

To Crunch or Not to Crunch: An Evidence-Based Examination of Spinal Flexion Exercises, Their Potential Risks, and Their Applicability to Program Design

Bret Contreras, MA, CSCS¹ and Brad Schoenfeld, MSc, CSCS²

¹Auckland University of Technology, Auckland, New Zealand; and ²Global Fitness Services, Scarsdale, New York

SUMMARY

THE CRUNCH AND ITS MANY VARIATIONS HAVE LONG BEEN CONSIDERED A STAPLE EXERCISE IN FITNESS PROGRAMS. HOWEVER, RECENTLY, SOME FITNESS PROFESSIONALS HAVE QUESTIONED THE WISDOM OF PERFORMING FLEXION-BASED SPINAL EXERCISES, SUCH AS THE CRUNCH. CONCERNS ARE USUALLY PREDICATED ON THE BELIEF THAT THE SPINE HAS A FINITE NUMBER OF BENDING CYCLES AND THAT EXCEEDING THIS LIMIT WILL HASTEN THE ONSET OF VERTEBRAL DEGENERATION. THIS ARTICLE WILL SEEK TO REVIEW THE RESEARCH PERTAINING TO THE RISKS OF PERFORMING DYNAMIC SPINAL FLEXION EXERCISES AND WILL DISCUSS THE APPLICATION OF THESE FINDINGS TO EXERCISE PERFORMANCE.

The crunch and its many variations have long been considered a staple exercise in fitness programs. These exercises involve dynamic flexion of the spine in the sagittal plane and are performed to increase abdominal strength and development (124), particularly in the rectus abdominis and obliques musculature. Strength and conditioning coaches frequently include such exercises as a component of athletic routines designed to enhance sporting performance (45).

Recently, however, some fitness professionals have questioned the wisdom of performing flexion-based spinal exercises, such as the crunch (23,75,110). Concerns are usually predicated on the belief that the spine has a finite number of bending cycles and that exceeding this limit will hasten the onset of disc damage (75). Proponents of the theory claim that spinal flexion therefore should be saved for activities of daily

living such as tying one's shoes rather than "wasted" on crunches and other flexion-based abdominal exercises. Opponents of the theory counter that an alarming discrepancy exists between laboratory results and what is occurring in gyms and athletic facilities around the world with respect to total flexion cycles and spinal injury and cite a lack of evidence showing any detriments. Therefore, the purpose of this article will be 3-fold: First, to review the relevant research pertaining to the risks of performing dynamic spinal flexion exercises; second, to explore the potential benefits associated with spinal flexion exercises; and third, to discuss the application of these findings to exercise program design.

KEY WORDS:

spinal flexion; crunch; trunk flexion; spinal biomechanics

OVERVIEW OF DEGENERATIVE DISC DISEASE

The intervertebral discs form cartilaginous joints between adjacent vertebrae, which stabilize the spine by anchoring the vertebrae to one another. The discs also facilitate multiplanar spinal movement and help absorb vertebral shock. Discs have 3 distinct portions: an outer layer annular fibrosus, a central nucleus pulposus, and 2 hyaline cartilage end plates (64). The annulus, which has an inner and outer component, consists of multiple layers of fibrocartilage, primarily a combination of type I and type II collagen (39). The annulus serves to resist outward pressure, also known as tensile or hoop stresses, during axial compression and to stabilize the vertebral joint during motion (138). The annulus also serves to contain the inner nucleus, which is a gel-like structure composed of a mixture of chondrocytes, collagen, elastin, and proteoglycans (130). Proteoglycans serve to resist compressive loading because of their glycosaminoglycan (GAGs) content (114). Glycosaminoglycans are long-branch polysaccharides that attract and bind to water and provide osmotic pressure. The nucleus functions as a “water pillow,” helping to cushion the vertebrae from axial loads and distribute pressures uniformly over adjacent vertebral end plates (111). The end plates contain primarily type II collagen (55), are less than 1 mm thick, and contain fibers that extend into the disc (138). In addition to preventing the nucleus from protruding into adjacent vertebrae, the end plates also help to absorb hydrostatic pressure caused by spinal loading (26,81) and allow for nutrient diffusion (131).

Degenerative disc disease is a multifactorial process involving genetic, mechanical, biological, and environmental factors (59). The first common signs of disc degeneration often appear between 11 and 16 years of age, with approximately 20% of teenagers displaying mild disc degeneration (79). However, minor signs of degeneration, such as mild cleft formation and granular changes to the nucleus, appear in disc of 2 year olds

(21). Discs tend to progressively deteriorate with age, with a majority of discs showing signs of degeneration by the time a person is 70 years old (79). Age-related degeneration involves a reduction in proteoglycan and collagen levels (114), a 5-fold reduction in the fixed charge density (a measure of mechanochemical strength) of GAGs in the nucleus (60), and a 2-fold decrease in hydration between adolescent discs and discs of 80 year olds (129), which diminishes the disc’s height and load-bearing capabilities (8,22). Men tend to exhibit more disc degeneration than women, which is thought to be because of a combination of increased trunk strength, increased resistance lever arms that heighten spinal forces and stresses, increased heavy loading, and increased distance for nutrient travel (79).

Intervertebral disc degeneration can manifest from a structural disturbance in the annulus, nucleus, or end plate (7). Aging, apoptosis, collagen abnormalities, vascular ingrowth, mechanical loading, and proteoglycan abnormalities can all contribute to disc degeneration (71). As discs degenerate, focal defects arise in the cartilage end plate, the nuclei become increasingly more consolidated and fibrous, and the number of layers in the annulus diminishes (119). This has been shown to alter disc height, spinal biomechanics, and load-bearing capabilities (99) and ultimately can lead to spinal stenosis—an advanced form of degenerative disc disease that causes compression of the contents of the spinal canal, particularly the neural structures (93). End plate calcification also contributes to disc degeneration by decreasing nutrient diffusion that interferes with the pH balance and increases inflammatory responses in the nucleus (34). Yet despite a clear association between degenerative spinal changes and an increased incidence of lower back pain (LBP) (65), many afflicted individuals are nevertheless asymptomatic (19,20,139).

DOES SPINAL FLEXION CAUSE DISC INJURY?

A variety of research approaches have been used to elucidate spinal

biomechanics and their impact on disc pathophysiology, including the use of animal and human in vivo (i.e., within the living) models, animal and human in vitro models (i.e., within the glass), and computer-based in silico models (63). In particular, in vitro research has implicated repetitive lumbar flexion as the primary mechanism of disc herniation (protrusion of disc material beyond the confines of the annular lining) and prolapse (a bulging of nucleus pulposus through annulus fibrosus) because evidence shows that these pathologies proceed progressively from the inside outward through nuclear migration toward the weakest region of the annulus, the posterolateral portion (62,127).

Most in vitro studies on spinal biomechanics that are applicable to the crunch exercise have used cervical porcine models (30,35,36,70,123). These models involve mounting spinal motion segments in custom apparatuses that apply continuous compressive loads combined with dynamic flexion and extension moments. Total bending cycles have ranged from 4,400 to 86,400, with compression loads equating to approximately 1,500 N. Considering that Axler and McGill (13) found that a basic crunch variation elicited around 2,000 N of compression, the amount of compression in the various studies is reasonable for making comparisons with the crunch exercise. In each of the aforementioned studies, a majority of the discs experienced either complete or partial herniations, particularly to the posterior annulus. This suggests a cause-effect relationship between spinal flexion and disc damage. The results of the studies are summarized in the Table.

Although the aforementioned studies seem to lend credence to the potential risks of repeated spinal bending, there are several issues with attempting to extrapolate conclusions from a laboratory setting to the gym. First and foremost, the studies in question were performed in vitro, which is limited by the removal of musculature and does not replicate the in vivo response to the

Table
Summary of in vitro studies on spinal biomechanics reporting spinal compression forces applicable to the crunch exercise

Study	Type of Spine	Number of Subjects	Amount of Compression, N	Number of Cycles	Number of Herniations
Callaghan and McGill (30)	Porcine cervical	26	260–1,472	86,400	15
Drake et al. (35)	Porcine cervical	9	1,472	6,000	7
Tampier et al. (123)	Porcine cervical	16	1,472	4,400–14,00	8
Drake and Callaghan (36)	Porcine cervical	8	1,500	10,000	8
Marshall and McGill (70)	Porcine cervical	10	1,500	6,000	4

human spine during normal movement (98,141–143,147). As with all living tissue, the vertebrae and its supporting structures remodel when subjected to applied stress (24). Consistent with Wolff's and Davis's Laws, deformation of cellular tissues are met by a corresponding increase in the stiffness of the matrix, which in turn helps to resist future deformation (102,103). The vertebrae and intervertebral discs are no exception because they have been shown to adaptively strengthen when exposed to progressive exercise (2,24,66,92). Cadaveric tissue does not have the capacity to remodel.

Another important point to consider when interpreting results of in vitro studies involving cyclic spinal loading is that natural fluid flow is compromised. Van der Veen et al. (132) found that although porcine lumbar motion segments showed outflow of fluid during loading, inflow failed to occur during unloading, thereby decreasing disc height and interfering with normal disc biomechanics.

In vitro comparisons are further complicated by the use of animal models. Although animal models do have structural similarities to the human spine (146,29), especially the porcine cervical spine in comparison with the human lumbar spine, numerous anatomical and physiological variations nevertheless exist (130). Of particular relevance to flexion studies is the fact that the absolute ranges of motion are smaller in porcine subjects compared with humans (10). These variations are

most prominent in flexion and extension, which may mitigate the ability to draw applicable conclusions to human dynamic spinal exercise.

Furthermore, the studies in question attempted to mimic loading patterns of occupational workers by subjecting spinal segments to thousands of continuous bending cycles, which is far beyond what is normally performed in the course of a dynamic exercise program. Typical core strengthening routines use a limited number of dynamic repetitions, and on completion of a set, trainees then rest for a given period before performing another set. Thus, total bending cycles per session ultimately amount to a fraction of those used in the cited research protocols, and these cycles are performed intermittently rather than continuously. Rodacki et al. (97) found that despite the moderate values of compression associated with the traditional crunch, the transient nature of the load (i.e., the short peak period of compressive spinal force) did not induce fluid loss. In fact, abdominal flexion exercise was actually found to be superior to the Fowler's position (a semi-recumbent position used in therapy to alleviate pressure on the spine) with respect to spinal unloading, presumably mediated by a greater fluid influx rate than when sustaining a static recumbent posture (97).

It should also be noted that after an exercise bout, spinal tissues are allowed to recuperate until the next training session, thereby alleviating disc stress and affording the structures time to remodel. Exercise-induced disc

damage results when fatigue failure outpaces the rate of adaptive remodeling, which depends on the intensity of load, the abruptness of its increase, and the age and health of the trainee (2). Provided that dynamic spinal exercise is performed in a manner that does not exceed individual disc-loading capacity, the evidence would seem to suggest a positive adaptation of the supporting tissues. In support of this contention, Videman et al. (136) found that moderate physical loading resulted in the least disc pathology, with the greatest degeneration seen at extreme levels of activity and inactivity.

In addition, the role of genetics needs to be taken into consideration. Despite the commonly held belief that spinal degeneration is most often caused by the wear and tear from mechanical loading, this seems to play only a minor role in the process (17). Instead, it has been shown that approximately 74% of the variance is explained by hereditary factors (15). Battie et al. (17) identified specific gene forms associated with disc degeneration that hasten degenerative vertebral changes in the absence of repetitive trauma. Hereditary factors, such as size and shape of the spinal structures, and biochemical constituents that build or break down the disc can highly influence disc pathology, as can gene-environment interactions (17).

In a case-control study involving 45 monozygotic male twin pairs, Battie et al. (16) found that subjects who spent more than 5 times more hours driving and handled more than 1.7 times more

occupational lifting showed no increases in disc degeneration compared with their twin siblings and, although values did not reach statistical significance, actually displayed fewer lower lumbar disc herniations. In addition, Varlotta et al. (133) found that the relative risk of lumbar disc herniation before the age of 21 years is approximately 5 times greater in subjects who have a positive family history. Furthermore, physically active individuals seem to experience less back pain than sedentary individuals (44,78).

Moreover, the studies in question do not necessarily replicate spinal motion during dynamic lumbar flexion exercise. For example, the traditional crunch exercise involves flexing the trunk to approximately 30° of spinal flexion so that only the head and shoulders are lifted from the floor, making the thoracic spine the region of greatest flexion motion (105,117). Further, Adams and Hutton (6) showed that taking a flexed lumbar spine from an end range of flexion at 13° to 11° of flexion, a 2° differential, resulted in a 50% reduction in resistance to bending moment and therefore a 50% reduction in bending stress to the posterior annulus and intervertebral ligaments. Thus, both the location and degree of flexion will have a significant impact on spinal kinetics.

Finally, although abdominal exercises create compressive forces by way of muscular contraction, they also increase intra-abdominal pressure (IAP) (32). Three-dimensional biomechanical models predict reductions in compressive forces of approximately 18% when IAP is factored into spinal flexion efforts (118). Hence, IAP produced during spinal flexion exercise may serve to moderate compressive forces, helping to unload the spine and facilitate fluid absorption in the discs (97). Because *in vitro* research models to date have not incorporated IAP, conclusions drawn may be limited with respect to the safety of spinal flexion exercises. However, it should be noted that the unloading effects of IAP may be diminished with high levels

of abdominal muscle coactivation (12). Additional research is needed to shed further light on this topic with particular attention focused on evaluating the effects of IAP on compressive forces in subjects performing spinal flexion exercise including the crunch.

It should also be noted that some epidemiological studies show an increased risk of spinal injuries in athletes involved in sporting activities that require repeated spinal flexion. Injuries to the spinal column, including disc degeneration and herniations, have been found to occur with greater frequency in gymnasts, rowers, and football players (120,122,135,144). Furthermore, elite athletes experience such injuries more frequently than nonelite athletes (88,120). However, a cause-effect relationship between spinal flexion and injury in these athletes has not been established, and the ballistic nature of such sporting activities has little applicability to controlled dynamic abdominal exercises.

BENEFITS OF SPINAL FLEXION EXERCISES

If dynamic flexion exercises in fact do not pose a significant injury risk in the absence of spinal pathology, then the natural question is whether performing these movements confers benefits over and above static-based exercises. The following potential benefits can be identified.

First, spinal motion has been shown to facilitate nutrient delivery to the intervertebral discs (50,51). The mechanism of action is theorized to be related to a pumping action that augments transport and diffusion of molecules into discs. Motion causes more fluid to flow out of the disc, which is reversed when the spine is unloaded (5). Fluid flow is better at transporting large molecules, whereas diffusion is better at transporting smaller molecules (128). This has a particular significance for spinal tissue given that age-related decreases in disc nutritional status is considered a primary cause of disc degeneration, leading to an accrual of cellular waste products, degradation of matrix molecules, and a fall in pH levels that further

compromise cell function and possibly initiate apoptosis (27,52,71,130).

Postures involving flexion of the spine are superior to neutral and extended postures in terms of promoting increased fluid exchange in the disc, especially the nucleus pulposus (5). One deficiency of neutral posture is that it favors diffusion in the anterior portion of the disc over the posterior portion. Flexed postures reverse this imbalance by stretching the posterior annulus, thereby decreasing the distance for nutrients to travel. The posterior region of the disc contains a region that is deficient of nutrient supplement from all sources (69), and flexion reduces the thickness of the posterior portion of the disc by 37%, which ensures sufficient supply of glucose to the entire posterior region of the disc (5). Flexion increases diffusion of small solutes and fluid flow of large solutes. This is important considering that disc degeneration has been linked to inadequate metabolite transport (51,83) and that populations adopting flexed postures show less incidence of disc disease (40). The crunch exercise produces tensile stresses on the posterior annulus—in flexion, the posterior annulus has been shown to extend up to 60% of its original height (90)—and tensile stress has shown to exert a protective effect on disc cells by decreasing the expression of catabolic mediators during inflammation (107). By enhancing nutrient uptake and limiting inflammatory-based catabolism, regimented flexion exercise may actually confer a positive effect on the long-term spinal health and promote disc healing in the periphery (9). In fact, research suggests that spinal flexion and extension exercises can be valuable in reducing LBP (38,43,96). Although pain or lack of pain is not necessarily an indicator of spinal health, it nevertheless is interesting to speculate that spinal flexion movements may actually confer therapeutic benefits provided exercise does not exceed the adaptive capacity of the tissue.

In addition, spinal flexion exercises may help to improve functional spinal

flexibility and thereby reduce the onset of LBP. Multiple studies have found that a lack of sagittal plane spinal flexibility is associated with an increased incidence of LBP (28,37,73,89). Resistance exercise has been shown to serve as an active form of flexibility training, helping to improve joint mobility within a functional range of motion (14,80,106), and spinal flexion exercises have been shown to increase sagittal plane spinal mobility (38). Improved flexibility associated with resistance training has been attributed to increased connective tissue strength, increased muscular strength, and improved motor learning, and/or neuromuscular coordination (80). At the same time, dynamic strengthening of the supporting musculature and ligamentous tissue may attenuate spinal hypermobility in those afflicted, which has also been implicated as a cause of LBP (119). Hence, a case can be made that a well-designed resistance training program that includes dynamic spinal flexion may bestow a preventative effect against LBP. However, it should be noted that some studies have failed to reveal significant differences in the sagittal plane spinal flexibility between pain free subjects and those with LBP (94), and 1 study indicated that lumbar spinal flexibility is associated with disc degeneration (48). Moreover, we cannot necessarily determine a cause-effect relationship between poor spinal flexibility and an increased risk of injury. Further research is warranted to draw pertinent conclusions on the topic.

Finally, flexion-based spinal movements help to optimize hypertrophy of the rectus abdominis muscle. The crunch exercise and its variations have been shown to target the rectus abdominis to a much greater extent than the other core muscles. McGill (74) found that a variant of the crunch activated 50% of maximal voluntary contraction (MVC) of the rectus abdominis but only 20%, 10%, 10%, and 10% of MVC of the external obliques, internal obliques, transverse abdominis, and psoas major, respectively. Given that a direct association has been noted between muscle cross-sectional area and muscle strength

(42,72), muscle hypertrophy has specific relevance to athletes who require extensive core strength. Moreover, muscle hypertrophy of the rectus abdominis is also integral to aesthetic appearance of the abdominal musculature and is therefore highly desired by bodybuilders and other fitness enthusiasts.

The hypertrophic superiority of dynamic movement can be partly attributed to the eccentric component, which has been shown to have the greatest effect on promoting muscle development (41,49,53,100). Eccentric exercise has been linked to a preferential recruitment of fast twitch muscle fibers (85,112,121) and perhaps recruitment of previously inactive motor units (77,84). Given that fast twitch fibers have the greatest growth potential, their recruitment would necessarily contribute to greater increases in muscle cross-sectional area.

Eccentric exercise is also associated with greater muscle damage, which has been shown to mediate a hypertrophic response (77,108). Muscle damage induced by eccentric exercise upregulates MyoD messenger RNA expression (57) and has been implicated in the release of various growth factors that regulate satellite cell proliferation and differentiation (126,137).

In addition, dynamic muscle actions have been shown to induce significantly greater metabolic stress than static contractions (25). Specifically, the buildup of metabolites, such as lactate, hydrogen ion, and inorganic phosphate, has been shown to mediate a hypertrophic response (101,109,116), and some researchers have speculated that metabolic stress may be more important than high force development in optimizing muscle development (113). The stress-induced mechanisms theorized to increase muscle hypertrophy include alterations in hormonal milieu, cell swelling, free radical production, and increased activity of growth-oriented transcription factors (108). Russ (104) displayed that phosphorylation of Akt, a protein kinase associated with mTOR pathway signaling and thus regulation

of protein synthesis, is significantly greater in eccentric contractions compared with isometric contractions. This may be because of heightened metabolic stress, greater muscle damage, or a combination of both.

PRACTICAL APPLICATIONS

Taking all factors into account, it would seem that dynamic spinal flexion exercises provide a favorable risk to reward ratio provided that trainees have no existing spinal injuries or associated contraindications, such as disc herniation, disc prolapse, and/or flexion intolerance. However, several caveats need to be taken into consideration to maximize spinal health.

First and foremost, because hereditary factors have a tremendous impact on the disc degeneration, it is difficult to know the precise amount of volume, intensity, and frequency sufficient to stimulate soft tissue strengthening adaptations without exceeding the recovery ability of the spine. It has been theorized that a “safe window” of tissue mechanical loading exists that facilitates healthy maintenance of spinal discs (119). There is evidence supporting this theory because it pertains to spinal compression (145); however, further research is needed to determine whether this applies to other types of spinal loading including flexion.

An epidemiological study by Mundt et al. (82) found that participation in sports such as baseball, softball, golf, swimming, diving, jogging, aerobics, racquet sports, and weight lifting are not associated with increased risk of lumbar disc herniation, and they even may offer a protective effect against herniation. Kelsey et al. (58) reported similar findings with respect to disc prolapse. Many of these sports involve a high frequency of spinal motion including flexion, which casts doubt on the theory that humans have a limited number of flexion cycles. Unfortunately, there is no way to determine when an individual's training volume and/or intensity falls outside this range and thus predisposes the spine to localized overload injury.

Given that the spine and core musculature are loaded during nonmachine-based exercise performance, such as during squats, deadlifts, chin-ups, and push-ups, most training can be considered “core training.” Therefore, it is best to err on the side of caution and limit the amount of lumbar flexion exercise to ensure that the tissue remains in “eustress” and does not become “distressed.” Based on the current data, the authors recommend that a sound core strengthening routine should not exceed approximately 60 repetitions of lumbar flexion cycles per training session. Untrained individuals should begin with a substantially lower volume. A conservative estimate would be to start with 2 sets of 15 repetitions and gradually build up tolerance over time.

In addition, it is important to allow for sufficient rest between dynamic spinal flexion sessions. The time course of postexercise muscle protein synthesis lasts approximately 48 hours (67). Training a muscle group before protein synthesis has completed its course can impair muscle development (47) and potentially lead to localized overtraining. Thus, the notion that it is optimal to perform dynamic abdominal exercises on a daily basis is misguided. Because the intervertebral discs are poorly vascularized with low levels of metabolite transport, their rate of remodeling lags behind that of other skeletal tissues (69,115), which may necessitate even greater time for recuperation. Taking all factors into account, a minimum of 48 hours should be afforded between dynamic spinal flexion exercise sessions, and it may be prudent to allow 72 hours or more depending on individual response.

Although some core training programs include ultrahigh repetition sets of crunches, for example, multiple sets of a hundred repetitions or more, this type of protocol has little functional applicability. After all, when does an individual need to continuously flex the spine in everyday life? It is therefore recommended that flexion-based spinal exercises be reserved for improving strength and/or hypertrophy of

the abdominal musculature as opposed to heightening muscular endurance. A repetition range of approximately 6–15 repetitions is advised for achieving this goal (108). External resistance should be used when necessary to elicit an overload response within this target repetition range. Those seeking improvements in local muscular endurance would be best served by performing static, neutral posture exercises that are held for extended periods. Specific guidelines will vary dramatically according to the individual’s needs and abilities, but a general recommendation for untrained individuals would be to perform 3–4 sets of 10- to 15-second holds in multiple planes. Advanced exercisers seeking increases in static endurance might perform 3–4 sets of 60 seconds or more in multiple planes, whereas advanced exercisers seeking increases in static power could stick to the 10- to 15-second holds but perform more challenging variations or increase external resistance to promote further adaptation. Athletes who engage in sports where spinal flexion exercise or other inherently dangerous motions for the discs, such as spinal rotation, is prominent and volumes of flexion cycles and training frequencies above our recommendations are exceeded should consider the possibility of excluding spinal flexion exercise from their routines.

Exercise tempo is another important consideration. Several studies have shown that repetitions performed at a speed of 1 second elicit greater muscle activation than those performed more slowly (134), and faster repetitions may selectively recruit the rectus abdominis (87). Given the principle of specificity, rapid speeds of movement would also tend to have greater transfer to athletic activities that require dynamic core power, such as wrestling (54), throwing a baseball (56), tennis (33), gymnastics (91), soccer (125), swimming (68), and track and field (46). However, an increased repetition speed could subject the spinal tissues to excessive forces that may lead to injury (6,86). For nonathletic

populations, the risks of faster repetitions would appear to outweigh the potential rewards and thus a slightly slower tempo of approximately 2 seconds may be more appropriate with respect to maintaining spinal health. As for athletic populations, more research is needed to show whether explosive dynamic core exercises lead to positive adaptations that strengthen tissues and prevent injury or whether they subject the athlete to greater risk of injury by adding more stress to the tissues.

It also is important to consider the effects of diurnal variation on spinal kinetics. During sleep, loading on the discs is reduced, allowing them to absorb more fluid and increase in volume (129). Fluid is then expelled throughout the day as normal daily spinal loading ensues. In the early morning, intradiscal pressure is 240% higher than before going to bed (140), and bending stresses are increased at the discs by 300% and at the ligaments of the neural arch by 80% because of hydration and absence of creep (4). As the day goes on, discs bulge more, become stiffer in compression, become more elastic and flexible in bending, affinity for water increases, and the risk of disc prolapse decreases (1). After just 30 minutes of waking, discs lose 54% of the loss of daily disc height and water content and 90% within the first hour (95). For this reason, spinal flexion exercises should be avoided within at least 1 hour of rising. To be conservative, athletes may want to allow a minimum of 2 hours or more before engaging in exercises that involve spinal flexion.

There is some evidence that spinal flexion exercises should also be avoided after prolonged sitting. It has been shown that discs actually gain height after sitting (11,61) and decrease lumbar range of motion (31), which reduces slack in the flexion-resisting structures including ligaments and the posterior annulus while increasing the risk of injury to those structures (4,18). However, as noted by Beach et al. (18), individual differences in sitting posture lead to large variations in tissue

response. Some individuals actually gain lumbar range of motion from sitting, which can also increase the risk of injury because of viscoelastic creep (76), stress relaxation (3), or fluid loss (5), which increases joint laxity (4). Considering that approximately 50% of stiffness is regained within 2 minutes of rising after 20 minutes of full flexion (76), it seems prudent to allow at least several minutes to elapse, perhaps 5 or more, before engaging in spinal flexion exercises after a period of prolonged sitting and to walk around to facilitate dehydration of the disc.

CONCLUSION

Based on current research, it is premature to conclude that the human spine has a limited number of bending cycles. The claim that dynamic flexion exercises are injurious to the spine in otherwise healthy individuals remains highly speculative and is based largely on the extrapolation of in vitro animal data that is of questionable relevance to in vivo human spinal biomechanics. Although it appears that a large number of continuous bending cycles may ultimately have a detrimental effect on spinal tissues, no evidence exists that a low-volume strength-based exercise routine that includes dynamic spinal flexion movements will hasten the onset of disc degeneration, and a case can be made that such exercises may in fact produce a beneficial effect in terms of disc health. Contraindications for spinal flexion movements would only seem applicable with respect to those with existing spinal pathology, such as disc herniation/flop or flexion intolerance.

To date, the authors are not aware of any study that has investigated the effects of spinal flexion exercise on human spines in vivo. Further research is needed to evaluate both the acute and chronic effects of dynamic spinal flexion exercises in human subjects in vivo so that more definitive conclusions can be drawn on the topic. This research should include magnetic resonance imaging of intervertebral discs to assess disc health preceding and following human spinal flexion protocols of varying loads, repetitions,

tempos, and ranges of motion. It is hoped that this article will serve to spark new research in this area.

With respect to program design, basic core strength and endurance will be realized through performance of most nonmachine-based exercises such as squats, rows, deadlifts, and push-ups. That said, targeted core exercises may serve to enhance sports performance, functional capacity, and physique aesthetics. Consistent with the principle of specificity, core program design should take into account the individual goals and abilities of the exerciser with respect to their need for muscular hypertrophy, power, strength, and/or endurance, and the types of joint actions involved in their sport. A variety of abdominal exercises are necessary to sufficiently work the abdominal musculature, and these exercises will differ based on training objectives (13). Variety in spinal loading is associated with lower risk of spinal pathology (136). A balanced multiplanar approach to core training that incorporates a combination of isometric and dynamic exercises is warranted to prevent any particular spinal segment from accentuated stress and to ensure proper spine-stabilizing biomechanics.



Bret Contreras is currently pursuing his PhD at AUT University.



Brad Schoenfeld is the owner/director of Global Fitness Services.

REFERENCES

1. Adams A, Dolan P, Hutton W, and Porter R. Diurnal changes in spinal mechanics and their clinical significance. *J Bone Joint Surg* 72B: 266–270, 1990.

2. Adams MA and Dolan P. Time-dependent changes in the lumbar spine's resistance to bending. *Clin Biomech (Bristol, Avon)* 11: 194–200, 1996.
3. Adams MA and Dolan P. Could sudden increases in physical activity cause degeneration of intervertebral discs? *Lancet* 350: 734–735, 1997.
4. Adams MA, Dolan P, and Hutton WC. Diurnal variations in the stresses on the lumbar spine. *Spine (Phila Pa 1976)* 12: 130–137, 1987.
5. Adams MA and Hutton WC. The effect of posture on the fluid content of lumbar intervertebral discs. *Spine (Phila Pa 1976)* 8: 665–671, 1983.
6. Adams MA and Hutton WC. The effect of posture on diffusion into lumbar intervertebral discs. *J Anat* 147: 121–134, 1986.
7. Adams MA, May S, Freeman BJ, Morrison HP, and Dolan P. Mechanical initiation of intervertebral disc degeneration. *Spine (Phila Pa 1976)* 25: 1625–1636, 2000.
8. Adams MA, McNally DS, and Dolan P. Stress distribution inside intervertebral discs: The effects of age and degeneration. *J Bone Joint Surg Br* 78: 965–972, 1996.
9. Adams MA, Stefanakis M, and Dolan P. Healing of a painful intervertebral disc should not be confused with reversing disc degeneration: Implications for physical therapies for discogenic back pain. *Clin Biomech (Bristol, Avon)* 25: 961–971, 2010.
10. Alini M, Eisenstein SM, Ito K, Little C, Kettler AA, Masuda K, Melrose J, Ralphs J, Stokes I, and Wilke HJ. Are animal models useful for studying human disc disorders/ degeneration? *Eur Spine J* 17: 2–19, 2008.
11. Althoff I, Brinckmann P, Frobin W, Sandover J, and Burton K. An improved method of stature measurement for quantitative determination of spinal loading. *Spine (Phila Pa 1976)* 17: 682–693, 1992.
12. Arjmand N and Shirazi-Adl A. Role of intra-abdominal pressure in the unloading and stabilization of the human spine during static lifting tasks. *Eur Spine J* 15: 1265–1275, 2006.
13. Axler CT and McGill SM. Low back loads over a variety of abdominal exercises: Searching for the safest abdominal challenge. *Med Sci Sports Exerc* 29: 804–811, 1997.
14. Barbosa AR, Santarém JM, Filho WJ, and Marucci Mde F. Effects of resistance training on the sit-and-reach test in elderly

- women. *J Strength Cond Res* 16: 14–18, 2002.
15. Battié MC and Videman T. Lumbar disc degeneration: Epidemiology and genetics. *J Bone Joint Surg Am* 88(Suppl 2): 3–9, 2006.
 16. Battié MC, Videman T, Gibbons LE, Manninen H, Gill K, Pope M, and Kaprio J. Occupational driving and lumbar disc degeneration: A case-control study. *Lancet* 360: 1369–1374, 2002.
 17. Battié MC, Videman T, Kaprio J, Gibbons LE, Gill K, Manninen H, Saarela J, and Peltonen L. The Twin spine study: Contributions to a changing view of disc degeneration. *Spine J* 9: 47–59, 2009.
 18. Beach TA, Parkinson RJ, Stothart JP, and Callaghan JP. Effects of prolonged sitting on the passive flexion stiffness of the in vivo lumbar spine. *Spine J* 5: 145–154, 2005.
 19. Boden SD, Davis DO, Dina TS, Patronas NJ, and Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am* 72: 403–408, 1990.
 20. Boos N, Rieder R, Schade V, Spratt KF, Semmer N, and Aebi M. 1995 Volvo Award in clinical sciences. The diagnostic accuracy of magnetic resonance imaging, work perception, and psychosocial factors in identifying symptomatic disc herniations. *Spine (Phila Pa 1976)* 20: 2613–2625, 1995.
 21. Boos N, Weissbach S, Rohrbach H, Weiler C, Spratt KF, and Nerlich AG. Classification of age-related changes in lumbar intervertebral discs. *Spine (Phila Pa 1976)* 27: 2631–2644, 2002.
 22. Boxberger J, Orlansky A, Sen S, and Elliot D. Reduced nucleus pulposus glycosaminoglycan content alters intervertebral disc dynamic viscoelastic mechanics. *J Biomech* 42: 1941–1946, 2009.
 23. Boyle M. *Advances in Functional Training: Training Techniques for Coaches, Personal Trainers and Athletes*. Aptos, CA: On Target Publications, 2010. pp. 88.
 24. Brickley-Parsons D and Glimcher MJ. Is the chemistry of collagen in intervertebral discs an expression of Wolff's Law? A study of the human lumbar spine. *Spine (Phila Pa 1976)* 9: 148–163, 1984.
 25. Bridges CR, Clark BJ, Hammond RL, and Stephenson LW. Skeletal muscle bioenergetics during frequency dependent fatigue. *Am J Physiol* 1260: C643–C651, 1991.
 26. Broberg KB. On the mechanical behaviour of intervertebral discs. *Spine (Phila Pa 1976)* 8:151–165, 1983.
 27. Buckwalter JA. Aging and degeneration of the human intervertebral disc. *Spine (Phila Pa 1976)* 20: 1307–1314, 1995.
 28. Burton AK, Tillotson KM, and Troup DG. Variations in lumbar sagittal mobility with low back trouble. *Spine (Phila Pa 1976)* 14: 584–590, 1989.
 29. Busscher I, Ploegmakers JJ, Verkerke GJ, and Veldhuizen AG. Comparative anatomical dimensions of the complete human and porcine spine. *Eur Spine J* 19: 1104–1114, 2010.
 30. Callaghan JP and McGill SM. Intervertebral disc herniation: Studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. *Clin Biomech (Bristol, Avon)* 16:28–37, 2001.
 31. Callaghan JP and McGill SM. Low back joint loading and kinematics during standing and unsupported sitting. *Ergonomics* 44: 280–294, 2001.
 32. Cholewicki J, Ivancic P, and Radebold A. Can increased intra-abdominal pressure in humans be decoupled from trunk muscle co-contraction during steady state isometric exertions? *Eur J Appl Physiol* 87: 127–133, 2002.
 33. Chow JW, Shim JH, and Lim YT. Lower trunk muscle activity during the tennis serve. *J Sci Med Sport* 6: 512–518, 2003.
 34. DeWald RL. *Spinal Deformities: The Comprehensive Text*. New York, NY: Thieme, 2003. pp. 213.
 35. Drake JD, Aultman CD, McGill SM, and Callaghan JP. The influence of static axial torque in combined loading on intervertebral joint failure mechanics using a porcine model. *Clin Biomech* 20: 1038–1045, 2005.
 36. Drake JD and Callaghan JP. Intervertebral neural foramina deformation due to two types of repetitive combined loading. *Clin Biomech* 24: 1–6, 2009.
 37. Dvorak J, Panjabi MM, Novotny JE, Chang DG, and Grob D. Clinical validation of functional flexion-extension roentgenograms of the lumbar spine. *Spine (Phila Pa 1976)* 16: 943–950, 1991.
 38. Elnagger IM, Nordin M, Sheikhzadeh A, Parnianpour M, and Kahanovitz N. Effects of spinal flexion and extension exercises on low-back pain and spinal mobility in chronic mechanical low-back pain patients. *Spine (Phila Pa 1976)* 16: 967–972, 1991.
 39. Eyre DR and Muir H. Quantitative analysis of types I and II collagens in human intervertebral discs at various ages. *Biochim Biophys Acta* 492: 29–42, 1977.
 40. Fahrni WH and Trueman GE. Comparative radiological study of the spines of a primitive population with North Americans and Northern Europeans. *J Bone Joint Surg* 47B: 552–555, 1965.
 41. Farthing JP and Chilibeck PD. The effects of eccentric and concentric training at different velocities on muscle hypertrophy. *Eur J Appl Physiol* 89: 578–586, 2003.
 42. Fitts RH, McDonald KS, and Schluter JM. The determinants of skeletal muscle force and power: Their adaptability with changes in activity pattern. *J Biomech* 24: 111–122, 1991.
 43. França FR, Burke TN, Hanada ES, and Marques AP. Segmental stabilization and muscular strengthening in chronic low back pain: A comparative study. *Clinics (Sao Paulo)* 65: 1013–1017, 2010.
 44. Frymoyer JW. Epidemiology. In: *New Perspectives in Low Back Pain*. Frymoyer JW, Gordon SL, eds. Park Ridge, IL: American Academy of Orthopaedic Surgeons, 1989. pp. 19–34.
 45. Gadenken SB. Off-season strength, power, and plyometric training for Kansas State volleyball. *Strength Cond J* 21(6): 49–55, 1999.
 46. Gainor BJ, Hagen RJ, and Allen WC. Biomechanics of the spine in the pole vaulter as related to spondylolysis. *Am J Sports Med* 11: 53–57, 1983.
 47. Haddad F and Adams GR. Selected contribution: Acute cellular and molecular responses to resistance exercise. *J Appl Physiol* 93: 394–403, 2002.
 48. Haughton VM, Schmidt TA, Keele K, An HS, and Lim TH. Flexibility of lumbar spinal motion segments correlated to type of tears in the annulus fibrosus. *J Neurosurg* 92: 81–86, 2000.
 49. Higbie EJ, Cureton KJ, Warren GL III, and Prior BM. Effects of concentric and eccentric training on muscle strength, cross-sectional area, and neural activation. *J App Physiol* 81: 2173–2181, 1996.
 50. Holm S and Nachemson A. Nutritional changes in the canine intervertebral disc after spinal fusion. *Clin Orthop Relat Res* 169: 243–258, 1982.

51. Holm S and Nachemson A. Variations in the nutrition of the canine intervertebral disc induced by motion. *Spine (Phila Pa 1976)* 8: 866–874, 1983.
52. Horner HA and Urban JP. 2001 Volvo Award Winner in Basic Science Studies: Effect of nutrient supply on the viability of cells from the nucleus pulposus of the intervertebral disc. *Spine (Phila Pa 1976)* 26: 2543–2549, 2001.
53. Hortobágyi T, Barrier J, Beard D, Braspenincx J, and Koens J. Greater initial adaptations to submaximal muscle lengthening than maximal shortening. *J Appl Physiol* 81: 1677–1682, 1996.
54. Iwai K, Okada T, Nakazato K, Fujimoto H, Yamamoto Y, and Nakajima H. Sport-specific characteristics of trunk muscles in collegiate wrestlers and judokas. *J Strength Cond Res* 22: 350–358, 2008.
55. Jackson AR and Gu WY. Transport properties of cartilaginous tissues. *Curr Rheumatol Rev* 5: 40–50, 2009.
56. Jacobs P. The overhand baseball pitch. A kinesiological analysis and related strength-conditioning programming. *Strength Cond J* 9(1): 5–13, 1987.
57. Jency NE, Sims JK, Dieli-Conwright CM, Sattler FR, Rice JC, and Schroeder ET. Exercise does not influence myostatin and follistatin messenger RNA expression in young women. *J Strength Cond Res* 24: 522–530, 2010.
58. Kelsey JL, Githens PB, O'Conner T, Weil U, Calogero JA, Holford TR, White AA III, Walter SD, Ostfeld AM, and Southwick WO. Acute prolapsed lumbar intervertebral disc: An epidemiologic study with special reference to driving automobiles and cigarette smoking. *Spine (Phila Pa 1976)* 6: 608–613, 1984.
59. Larson J, Levicoff E, Gilbertson L, and Kang J. Biologic modification of animal models of intervertebral disc degeneration. *J Bone Joint Surg* 88: 83–87, 2006.
60. Lawrence JS. The epidemiology of rheumatic diseases. In: *Textbook of the Rheumatic Disease*. WSC Copeman. Edinburgh, United Kingdom: Churchill Livingstone, 1969. pp. 163–181.
61. Leivseth G and Drerup B. Spinal shrinkage during work in a sitting posture compared to work in a standing posture. *Clin Biomech* 12: 409–418, 1997.
62. Li SZ, Hu YG, and Chen PX. Study on the collagen on the different regions of disc and different sigmental disc. *Zhonghua Wai Ke Za Zhi* 32: 670–672, 1994.
63. Lotz JC. Animal models of intervertebral disc degeneration: Lessons learned. *Spine (Phila Pa 1976)* 29: 2742–2750, 2004.
64. Lotz JC, Hsieh AH, Walsh AL, Palmer EI, and Chin JR. Mechanobiology of the intervertebral disc. *Biochem Soc Trans* 30(Pt 6): 853–858, 2002.
65. Luoma K, Riihimäki H, Luukkonen R, Raininko R, Viikari-Juntura E, and Lamminen A. Low back pain in relation to lumbar disc degeneration. *Spine (Phila Pa 1976)* 25: 487–492, 2000.
66. Luoma K, Riihimäki H, Raininko R, Luukkonen R, Lamminen A, and Viikari-Juntura E. Lumbar disc degeneration in relation to occupation. *Scand J Work Environ Health* 24: 358–366, 1998.
67. MacDougall JD, Gibala MJ, Tarnopolsky MA, MacDonald JR, Interisano SA, and Yarasheski KE. The time course for elevated muscle protein synthesis following heavy resistance exercise. *Can J Appl Physiol* 20: 480–486, 1995.
68. Magnusson SP, Constantini NW, McHugh MP, and Gleim GW. Strength profiles and performance in Masters' level swimmers. *Am J Sports Med* 23: 626–631, 1995.
69. Maroudas A, Stockwell RA, Nachemson A, and Urban J. Factors involved in the nutrition of the human lumbar intervertebral disc: Cellularity and diffusion of glucose in vitro. *J Anat* 120: 113–130, 1975.
70. Marshall LW and McGill SM. The role of axial torque in disc herniation. *Clin Biomech (Bristol, Avon)* 25: 6–9, 2010.
71. Martin MD, Boxell CM, and Malone DG. Pathophysiology of lumbar disc degeneration: A review of the literature. *Neurosurg Focus* 13: E1, 2002.
72. Maughan RJ, Watson JS, and Weir J. Strength and cross-sectional area of human skeletal muscle. *J Physiol* 338: 37–49, 1983.
73. Mayer T, Tencer A, Kristiferson S, and Mooney V. Use of noninvasive techniques for quantification of spinal range-of-motion in normal subjects and chronic low back dysfunction patients. *Spine (Phila Pa 1976)* 9: 588–595, 1984.
74. McGill SM. *Low Back Disorders*. Champagne, IL: Human Kinetics, 2002. pp. 105.
75. McGill SM. Core training: Evidence translating to better performance and injury prevention. *Strength Cond J* 32(3): 33–45, 2010.
76. McGill SM and Brown S. Creep response of the lumbar spine to prolonged full flexion. *Clin Biomech (Bristol, Avon)* 7: 43–46, 1992.
77. McHugh MP, Connolly DA, Eston RG, and Gleim GW. Electromyographic analysis of exercise resulting in symptoms of muscle damage. *J Sports Sci* 18: 163–172, 2000.
78. McKenzie R and Donelson R. Mechanical diagnosis and therapy for low back pain. Toward a better understanding. In: *The Lumbar Spine* (2nd ed). Weisel SW, Weinstein JN, Herkowitz H, eds. Philadelphia, PA: W.B. Saunders, 1996. pp. 998–1011.
79. Miller J, Schmatz C, and Schultz A. Lumbar disc degeneration: Correlation with age, sex, and spine level in 600 autopsy specimens. *Spine (Phila Pa 1976)* 13: 173–178, 1988.
80. Mookerjee S and Ratamess NA. Comparison of strength differences and joint action durations between full and partial range-of-motion bench press exercise. *J Strength Cond Res* 13: 76–81, 1999.
81. Moore RJ. The vertebral endplate: Disc degeneration, disc regeneration. *Eur Spine J* 15: S333–S337, 2006.
82. Mundt DJ, Kelsey JL, Golden AL, Panjabi MM, Pastides H, Berg AT, Sklar J, and Hosea T. An epidemiologic study of sports and weight lifting as possible risk factors for herniated lumbar and cervical discs. *Am J Sports Med* 21: 854–860, 1993.
83. Nachemson A, Lewin T, Maroudas A, and Freeman MA. In vitro diffusion of dye through the end-plates and the annulus fibrosus of human intervertebral discs. *Acta orthop Scand* 41: 589–607, 1970.
84. Nardone A, Romanò C, and Schieppati M. Selective recruitment of high-threshold human motor units during voluntary isotonic lengthening of active muscles. *J Physiol* 409: 451–471, 1989.
85. Nardone A and Schieppati M. Shift of activity from slow to fast muscle during voluntary lengthening contractions of the triceps surae muscles in humans. *J Physiol* 395: 363–381, 1988.
86. Norris CM. Abdominal muscle training in sport. *Br J Sports Med* 27: 19–27, 1993.
87. Norris CM. Functional load abdominal training: Part 1. *Phys Ther Sport* 2: 29–39, 2001.
88. Ong A, Anderson J, and Roche J. A pilot study of the prevalence of lumbar disc degeneration in elite athletes with lower back pain at the Sydney 2000 Olympic Games. *Br J Sports Med* 37: 263–266, 2003.

89. Pearcy MJ. Stereo radiography of lumbar spine motion. *Acta Orthop Scand* 212: 1–45, 1985.
90. Pearcy MJ and Tibrewal SB. Lumbar intervertebral disc and ligament deformations measured in vivo. *Clin Orthop Relat Res* 191: 281–286, 1984.
91. Peltonen JE, Taimela S, Erkintalo M, Salminen JJ, Oksanen A, and Kujala UM. Back extensor and psoas muscle cross-sectional area, prior physical training, and trunk muscle strength: A longitudinal study in adolescent girls. *Eur J Appl Physiol* 77: 66–71, 1998.
92. Porter RW, Adams MA, and Hutton WC. Physical activity and the strength of the lumbar spine. *Spine (Phila Pa 1976)* 14: 201–203, 1989.
93. Postacchini F. Management of lumbar spinal stenosis. *J Bone Joint Surg (Br)* 78: 154–164, 1996.
94. Rae P, Venner RM, and Waddell G. A simple clinical technique of measuring lumbar flexion. *J R Coll Surg Edinb* 29: 281–284, 1981.
95. Reilly T, Tyrrell A, and Troup JD. Circadian variation in human stature. *Chronobiol Int* 1: 121–126, 1984.
96. Revel M. Rehabilitation of low back pain patients. *Rev Rhum Engl Ed* 62: 35–44, 1995.
97. Rodacki NC, Rodacki LF, Ugrinowitsch C, Zielenski D, and Budal da Costa R. Spinal unloading after abdominal exercises. *Clin Biomech (Bristol, Avon)* 23: 8–14, 2008.
98. Rohlmann A, Bauer L, Zander T, Bergmann G, and Wilke HJ. Determination of trunk muscle forces for flexion and extension by using a validated finite element model of the lumbar spine and measured in vivo data. *J Biomech (Bristol, Avon)* 39: 981–989, 2006.
99. Rohlmann A, Zander T, Schmidt H, Wilke HJ, and Bergmann G. Analysis of the influence of disc degeneration on the mechanical behaviour of a lumbar motion segment using the finite element method. *J Biomech (Bristol, Avon)* 39: 2484–2490, 2006.
100. Roig M, O'Brien K, Kirk G, Murray R, McKinnon P, Shadgan B, and Reid WD. The effects of eccentric versus concentric resistance training on muscle strength and mass in healthy adults: A systematic review with meta-analysis. *Br J Sports Med* 43: 556–558, 2009.
101. Rooney KJ, Herbert RD, and Balnave RJ. Fatigue contributes to the strength training stimulus. *Med Sci Sports Exerc* 26: 1160–1164, 1994.
102. Rubin L and Schweitzer S. The use of acellular biologic tissue patches in foot and ankle surgery. *Clin Podiatr Med Surg* 22: 533–552, 2005.
103. Ruff C, Holt B, and Trinkaus E. Who's afraid of the big bad Wolff?: "Wolff's law" and bone functional adaptation. *Am J Phys Anthropol* 129: 484–498, 2006.
104. Russ DW. Active and passive tension interact to promote Akt signaling with muscle contraction. *Med Sci Sports Exerc* 40: 88–95, 2008.
105. Sands WA and McNeal JR. A kinematic comparison of four abdominal training devices and a traditional abdominal crunch. *J Strength Cond Res* 16: 135, 2002.
106. Santos E, Rhea MR, Simão R, Dias I, de Salles BF, Novaes J, Leite T, Blair JC, and Bunker DJ. Influence of moderately intense strength training on flexibility in sedentary young women. *J Strength Cond Res* 24: 3144–3149, 2010.
107. Sowa G and Agarwal S. Cyclic tensile stress exerts a protective effect on intervertebral disc cells. *Am J Phys Med Rehabil* 87: 537–544, 2008.
108. Schoenfeld BJ. The mechanisms of muscle hypertrophy and their application to resistance training. *J Strength Cond Res* 24: 2857–2875, 2010.
109. Schott J, McCully K, and Rutherford OM. The role of metabolites in strength training. II. Short versus long isometric contractions. *Eur J Appl Physiol* 71: 337–341, 1995.
110. Schuler L and Cosgrove A. *The New Rules of Lifting for Abs: A Myth-Busting Fitness Plan for Men and Women Who Want a Strong Core and a Pain-Free Back*. New York, NY: Avery, 2010. pp. 20.
111. Schuenke M, Schulte E, and Schumacher U. *Atlas of Anatomy: General Anatomy and Musculoskeletal System* New York, NY: Thieme, 2006. pp. 93.
112. Shepstone TN, Tang JE, Dallaire S, Schuenke MD, Staron RS, and Phillips SM. Short-term high- vs. low-velocity isokinetic lengthening training results in greater hypertrophy of the elbow flexors in young men. *J Appl Physiol* 98: 1768–1776, 2005.
113. Shinohara M, Kouzaki M, Yoshihisa T, and Fukunaga T. Efficacy of tourniquet ischemia for strength training with low resistance. *Euro J Appl Physiol* 77: 189–191, 1998.
114. Singh K, Masuda K, Thonar E, An H, and Cs-Szabo G. Age related changes in the extracellular matrix of nucleus pulposus and annulus fibrosus of human intervertebral disc. *Spine (Phila Pa 1976)* 34: 10–16, 2009.
115. Skrzypiec D, Tarala M, Pollintine P, Dolan P, and Adams MA. When are intervertebral discs stronger than their adjacent vertebrae? *Spine (Phila Pa 1976)* 32: 2455–2461, 2007.
116. Smith RC and Rutherford OM. The role of metabolites in strength training. I. A comparison of eccentric and concentric contractions. *Eur J Appl Physiol Occup Physiol* 71: 332–336, 1995.
117. Sternlicht E and Rugg S. Electromyographic analysis of abdominal muscle activity using portable abdominal exercise devices and a traditional crunch. *J Strength Cond Res* 17: 463–468, 2003.
118. Stokes IA, Gardner-Morse MG, and Henry SM. Intra-abdominal pressure and abdominal wall muscular function: Spinal unloading mechanism. *Clin Biomech (Bristol, Avon)* 25: 859–866, 2010.
119. Stokes IA and Iatridis JC. Mechanical conditions that accelerate intervertebral disc degeneration: Overload versus immobilization. *Spine (Phila Pa 1976)* 29: 2724–2732, 2004.
120. Swärd L, Hellström M, Jacobsson B, Nyman R, and Peterson L. Disc degeneration and associated abnormalities of the spine in elite gymnasts. A magnetic resonance imaging study. *Spine (Phila Pa 1976)* 16: 437–443, 1991.
121. Takarada Y, Takazawa H, Sato Y, Takebayashi S, Tanaka Y, and Ishii N. Effects of resistance exercise combined with moderate vascular occlusion on muscular function in humans. *J Appl Physiol* 88: 2097–2106, 2000b.
122. Tall RL and DeVault W. Spinal injury in sport: Epidemiologic considerations. *Clin Sports Med* 12: 441–448, 1993.
123. Tampier C, Drake JD, Callaghan JP, and McGill SM. Progressive disc herniation: An investigation of the mechanism using radiologic, histochemical, and microscopic dissection techniques on a porcine model. *Spine (Phila Pa 1976)* 32: 2869–2874, 2007.
124. Thomas TR and Ridder MB. Resistance exercise program effects on abdominal function and physique. *J Sports Med Phys Fitness* 29: 45–48, 1989.

125. Togari H and Asami T. A study of throw-in training in soccer. Proceedings of the Department of Physical Education, College of General Education, University of Tokyo, Tokyo, Japan (vol 6). 1972. pp. 33–38.
126. Toigo M and Boutellier U. New fundamental resistance exercise determinants of molecular and cellular muscle adaptations. *Eur J Appl Physiol* 97: 643–663, 2006.
127. Tsuji H, Hirano N, Ohshima H, Ishihara H, Terahata N, and Motoe T. Structural variation of the anterior and posterior annulus fibrosus in the development of human lumbar intervertebral disc. A risk factor for intervertebral disc rupture. *Spine (Phila Pa 1976)* 18: 204–210, 1993.
128. Urban JP, Holm S, Maroudas A, and Nachemson A. Nutrition of the intervertebral disc: Effect of fluid flow on solute transport. *Clin Orthop Relat Res* 170: 296, 1982.
129. Urban JP and McMullin JF. Swelling pressure of the lumbar intervertebral discs: Influence of age, spinal level, composition, and degeneration. *Spine (Phila Pa 1976)* 13: 179–187, 1988.
130. Urban JP and Roberts S. Degeneration of the intervertebral disc. *Arthritis Res Ther* 5: 120–130, 2003.
131. Urban JP, Smith S, and Fairbank JC. Nutrition of the intervertebral disc. *Spine (Phila Pa 1976)* 29: 2700–2709, 2004.
132. Van der Veen A, Mullender M, Smit T, Kingma I, and Van Dieen J. Flow-related mechanics of the intervertebral disc: The validity of an in vitro model. *Spine (Phila Pa 1976)* 30: E5340–E539, 2005.
133. Varlotta GP, Brown MD, Kelsey JL, and Golden AL. Familial predisposition for herniation of a lumbar disc in patients who are less than twenty-one years old. *J Bone Joint Surg Am* 73: 124–128, 1991.
134. Vera-Garcia FJ, Flores-Parodi B, Elvira JL, and Sarti MA. Influence of trunk curl-up speed on muscular recruitment. *J Strength Cond Res* 22: 684–690, 2008.
135. Videman T, Battié MC, Gibbons LE, Manninen H, Gill K, Fisher LD, and Koskenvuo M. Lifetime exercise and disk degeneration: An MRI study of monozygotic twins. *Med Sci Sports Exerc* 29: 1350–1356, 1997.
136. Videman T, Nurminen M, and Troup JD. 1990 Volvo Award in clinical sciences. Lumbar spinal pathology in cadaveric material in relation to history of back pain, occupation, and physical loading. *Spine (Phila Pa 1976)* 15: 728–740, 1990.
137. Vierck J, O'Reilly B, Hossner K, Antonio J, Byrne K, Bucci L, and Dodson M. Satellite cell regulation following myotrauma caused by resistance exercise. *Cell Biol Int* 24: 263–272, 2000.
138. Walker J III, El Abd O, Isaac Z, and Muzin S. Discography in practice: A clinical and historical view. *Curr Rev Musculoskelet Med* 1: 69–83, 2008.
139. Wiesel SW, Tsourmas N, Feffer HL, Citrin CM, and 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)* 9: 549–551, 1984.
140. Wilke HJ, Neef P, Caimi M, Hoogland T, and Claes L. New intradiscal pressure measurements in vivo during daily activities. *Spine (Phila Pa 1976)* 24: 755–762, 1999.
141. Wilke HJ, Rohlmann A, Neller S, Schultheiss M, Bergmann G, Graichen F, and Claes LE. Is it possible to simulate physiologic loading conditions by applying pure moments? A comparison of in vivo and in vitro load components in an internal fixator. *Spine (Phila Pa 1976)* 26: 636–642, 2001.
142. Wilke HJ, Wolf S, Claes LE, Arand M, and Wiesand A. Influence of varying muscle forces on intradiscal pressure: An in vitro study. *J Biomech* 29: 549–55, 1996.
143. Wilke HJ, Wolf S, Claes LE, and Wiesand A. Stability increase of the lumbar spine with different muscle groups. *Spine (Phila Pa 1976)* 20: 192–198, 1995.
144. Wilson F, Gissane C, Gormley J, and Simms C. A 12-month prospective cohort study of injury in international rowers. *Br J Sports Med* 44: 207–214, 2010.
145. Wuertz K, Godburn K, MacLean JJ, Barbir A, Donnelly JS, Roughley PJ, Alini M, and Iatridis JC. In vivo remodeling of intervertebral discs in response to short- and long-term dynamic compression. *J Orthop Res* 27: 1235–1242, 2009.
146. Yingling VR, Callaghan JP, and McGill SM. The porcine cervical spine as a reasonable model of the human lumbar spine: An anatomical, geometric, and functional comparison. *J Spinal Disorders* 12: 415–423, 1999.
147. Zander T, Rohlmann A, Calisse J, and Bergmann G. Estimation of muscle forces in the lumbar spine during upper-body inclination. *Clin Biomech (Bristol, Avon)* 16(Suppl 1): S73–S80, 2001.