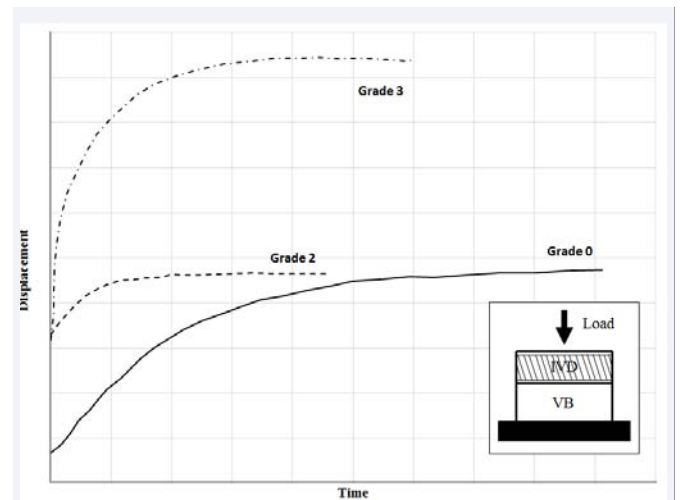
Data from Grieve [11] and Pearcy et al. [13]

\*Data from Bible et al. [12]. Values represent median (range)

past 9 degrees of rotation, with disc failure occurring between 10 and 26 degrees of rotation. In addition, Wilder et al. [20] demonstrated that long-term (1hr) combined loading (370N applied axially 4mm forward of the original balance point) and vibration (80N peak to peak at 5 Hz) can cause tracking tears of the annulus and bulging in the disc in isolated FSUs, suggesting possible relationship between prolonged sitting and disc herniation; this observation goes in line with the time-dependent behavior of the disc. Furthermore, the isentropic behavior of the intervertebral disc explains why a lumbar disc exhibits greater strain in lateral shear and axial compression than in anterior or posterior shear [21]. Being a viscoelastic material, intervertebral discs display creep and relaxation behaviors. An investigation by White and Panjabi [22] in the viscoelastic behavior of the spine demonstrated that a less viscous disc (zero degeneration) reaches its end deformation slowly while a more viscous disc reaches the end deformation faster (grade 2 and 3) (Figure 3). In addition, creep responses in compression are nonlinear, where stiffness and damping increase with larger forces [23].

### Aging and Degeneration

Aging is frequently mentioned as the main cause for degeneration; however, it is not the only one [15]; smoking, occupation and genetics have been also postulated as important risk factors leading to this condition [24,25]. On the other

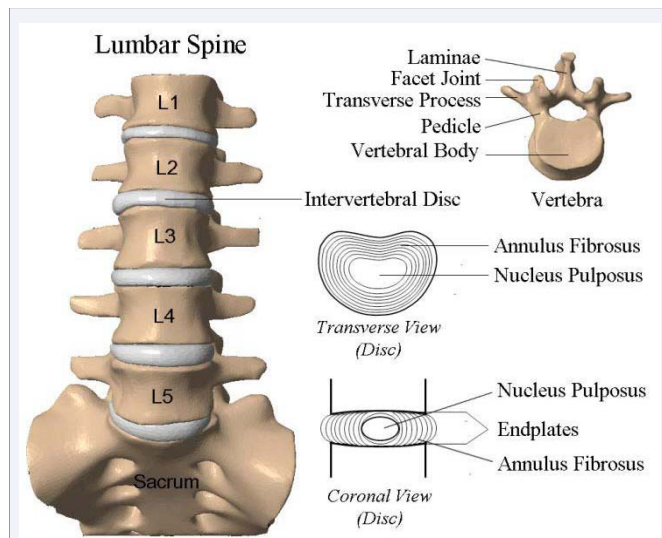


**Figure 3** Creep behavior of intervertebral discs. A 0-3 scale was used to quantify the extent of degradation present, with Grade 0 being a normal disc and the value increasing as the extent of degradation increased. A 93 N load was suddenly impressed and maintained at time zero. Adapted from White and Panjabi [22].

hand, mechanical loading may also play a role on accelerating degeneration [26,27]. An investigation of young adults (20-35 years old) showed disc degeneration or bulging at least one lumbar level [1]. Additionally, the study reported that more than one third of the subjects between 60 and 80 years old presented the same degenerative condition in all but one lumbar disc, which suggested that the lesions revealed by MRIs in elderly are less likely to be of clinical relevance [1]. The direct cause of degeneration is still unknown. The clinical cascade of events can start with at least one risk factor, a condition that leads to increased dehydration and loss of proteoglycan content, which in turns reduces the shock absorption capacity of the intervertebral disc. The results of these physiological changes are increased motion and stress (degeneration 1- 3), which can later trigger various pathologies (i.e. end stage degeneration 5, increased stiffness, herniation, prolapse, etc.). Fujiwara and colleagues [28] observed motion tended to increase with respect to degeneration (up to grade IV) and decreased at grade V. On the other hand, degeneration has shown to increase the tensile stiffness of the annulus [29] and affect the in vitro creep response of the disc [23]; however, no consistent correlation between the elastic behavior of the intervertebral disc and degeneration has been postulated [30]. Alternatively, gender has also shown to play an important role in degeneration. Wang and colleagues [31] found that the prevalence and severity of disc space narrowing is higher in elderly women than in elderly men, with the rate of disc space narrowing greater for females. Although, it is worth noting that Nachemson and colleagues [30] concluded that the disc height of severely degenerated spines was average in both genders. During in vitro testing, female specimens generally display lower mechanical property values relative to males [30,32,33], which has contributed to propose distinctions between female and male range of motion (Table 3).

### Low velocity rear-end collisions mechanism

According to recent information published by the National



**Figure 2** Schematic representation of the lumbar spine (anterior view), a lumbar vertebra (axial view) and the intervertebral disc (transverse and coronal views).

**Table 2:** Lumbar disc cross-sectional area and average disc thickness (from Lin et al. [70]).

Level	Cross-sectional area (mm <sup>2</sup> )	Mean disc thickness (mm)
L1-L2 (6)*	1161 - 2452	7.1 - 10.5
L2-L3 (3)*	1350 - 2207	9.0 - 10.3
L3-L4 (5)*	1510 - 1839	9.1 - 11.9
L4-L5	1632 - 1961	11.7 - 12.5

The number in parenthesis indicates the number of specimens measured.

**Table 3:** Lumbar (mean) range of motion for different age intervals as a function of gender (from Twomey [34]).

Motion	Age (years)	Male (degrees)	Female (degrees)
Flexion	13 - 19	33	42
	20 - 35	33	38
Extension	36 - 59	28	27
	60+	22	22
	13 - 19	9	13
	20 -35	15	18
	36 -59	11	13
	60+	10	10
	13 - 19	16	20
Axial Rotation	20 -35	18	19
	36 -59	13	13
	60+	12	12

Highway Traffic Safety Association [35], rear-end crashes account for approximately 29 percent of all vehicular collisions, making this type of collision the most frequently occurring type as well as a significant subcategory of vehicular collisions from both a public safety and health care viewpoints. The United States Census Bureau [36] reports that there were 10.8 million motor vehicle accidents during the year 2009 in the United States, responsible for more than 2.3 million individuals treated in emergency room facilities with vehicle collision-related injuries [37]. A rear end collision occurs when one vehicle (the “target”) is impacted in the vehicle’s rear by the front of another vehicle (the “bullet” or impacting vehicle). By definition, the velocity of the bullet vehicle has to be greater than that of the target vehicle. In many cases, the “bullet” vehicle is at a total stop or in the process of slowing. Most rear-end collisions display little to no offset, where offset is defined as the extent to which the impact is not “center-to-center”. As offset increases, the possibility that he target vehicle may experience some rotation (yaw)

Increases; otherwise, minimal offset rear-end collisions are characterized by movement in the front-to-back direction.

### Low velocity rear-end impacts, also called low severity and minimal

**Impact:** generally refer to relative impact speeds (or target vehicle delta-v) of less than 8 mph (~13 km/h) or less [38,39], although some investigations may include impacts up to 15 mph in their definition [40]. Normally, such low velocity impacts are characterized by no to limited observable damage to the rear of the target vehicle. The Farmer Study [41] defines a *minor* rearend impact as one where the damage to the rear of the target vehicle is only to the bumper, bumper cover, rear body panel, or tail light assemblies only, with *minor* being equivalent to *low velocity*. It has been suggested that the risk of injury in delta-V between 3 and 5mph is minimal in human volunteers testing [42], however cervical injuries, specifically, are more likely to occur in this type of collisions. A typical rear-end collision consists of several phases and depends on the group (Figure 4). In comparison, many investigators simply describe the occupant kinematics in terms of only 2 or 3 phases. In the 2-phase version, Phase 1 is simply the

period during which the occupant moves backwards and Phase 2 is when the occupant moves forward (relative to the vehicle), until such motion comes to a stop [39]. Most investigators agree that the likelihood of injury is during the initial phase; although, latter phase injuries may still be possible [44,45]. Investigations use a variety of occupants and methods, including: live human subjects (volunteers), anthropomorphic test devices or ATDs (popularly called *crash test dummies*), computer simulations, cadaveric models, and statistical analysis from real world collision data, such that found in the Crash Injury Research and Engineering Network (CIREN) database. Some investigations with primates have been described [46], but bio-fidelity represents an issue. Investigations based on human subjects are typically limited to *low velocity* conditions since safety of the participants needs to be guaranteed and approved by an Institutional Review Board (IRB). Sled testing, which is an enclosed vehicle model (including a car seat, steering wheels, etc.) That moves under controlled conditions, provides a high degree of control and reproducibility. Some studies involve actual vehicles, often impacted by a moving barrier structure that may be either deformable or rigid, but they are usually destructive. Position is an important factor in collision testing. Most rear-end collision studies using human subjects use normal driving or sitting positions, where some restraints are used; the occupant is looking straight ahead, the torso is in contact with the seat back, feet on the floor and hands on the steering wheel (for a driver) or in lap (for a passenger). Alternative positions, such as leaning forward or sideways, head facing left or right and upper torso rotated, are possible; these have been shown to potentially have greater risk of injury on cervical region [47]. However, information related to the effects of out-of-position in lumbar injuries is limited. Gushue et al. [48] concluded that sitting position was a factor associated with the magnitude of the peak forces (compression and shear) experienced in the lumbar spine during simulated low- speed rear impacts; however, they acknowledged these forces were below the magnitudes reported in the literature associated with lumbar injuries. Seat back properties have a significant influence on injury risk in rear-end collisions. For example, Viano [49] found that seat made of low stiffness material and stronger frame reduces the risk of cervical injury. Minton and colleagues [50] found that the incidence of lumbar strain correlates with seat back positioning, noting that greater seat back decline angle may increase the gap between the shoulders and the seat back. Although no directly related to a car seat, Hoyes & Henderson [42] showed how different chair designs/materials can affect the acceleration patterns of the head, chest and lower back experienced during a sitting simulation, demonstrating that the delta-V threshold experienced in everyday activities, such as sitting, could be similar to those reported in a low speed rear-collision. Rear-end collisions can be characterized as low force collisions. Figure 5 presents the results from a study performed by Szabo & Welcher [51] where the acceleration (in terms of the force of gravity, G) in a forward-backward direction of the head and lumbar region were compared to the vehicle’s acceleration, as a function of time. This study used human volunteer subjects and involved actual vehicle-to-vehicle (both vehicles were Volkswagens) impacts. Although lumbar acceleration history may be qualitatively similar to that of the vehicle history, many studies have shown that the head may experience accelerations in

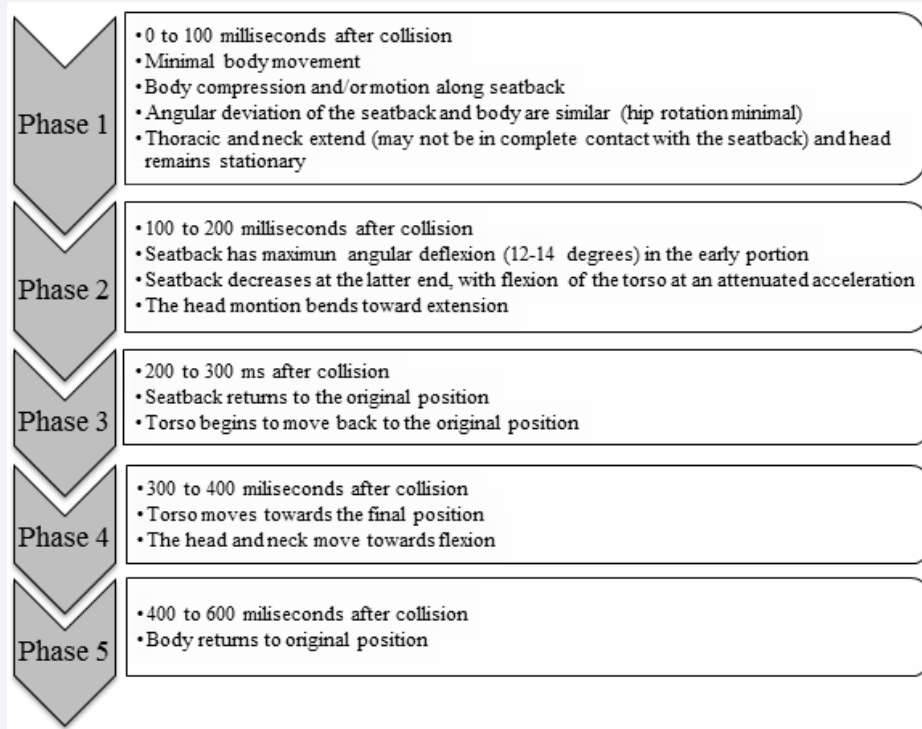


Figure 4 Phases on rear-end collision described by McConnell [43].

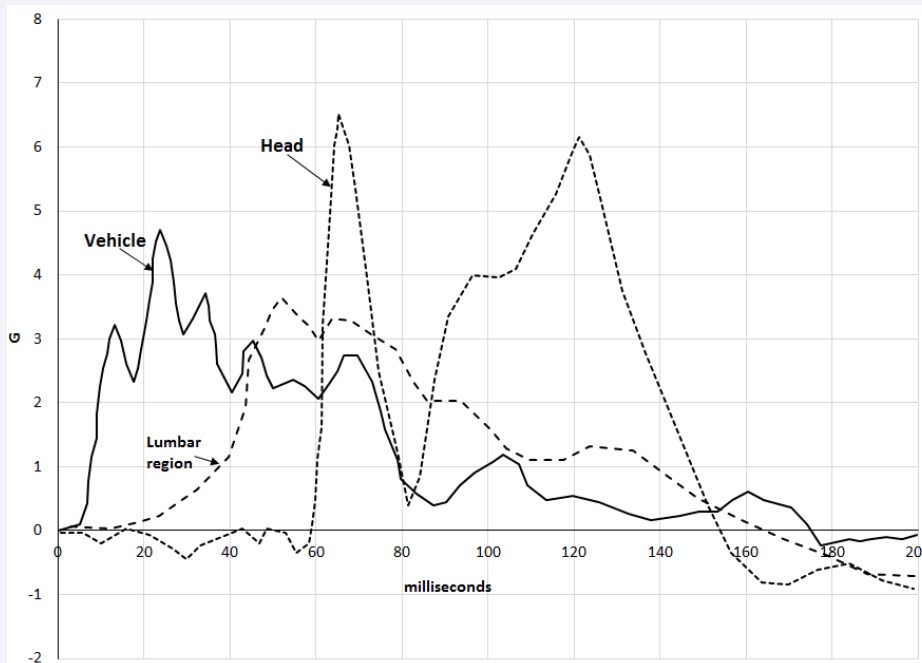


Figure 5 Representation of vehicle, head and lumbar accelerations as a function of time during a rear-end impact at 13.8 km/h (8.6mph) (adapted from Szabo & Welcher [51]).

the range of 2 to 3 times that of the vehicle [52,53], while the lumbar region experiences peak accelerations even lower than the maximum acceleration observed at the vehicle. Moreover, an examination to the National Automotive Sampling System (NASSCDS) database from 1993 to 2009 suggested that the

incidence of lumbar injuries (mostly musculoskeletal strains/sprains) is small for low, medium and even high speed rear impact crashes [54]. Likewise, investigations using ATDs suggest that low- to moderate-speed impacts are unlikely to cause significant damage to the lumbar discs [55]. The accelerations experience

by a subject during a low velocity rear-end collision (i.e. 3.4 to 3.8 g for 9-10 mph delta-v impacts and less than 2 g for 5 mph delta-v impacts) are comparable to so-called activities of daily living (ADLs) (Table 4). While these ADLs do not model the body response in a rear end collision, it does illustrate the observation that the forces one experiences in a rear-end collision (in terms of lumbar forces) may not exceed the ones experienced during some ADLs.

**Lumbar Disc Injuries:** Disc failure can be seen in perspective of a domino effect where asymptomatic degenerative changes in some regions could generate strain in adjacent structures that can later become symptomatic. Biomechanical properties of intervertebral discs are usually reported in terms of failure values (ultimate stress), frequently neglecting details about the elastic region limits in the stress-strain curve, the yield stress. While the failure values are certainly important, yield stresses may also play a role in understanding acute injuries and degenerative processes. As Adams and Hutton [19] have observed, spinal elements subjected to pure symmetrical compression display a nonlinear behavior as a result of collagen content. Compressive failure, specifically, is indicated (experimentally) when there is a splitting of the annulus and nucleus pulposus material interfaces. Most biomechanical data reflects experimental protocols employing symmetrical (axial) compression. However, most spinal body motions involve combined loading between compression, flexion/extension, lateral bending and/or axial torsion. Indeed, intervertebral disc failure associated with disc material bulging/rupture has been reported to most likely occur in the presence of combined loading [58]. As one example, flexion/extension establishes a compressive load in the anterior region of the disc and a tension load in the posterior

region (and vice versa for extension/flexion). Experimental data using lumbar cadaveric segments has shown how intervertebral discs can gradually prolapse, suggesting that this progressive event can evolve with time, especially in young discs which are considered to be at a higher risk. Although the time scale is still unknown, Adams and Hutton identified five important stages of this condition, which gradual disc prolapse begins with the self-selection of the disc (stage 1), followed by the distortion (stage 2) and breaking through (stage 3-equivalent to disc herniation) the lamellae of the annulus, and finalizes with the extrusion (stage 4) and rupture (stage 5) of the nuclear pulp. It is not well-understood at which stage pain occurs, however, it is likely to happen at stage 3. Sudden prolapse can also occur in the various stages described, being mature discs are a higher risk. [59] In terms of disc herniation mechanics, Adams and Hutton [60] demonstrated how a combination of hyper-flexion and compressive load in a laboratory setting (using human cadaveric spinal segments) can induce intervertebral disc prolapse, suggesting that compound motions, disc degeneration (i.e. aging) and the spinal level are three important factors influencing the probability of this condition. In addition, Callaghan & McGill [61] observed that there is a significant portion of the literature suggesting, directly and indirectly, that disc herniation requires the spine to be in a non-neutral position. Regardless the type of event, the rate, magnitude and direction of the force experienced by the spine during an injury dictates the characteristics of the injury. As described by White & Panjabi [58], pure compression forces, for example, trigger compression fracture with central concavity, as opposed to wedge compression of the vertebral body observed during lateral compression. Computer modeling has revealed that initiation sites of lumbar spinal injuries under

**Table 4:** Reported acceleration in the lumbar region of human subjects during activities of daily Living.

Activity	Reported G-force	Reference
Normal walking	1.45 - 2.07	Twomey [34]
Fast walking	2.95	Cheng [56]
Plop backwards into a soft chair	3.5 ± 1.0	Lee & Barnes [57]
Stepping of step 15.24 cm (6 inch) height	3.5 ± 2.6	Lee & Barnes [57]
Jogging in place	4.7 ± 2.0	Lee & Barnes [57]

**Table 5:** Possible lumbar intervertebral disc injury mechanisms.

Mechanism	Possible Injury Mechanism(s)
Axial Compression	<ul style="list-style-type: none"> <li>✓ It is not clear that axial compression occurs to any significant degree (in the absence of head contact with the vehicle roof)</li> <li>✓ At high speed collisions: Minor axial compression could occur during the "rebound"</li> </ul>
Compression in general	<ul style="list-style-type: none"> <li>✓ Some posterior compression could occur during lumbar extension (Impact Phase)</li> <li>✓ Some lateral compression could occur with significant rotational component (yaw), especially when the occupant contacts a vehicle door or other interior structure</li> <li>✓ Some anterior compression may occur during lumbar flexion (Rebound Phase)</li> </ul>
Tension (distraction)	<ul style="list-style-type: none"> <li>✓ Anterior tension/distraction could occur during lumbar extension (impact phase)</li> <li>✓ At high speed collisions: Tension could occur in restrained occupants (Impact Phase) when occupant "climbs" the seat back</li> </ul>
Shear	<ul style="list-style-type: none"> <li>✓ No obvious lumbar shear occurs (when lumbar region is in direct contact with the seat back -likely for restrained occupants)</li> <li>✓ Some shear may be experienced in the presence of seats with pronounced lumbar support systems</li> </ul>
Axial Rotation	
Axial Compression and Rotation	

low-dynamic (displacement rate of 0.1 m/s) forces are posterior vertebral fracture with rupture of interspinous ligament and facet joint capsules during flexion motion, while anterior vertebral body and interspinous process fractures occur in the initial stage of an injury during extension [62]. In contrary, if the force is applied at a high rate (4 m/s), the initiation sites of injury are anterior vertebral body fracture (through endplate rupture) with rupture of interspinous ligament for flexion, and anterior vertebral body fracture with posterior endplate rupture (vertebral body fracture) for extension [62]. In the attempt of understanding how healthy lumbar discs herniate, Wade et al. [63] evaluated the combined effects of compression rate and flexion; It was found that disc wall failure can occur at 8,900 N (range 4,900-14,200) in an isolated ovine lumbar segment, when it was exposed to both flexion and elevated rate of compression. The investigators recognized the mechanism of disc herniation is not well-understood and revealed the literature suggests non-flexed segments tend to fail via endplate and/or vertebral body fractures, while flexion increases the likelihood of disc (annular) failure. In terms of type of loading, repetitive loading have been suggested to be a greater influencing factor than compressive loading in non-degenerated disc herniation [61]. Moreover, these observations, besides emphasizing the importance of bending motion in the mechanism of disc injury, highlight how sagittal deformities may also play an important role in lumbar disc injuries.

**From a low-speed rear-end collision:** Interestingly, current crashworthiness testing, as conducted by the National Highway Traffic Safety Administration's (NHTSA) under the New Car Assessment Program (NCAP), derives the "star safety ratings" that only consider measurements involving: (a) the head, neck, chest, pelvis, and femur (legs) in the event of a frontal impact, (b) the head, chest, abdomen, and pelvis for front seat passengers in the event of a side-impact crash and (c) the head and pelvis for rear-seated occupants in the event of a side-impact crash. There are no criteria for rear-end collisions. NHTSA has noted that due to a limited budget, their testing program concentrates on collision types (frontal and side impact) that are responsible for the vast majority of deaths and 11 serious injuries. Additionally, more "technical testing" protocols (separate from "star safety ratings"), current NCAP protocol requires information regarding head acceleration (based on Head Injury Criterion, HIC), Chest Severity Index (CSI), Thoracic Trauma Index (TTI), pelvic acceleration, and femur loading [64]. Many investigators have contributed to the on-going research on the development of neck injury criteria; however, the efforts on developing lumbar-specific injury criteria appear to be minimal. Flexion-distraction injuries from high-speed motor vehicle accidents have been reported in the literature [6], suggesting the incidence in children may be higher than in adults [5,7]. A possible explanation to this incidence is that a child's center of gravity is relatively higher than that of an adult. Rao and colleagues [7] studied the occurrence of thoracic and lumbar spine injuries in moderate to high velocity impacts (of which many were frontal collisions), reporting that flexion-distraction injuries were more common in children and young adults and extension injuries were more common in older adults. Vertebral fractures were identified as common injuries. Table 5 summarizes how different injury mechanisms might be

established in a low velocity rear-end vehicular collision. The literature indicates that soft tissue sprain-strain injuries may occur; such injuries usually resolve after conservative treatment. To our knowledge, most injuries during collision events have been reported for the cervical spine during high-speed collisions, where the incidence of soft tissue injuries appears to be small, and has also been even reported inconsistent [65]. An extensive review performed by White & Panjabi [58] suggested the first component to fail under compressive forces is the vertebra via end-plate fracture, while torsional forces may produce annular tears in the absence of an endplate injury. Consequently, the notion of a healthy disc injury during a low speed rear-end collisions is vague, especially since the forces experienced by the lower spine may be even less hazardous due to the common immobilizing action of the seat belt. However, a review in minor rear crash forensic engineering data performed by Bartsch et al. [40] revealed the diagnostics of AIS1 injury (minor injury according to the abbreviated injury scale) was higher in the lumbar/sacral spine than those in the cervical region, without proposing a possible explanation. The fact that the lumbar region is daily exposed to larger (compression) forces (greater fatigue) could represent more risk for injury (and degeneration) during ADLs. Thus, the incidence of pain may be greater in the lumbar/sacral spine than in other regions, meaning there may be higher chances of lumbar/sacral injury diagnoses related to "asymptomatic" pre-existing conditions after a low speed rear-end collision event. On the other hand, aging and degeneration may affect the mechanisms of injury during a low velocity rear end vehicular collision previously described, since the biomechanical properties of the disc are different to those of a healthy disc (refer to *Aging and degeneration* discussion). Nevertheless, Bartsch et al [40] also noted that the diagnosed of AIS1 lumbar/sacral injury rate was equivalent in the degenerative and non-degenerative lumbar/sacral occupants. Asymptomatic intervertebral disc changes due to normal aging could also contribute to the erroneous association of a pre-existing condition with a single low-speed collision event. Erkintalo et al. [66] demonstrated how degenerative changes occur in both symptomatic and asymptomatic adolescents suggesting, however, that those with recurrent or chronic back pain are more susceptible to earlier and more frequent signs. Jensen et al. [67] reported the prevalence of abnormal findings in at least one lumbar intervertebral disc, after MRI examination, to be higher than 35% in asymptomatic subjects between 20 and 80 years old, with disc bulging the most common pathology. Likewise, Boden et al. [1] found that about 30% of 67 asymptomatic young adults (20-35 years old) with no history of back pain, sciatic or neurologic claudication, presented disc bulging or degeneration in at least one lumbar disc. Lumbar loading in a non-offset rear-end collision is the result of acceleration/deceleration only; there are no blunt trauma mechanisms resulting from forceful contacts with vehicle interior surfaces. The seat back is compressed by the lower torso and then the seat back pushes on the lower torso. The overall lumbar movement is limited, with the entire lumbar region (total length < 20 cm) moving as a unit. Some investigators speculate that disc injuries in rear-end collisions may require more than one form of biomechanical loading, which goes in line with the observation that combined loading is needed for a disc injury to occur. For example, Veres and colleagues [68] suggested the incidence of

annular tears and endplate rupture from the combination of torsion and flexion on an ovine lumbar herniated disc. From a broader perspective, it has been observed that chronic pain after vehicular collisions with no to minimal vehicle damage is less common in countries without the possibility of financial compensation (particularly beyond medical and repair costs) for accident victims [69]. One of the major challenges for conducting retrospective analyses using existing data in minor collisions is the limited availability of medical records of the participants prior to the collision, particularly imaging studies. On the other hand, the feasibility of performing prospective studies is limited. A thorough review of the relevant literature to explore the causal relationship (if any) between lumbar disc injuries and vehicular collisions (particularly low velocity events) would provide a more informed basis for any conclusions. The above discussion is limited to low velocity rear-end impacts with minimal to no offset. While there is evidence to suggest that lumbar disc injuries, such as annular tears, may result from such collisions when significant vehicular rotation (yaw) is also experienced, this is another area that needs further exploration. Scientific data available is limited, which may jeopardize identifying soft tissue injuries during low-speed collision events. Likewise, the effect of important factors, such as aging, degeneration, vehicle ergonomics and engineering, is not fully understood.

## CONCLUSIONS

There is not enough scientific evidence to support the claim that a lumbar disc injury occurs as the result of a low velocity collision, particularly during rear-end vehicular collisions. If soft tissue injuries occur, they may resolve after conservative treatment. However, the causal relationship between low velocity impacts and healthy disc pathologies has no scientific or medical foundations. From a biomechanical viewpoint, one must determine if any mechanism for such injuries occurs and if the associated forces are consistent with such injuries. Other investigators have suggested that such may have a psychological factor and/or to postural corrections induced by post-collision treatment (i.e. physical therapy). The notion of a disc injury from a low-speed rear-end collision being a litigation-driven event (i.e. financially driven) has not been eliminated.

## REFERENCES

1. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am.* 1990; 72: 403-408.
2. Wiesel SW, Tsourmas N, Feffer HL, Citrin CM, Patronas N. A study of computer-assisted tomography. I. The incidence of positive CAT scans in an asymptomatic group of patients. *Spine (Phila Pa 1976).* 1984; 9: 549-551.
3. Hitselberger WE, Witten RM. Abnormal myelograms in asymptomatic patients. *J Neurosurg.* 1968; 28: 204-206.
4. Pearce JM. Aspects of the failed back syndrome: role of litigation. *Spinal Cord.* 2000; 38: 63-70.
5. Rumball K, Jarvis J. Seat-belt injuries of the spine in young children. *J Bone Joint Surg Br.* 1992; 74: 571-574.
6. Knox JB, Wimberly RL, Riccio AI. Pediatric lateral distraction injury of the lumbar spine: a case report. *Spine J.* 2013; 13: e45-48.
7. Rao RD, Berry CA2, Yoganandan N3, Agarwal A2. Occupant and crash characteristics in thoracic and lumbar spine injuries resulting from motor vehicle collisions. *Spine J.* 2014; 14: 2355-2365.
8. Gray H, WH Lewis, *Anatomy of the Human Body.* 20th ed. 1918, Philadelphia and New York: Lea & Febiger.
9. Videman T, Battié MC, Gill K, Manninen H, Gibbons LE, Fisher LD. Magnetic resonance imaging findings and their relationships in the thoracic and lumbar spine. Insights into the etiopathogenesis of spinal degeneration. *Spine (Phila Pa 1976).* 1995; 20: 928-935.
10. Shirazi-Adl A. Analysis of large compression loads on lumbar spine in flexion and in torsion using a novel wrapping element. *J Biomech.* 2006; 39: 267-275.
11. Grieve G, *Common Vertebra. Joint Problems.* New York, Chirchili-Livingstone, 1981:82-124.
12. Bible JE, Biswas D, Miller CP, Whang PG, Grauer JN. Normal functional range of motion of the lumbar spine during 15 activities of daily living. *J Spinal Disord Tech.* 2010; 23: 106-112.
13. Pearcy M, Portek I, Shepherd J. Three-dimensional x-ray analysis of normal movement in the lumbar spine. *Spine (Phila Pa 1976).* 1984; 9: 294-297.
14. Lin HS, Liu YK, Adams KH. Mechanical response of the lumbar intervertebral joint under physiological (complex) loading. *J Bone Joint Surg Am.* 1978; 60: 41-55.
15. Farfan HF, Cossette JW, Robertson GH, Wells RV, Kraus H. The effects of torsion on the lumbar intervertebral joints: the role of torsion in the production of disc degeneration. *J Bone Joint Surg Am.* 1970; 52: 468-497.
16. EVANS FG, LISSNER HR. Biomechanical studies on the lumbar spine and pelvis. *J Bone Joint Surg Am.* 1959; 41-41A: 278-90.
17. Yamada, HEFG, *Strength of Biological Materials.* 1973, Huntington, N.Y.: Robert E. Krieger Pub. Co.
18. Andersson GB, McNeill TW, *Lumbar Spine Syndromes: Evaluation and Treatment.* 1989: Springer-Verlag.
19. Adams MA, Hutton WC. *Mechanics of the Intervertebral Disc. The biology of the intervertebral disc.* 1988; 2:39-71.
20. Wilder DG, Pope MH, Frymoyer JW. The biomechanics of lumbar disc herniation and the effect of overload and instability. *J Spinal Disord.* 1988; 1: 16-32.
21. Costi JJ, Stokes IA, Gardner-Morse M, Laible JP, Scoffone HM, Iatridis JC. Direct measurement of intervertebral disc maximum shear strain in six degrees of freedom: motions that place disc tissue at risk of injury. *J Biomech.* 2007; 40: 2457-2466.
22. White AA, Panjabi MM, *Clinical Biomechanics of the Spine.* 1978: Lippincott.
23. Kazarian L, GA Graves, *Compressive Strength Characteristics of the Human Vertebral Centrum.* *Spine (Phila Pa 1976),* 1977; 2:1-14.
24. Battié MC, Haynor DR, Fisher LD, Gill K, Gibbons LE, Videman T. Similarities in degenerative findings on magnetic resonance images of the lumbar spines of identical twins. *J Bone Joint Surg Am.* 1995; 77: 1662-1670.
25. Videman T, Leppävuori J, Kaprio J, Battié MC, Gibbons LE, Peltonen L. Intragenic polymorphisms of the vitamin D receptor gene associated with intervertebral disc degeneration. *Spine (Phila Pa 1976).* 1998; 23: 2477-2485.
26. Handa T, Ishihara H, Ohshima H, Osada R, Tsuji H, Obata K. Effects of hydrostatic pressure on matrix synthesis and matrix metalloproteinase



- production in the human lumbar intervertebral disc. *Spine (Phila Pa 1976)*. 1997; 22: 1085-1091.
27. Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? *Spine (Phila Pa 1976)*. 2006; 31: 2151-2161.
28. Fujiwara A, Lim TH, An HS, Tanaka N, Jeon CH, Andersson GB. The effect of disc degeneration and facet joint osteoarthritis on the segmental flexibility of the lumbar spine. *Spine (Phila Pa 1976)*. 2000; 25: 3036-3044.
29. Acaroglu ER, Iatridis JC, Setton LA, Foster RJ, Mow VC, Weidenbaum M. Degeneration and aging affect the tensile behavior of human lumbar annulus fibrosus. *Spine (Phila Pa 1976)*. 1995; 20: 2690-2701.
30. Nachemson AL, Schultz AB, Berkson MH. Mechanical properties of human lumbar spine motion segments. Influence of age, sex, disc level, and degeneration. *Spine (Phila Pa 1976)*. 1979; 4: 1-8.
31. Wang YX, Griffith JF, Zeng XJ, Deng M, Kwok AW, Leung JC. Prevalence and sex difference of lumbar disc space narrowing in elderly chinese men and women: osteoporotic fractures in men (Hong Kong) and osteoporotic fractures in women (Hong Kong) studies. *Arthritis Rheum*. 2013; 65: 1004-1010.
32. Wang YX, Griffith JF, Zeng XJ, Deng M, Kwok AW, Leung JC. Prevalence and sex difference of lumbar disc space narrowing in elderly chinese men and women: osteoporotic fractures in men (Hong Kong) and osteoporotic fractures in women (Hong Kong) studies. *Arthritis Rheum*. 2013; 65: 1004-1010.
33. Nachemson AL, Schultz AB, Berkson MH. Mechanical properties of human lumbar spine motion segments. Influence of age, sex, disc level, and degeneration. *Spine (Phila Pa 1976)*. 1979; 4: 1-8.
34. Stemper BD, Board D, Yoganandan N, Wolfla CE. Biomechanical properties of human thoracic spine disc segments. *J Craniovertebr Junction Spine*. 2010; 1: 18-22.
35. Twomey L. The effects of age on the ranges of motions of the lumbar region. *Aust J Physiother*. 1979; 25: 257-263.
36. Lee S, Llaneras E, Klauer S, Sudweeks J, Analyses of Rear-End Crashes and near-Crashes in the 100-Car Naturalistic Driving Study to Support Rear-Signaling Countermeasure Development. DOT HS, 2007; 810:846.
37. United States Census Bureau. (2012). the 2012 Statistical Abstract. Transportation: motor vehicle accidents and fatalities.
38. Centers for Disease Control and Prevention (CDC). Vital signs: nonfatal, motor vehicle--occupant injuries (2009) and seat belt use (2008) among adults --- United States. *MMWR Morb Mortal Wkly Rep*. 2011; 59: 1681-1686.
39. McConnell W, Howard R, Guzman HB, Bomar JB, Raddin JH, Benedic JV, Smith HL, et al. Analysis of Human Test Subject Kinematic Responses to Low Velocity Rear End Impacts. 1993, SAE International: SAE Technical Paper.
40. Szabo TJ, Welcher JB, Anderson RD, Rice MM, Ward JA, Paulo LR, et al. Human Occupant Kinematic Response to Low Speed Rear-End Impacts. SAE Technical Paper 1994; 940532.
41. Bartsch AJ, Gilbertson LG, Prakash V, Morr DR, Wiechel JF. Minor crashes and 'whiplash' in the United States. *Ann Adv Automot Med*. 2008; 52: 117-128.
42. Farmer CM, Wells JK, Werner JV. Relationship of head restraint positioning to driver neck injury in rear-end crashes. *Accid Anal Prev*. 1999; 31: 719-728.
43. Hoyes P, Henderson B. A study and comparison of the effects of low speed change vehicle collisions on the human body. *Accid Anal Prev*. 2013; 51: 318-324.
44. McConnell W, Howard R, Poppel JV, Krause R, Guzman HM, Bomar JB. Human Head and Neck Kinematics after Low Velocity Rear-End Impacts - Understanding "Whiplash". SAE Technical Paper. 1995; 952724.
45. Muser M, Walz FH, Zellmer H. Biomechanical Significance of the Rebound Phase in Low Speed Rear End Impacts. in Proceedings of the 2000 International Ircobi Conference on the Biomechanics of Impact. 2000.
46. Viano DC, Parenteau CS, Burnett R. Rebound after Rear Impacts. *Traffic Inj Prev*. 2013; 14: 181-187.
47. Yarnell P, Ommaya AK. Experimental cerebral concussion in the rhesus monkey. *Bull N Y Acad Med*. 1969; 45: 39-45.
48. Keifer O, Layson P, Reckamp B. The Effects of Seated Position on Occupant Kinematics in Low- Speed Rear-End Impacts. SAE Technical Paper 2005.
49. Gushue DL, Probst BW, Benda B, Joganich T, McDonough D, Markushewski ML. Effects of Velocity and Occupant Sitting Position on the Kinematics and Kinetics of the Lumbar Spine During Simulated Low-Speed Rear Impacts. American Society of Safety Engineers.
50. Viano DC. Seat design principles to reduce neck injuries in rear impacts. *Traffic Inj Prev*. 2008; 9: 552-560.
51. Minton R, Murray P, Pitcher M, Galasko C. Lower Back and Neck Strain Injuries: The Relative Roles of Seat Adjustment and Vehicle/Seat Design. in Proceedings of the 16th International Technical Conference on the Enhanced safety of Vehicles. Windsor, Ont, Canada. 1998.
52. Szabo TJ, Welcher JB, Human Subject Kinematics and Electromyographic Activity During Low Speed Rear Impacts. SAE Technical Paper, 1996.
53. Hoyes P, Henderson B. A study and comparison of the effects of low speed change vehicle collisions on the human body. *Accid Anal Prev*. 2013; 51: 318-324.
54. McKenzie JA, Williams JF. The dynamic behaviour of the head and cervical spine during 'whiplash'. *J Biomech*. 1971; 4: 477-490.
55. Gates, D, Bridges A, Welch TDJ, XLam TDJ, Scher I, Yamaguchi G, Lumbar Loads in Low to Moderate Speed Rear Impacts. SAE International, 2010.
56. Cheng CK, Chen HH, Chen CS, Lee SJ. Influences of walking speed change on the lumbosacral joint force distribution. *Biomed Mater Eng*. 1998; 8: 155-165.
57. Lee WE, Barnes JL. Activities of Daily Living: Lumbar Region Measurements. in Biomedical Engineering Society Annual Meeting. 2007. Los Angeles, CA.
58. White AA, Panjabi MM, Clinical Biomechanics of the Spine. 1990: Lippincott.
59. Adams MA, Hutton WC. Gradual disc prolapse. *Spine (Phila Pa 1976)*. 1985; 10: 524-531.
60. Adams MA, Hutton WC. Prolapsed intervertebral disc. A hyperflexion injury 1981 Volvo Award in Basic Science. *Spine (Phila Pa 1976)*. 1982; 7: 184-191.
61. Callaghan JP, McGill SM. Intervertebral disc herniation: studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. *Clin Biomech (Bristol, Avon)*. 2001; 16: 28-37.
62. Wagnac E, Arnoux PJ, Garo A, Aubin CE. Finite element analysis of the influence of loading rate on a model of the full lumbar spine under dynamic loading conditions. *Med Biol Eng Comput*. 2012; 50: 903-915.
63. Wade KR, Robertson PA, Thambyah A, Broom ND. How healthy discs herniate: a biomechanical and microstructural study investigating

- the combined effects of compression rate and flexion. *Spine (Phila Pa 1976)*. 2014; 39: 1018-1028.
64. National Highway Traffic Safety Administration, Vehicle Safety Research. (2013). NHTSA Test Guide. Version 5. Volume 1: Vehicle Tests.
65. Yoganandan N, Pintar FA. Biomechanics of Human Head-Neck in Rear Impacts. *International journal of vehicle design*, 2003; 32: 53-67.
66. Erkintalo MO, Salminen JJ, Alanen AM, Paaianen HE, Kormanen MJ. Development of degenerative changes in the lumbar intervertebral disk: results of a prospective MR imaging study in adolescents with and without low-back pain. *Radiology*. 1995; 196: 529-533.
67. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic Resonance Imaging of the Lumbar Spine in People without Back Pain. *N Engl J Med*. 1994; 331:69-73.
68. Veres SP, Robertson PA, Broom ND. The influence of torsion on disc herniation when combined with flexion. *Eur Spine J*. 2010; 19: 1468-1478.
69. Laborde JM. Biomechanics of minor automobile accidents: treatment implications for associated chronic spine symptoms. *J South Orthop Assoc*. 2000; 9: 187-192.
70. Lin HS, Liu YK, Adams KH. Mechanical response of the lumbar intervertebral joint under physiological (complex) loading. *J Bone Joint Surg Am*. 1978; 60: 41-55.

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