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Lumbar Stabilization: An Evidence-Based Approach for the Athlete With Low Back Pain

Morey J. Kolber PT, MSPT, CSCS, Kristina Beekhuizen, PhD, CSCS
Nova Southeastern University, Ft. Lauderdale, Florida

summary

This manuscript presents an overview of spinal stabilization for the lumbar spine. Emphasis is placed on the local stabilization musculature, which has received considerable support in the literature. A progressive stabilization program targeting the local stabilizing musculature is recommended for the diverse athletic population.

Introduction

Approximately 60–80% of the adult population will experience an episode of low back pain (LBP) at some point in their lives (13, 49, 58, 65). The natural history, however, is favorable, as over 80% of individuals may recover independently of treatment within 4–6 weeks of initial complaints (49, 75). Although the natural history is favorable, the reported recurrence rate of LBP is as high as

58–90% (13, 29, 49, 72, 73). LBP is only second to the common cold for physician visits, and the costs of LBP in some societies exceed that of coronary artery disease and diabetes combined (49). From an economic standpoint, the total cost of LBP including societal factors exceeds \$40 billion per year (20, 49, 75).

LBP is not limited to the nonathletic population, as individuals involved in athletic endeavors may be affected at an equal or greater frequency than the general population (6, 15, 23, 50, 58, 64, 71). Specific sports such as weightlifting and American football are associated with a higher incidence of degenerative conditions, stress fractures, and injuries of the lumbar spine when compared to the general population (3, 6, 23, 36, 37, 44). Athletes who perform sports involving repeated or forceful loading of the spine are considerably more prone to spondylolysis (stress fractures of the pars interarticularis) and instability of the low back (22, 25, 26, 40, 41, 59, 70). Stress fractures and spinal instability have been identified as risk factors for LBP in the athletic population (22, 36, 37).

Intervention for athletes with LBP is often based upon the educational dogma of the trainer, clinician, or strength and conditioning specialist. Differences of

opinion exist as to what the optimal exercises are for LBP (19). Among interventions for the athlete with LBP, spinal stabilization has received considerable attention and, therefore, will be the focus of this discussion. This manuscript will elucidate the research relating to spinal stabilization, discuss muscular changes associated with lumbar spine disorders, and propose an evidence-based lumbar stabilization program. Methods of achieving lumbar spine stabilization that are applicable to the diverse recreational and athletic population will be presented.

Etiology and Risk Factors

The etiology of LBP is multi-factorial with reports of over 50% of episodes occurring for no apparent reason (43). Risk factors for LBP related to muscle performance in the general and athletic population include delayed muscle activation, impaired muscle control, decreased endurance of the extensor musculature, and weakness of the extensors when compared to the flexors (1, 2, 5, 8, 9, 45, 51, 57, 62). Reports eluding to low back injuries from weight training, gymnastics, rugby, and other sports, such as American football, have been documented in the literature (3, 23, 36, 37, 63, 64). Football players, in general, increase

their risk of developing low back pain as their years of involvement with their sport increases (23). Specific conditions such as early degenerative changes and stress fractures of the lumbar spine are more common in American football players than that of the general population (3, 23, 36, 37). Low back injuries have been attributed to the recreational and competitive weight training population as well, with low back conditions reported at all age groups, including adolescents (3, 63). In the authors' observation, athletes in particular are afflicted with recurrent episodes of LBP due to the propensity to train through pain and the inherent focus on performance outweighing prevention.

Definitions

Global spinal stabilizers: Musculature primarily responsible for generating movement including the erector spinae, external obliques, quadratus lumborum, and rectus abdominis (4).

Local spinal stabilizers: Musculature with intervertebral attachments that are capable of providing intersegmental stability (61). The multifidus, transversus abdominis, and internal obliques are classified as local stabilizers (61).

Lumbar multifidus: (Figure 1) Deep spinal musculature responsible for spinal extension and posture when contracting bilaterally, and rotation when acting unilaterally (53, 77). Originates at the sacrum, iliac spine, and transverse processes of the spine, spans 2–4 segments and inserts into the spinous processes above the level of origin (42). The multifidus musculature is responsible for lumbar segmental stability as it is able to provide segmental stiffness and control in the neutral zone (29, 55, 76).

Lumbar segmental instability: (a) Loss of control or excessive motion in a segment's neutral zone (57); (b) decreased capacity of the stabilizing system to maintain the neutral zone within physiological limits; (c) a loss of stiffness be-

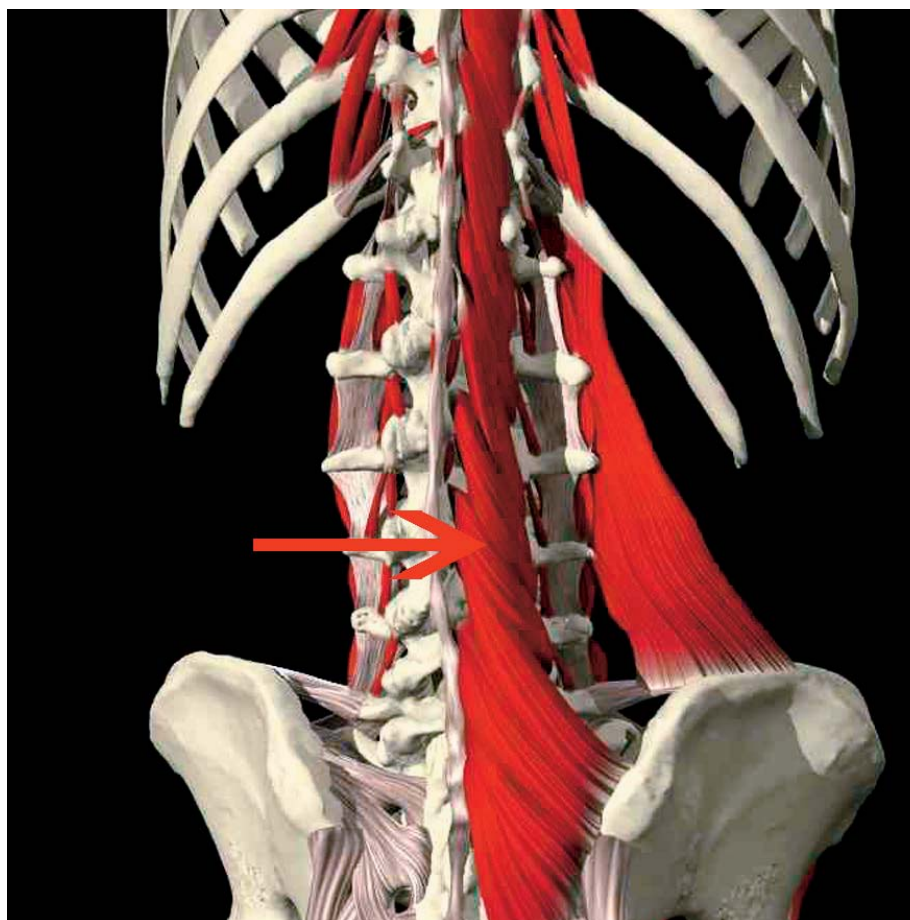


Figure 1. Multifidus muscle group. Lies directly in contact with vertebrae. (Copyright © Primal Pictures Ltd., www.primalpictures.com)

tween motion segments such that normal loads result in pain or stress (27). Segmental instability may be caused by weakness, degenerative disease, loss of passive tension and injury.

Spinal extensors: (Figure 2) Posteriorly located musculature of the vertebral column responsible for actively extending the spine and eccentrically controlling forward flexion. The erector spinae is the largest group of spinal extensors (53).

Spinal flexors: Anterior and laterally located musculature of the pelvis and vertebral column responsible for actively flexing the spine against gravity. The flexors isometrically contract as a means of stabilizing the ribs and pelvis during lifting, pushing, or pulling. Spinal flexors include

the abdominal musculature, psoas major, and internal/external obliques when acting bilaterally (53).

Spinal neutral zone: Range of displacement near the spine segments' neutral position where minimal resistance is required of the osteoligamentous structures. The neutral zone may increase with injury, articular degeneration, loss of passive stiffness, weakness, or inhibition of the stabilizing musculature (57). When the neutral zone is increased, the spine may become unstable (57).

Spinal stabilization exercises: Exercises designed to recruit muscles capable of enhancing stability of the spine(11) and stiffness through training muscular activation patterns. Spinal stability is desirable for prevention of aberrant mobility

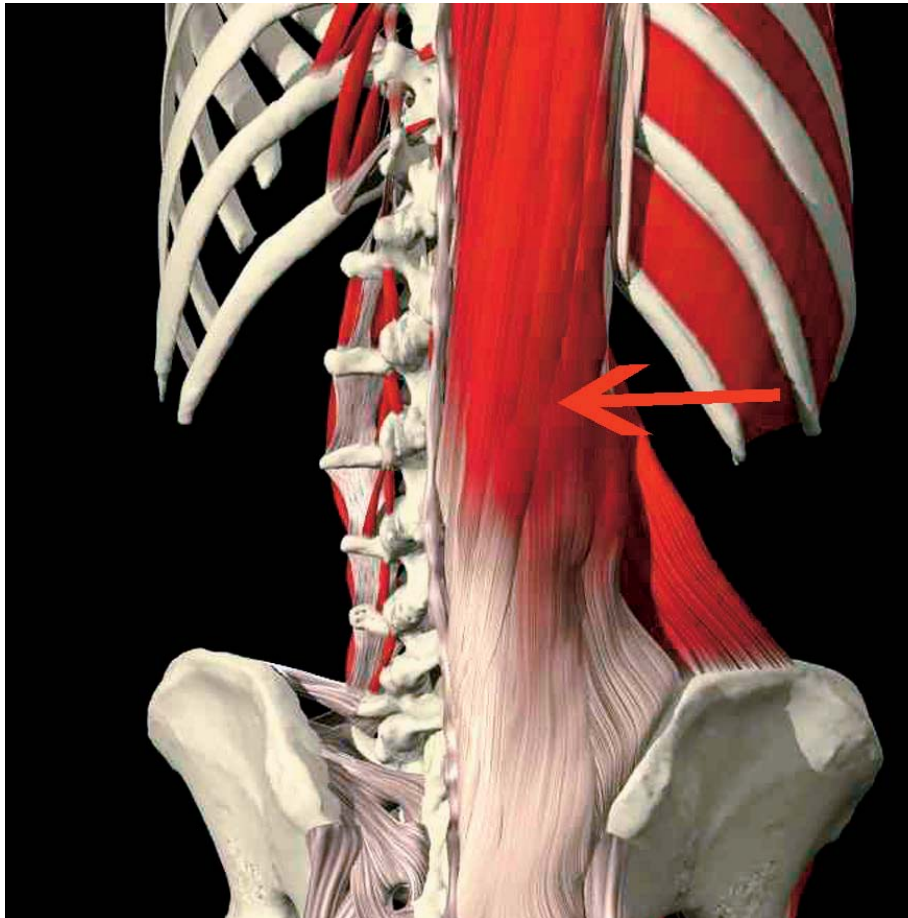


Figure 2. Spinal extensor musculature of the low back. (Copyright © Primal Pictures Ltd., www.primalpictures.com)

agnose the cause of LBP, however, are beyond the scope of this manuscript.

Deficits in strength, size, density, coordination, and activation of the lumbar stabilizing musculature following a low back injury or episode of LBP have been affirmed in the literature (1, 10, 11, 15–18, 35, 38, 39, 47, 60, 61, 66, 67), lending credibility to approaches designed to strengthen and stabilize the lumbar spine following injury.

Spinal Stabilization

Interventions for the athlete with LBP often include a confrontational approach, whereas the athlete will continue to participate in training to tolerance; however, the athlete is often encouraged to push beyond their limits through sport-specific functions, and intrinsic muscle stabilization does not present a training priority. Routines to treat and/or prevent LBP include general exercise, a graduated return to sport-specific tasks, spinal conditioning, and spinal stabilization exercises. Individuals designing programs for the athlete with LBP may or may not prescribe spinal stabilization as part of the conditioning program, as the focus is often to return to sport-specific training. Evidence is compelling to justify spinal stabilization exercises for the individual with LBP due to the associated loss of muscle function, atrophy, and weakness along with the preventative benefits substantiated in the literature (5–9, 11, 28–31, 33–35, 45, 51, 57, 62, 77).

Spinal stabilization essentially consists of both static and dynamic stabilization. When lifting or pushing heavy objects we position our spine in a rigid manner to increase torque and stabilize the trunk, which is referred to as static stabilization and requires activity primarily of the global stabilizers. Dynamic stabilization on the other hand is present through both neurological activation of the muscular system, direct muscle capabilities, and passive tension. Dynamic stabilization requires coordinated re-

at the neutral zone, decreasing pain and impairment associated with instability, and decreasing injury risk.

Transversus abdominis: (Figure 3) Transversely oriented deep abdominal responsible for local stabilization. Originates at the inner surface of the lower 6 ribs, diaphragm, thoracolumbar fascia, and iliac crest and inserts at the linea alba deep to the rectus abdominis (42). The action of the musculature is to draw the abdominal wall in toward the spine maintaining levels of intra-abdominal pressure and imparting tension to the thoracolumbar and sacroiliac spine (32–34, 55, 56, 61).

Diagnosis

Despite technological advances, the identification of a specific cause of LBP

may be indefinable (27, 46, 49, 52, 68, 69), and the pursuit of valid methods to diagnose and treat LBP remains a research priority. Although a multitude of conditions exist, it is not uncommon for the source of an individual's LBP to remain elusive despite diagnosis (52, 68, 69). Common clinical diagnoses include but are not limited to: osteoarthritis, discogenic disorders, spinal stenosis, joint abnormalities, facet and sacroiliac disorders, ligament sprains, muscle strains, spondylolysis, and spinal instability. Mechanical diagnosis of LBP based on classifying patients into relevant subgroups using diagnostic algorithms and testing clusters (13, 46, 49, 68, 69, 74, 78–80) has shown promise in the areas of discogenic disorders (14, 79), facet disorders (78, 79), and sacroiliac syndromes (79, 80). Methods to di-

cruitment of the local stabilization musculature (61). Following injury the dynamic stabilization system is often affected (28–30, 33, 34).

The goals of spinal stabilization are to (a) increase the capacity of the muscular stabilizing system to maintain the neutral zone of the spine within its physiological limits (29, 55, 56, 61, 76); (b) increase the low back's tolerance to insult through the conditioning of key musculature; (c) restore muscle size, strength, and endurance; (d) re-establish coordinated muscle activity as required for prevention of recurrence and restoration of function (11, 28); and (e) reduce pain associated with spinal instability.

Structures Responsible for Stabilization

Stabilization of the spine is achieved through passive structures and the neural/muscular systems. The passive structures are often insufficient for stabilization during dynamic activities that challenge the spine's neutral zone, particularly among individuals with LBP. Due to the relative insufficiency of the passive stabilizers the muscular stabilizers must therefore fulfill the need for stabilization; however, in the individual with LBP, this function is often suppressed or inhibited.

The passive structures, including ligaments, capsules, and osseous structures, provide stabilization through tension, bone congruence, and reflex activation of the stabilizing musculature. Injury, degenerative changes, and adaptive lengthening of the passive structures may reduce their ability to provide normal stiffness and reflex muscle activation (66, 67), thus compromising stability. When stability is compromised at a specific segment or multiple segments, the neutral zone increases. This increase can potentially (a) increase pain, (b) increase injury risk through suppressed function of reflex stabilizers, and (c) decrease sport performance and function.

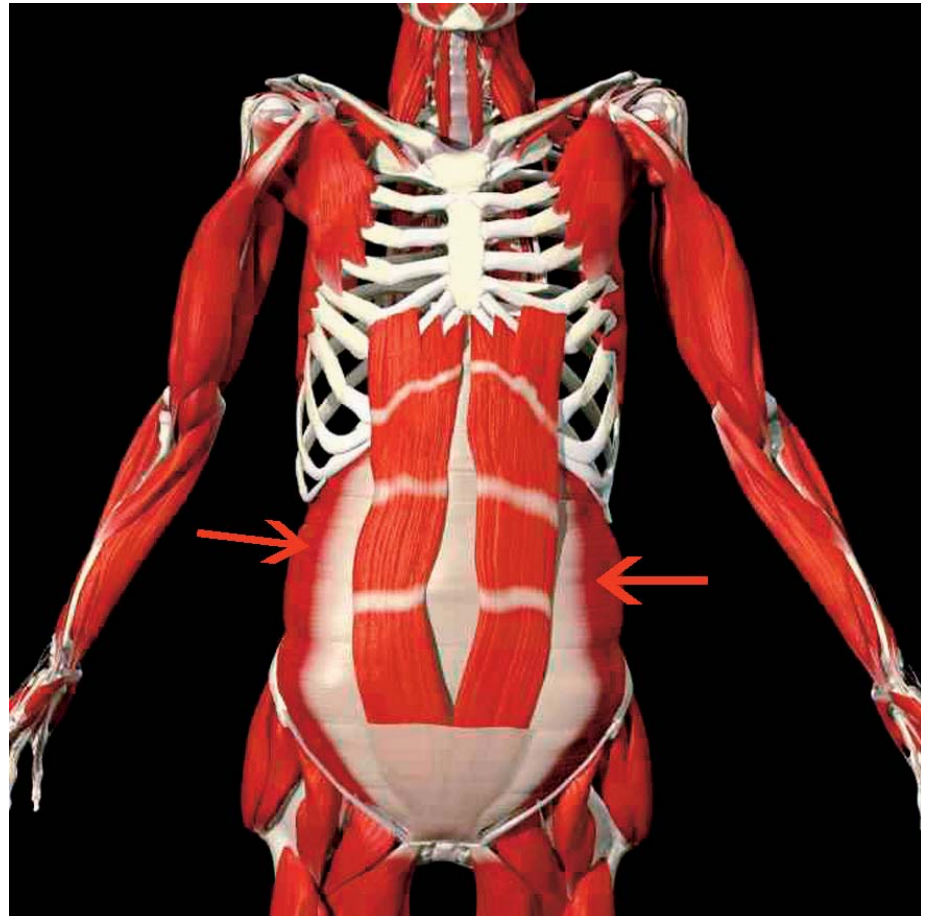


Figure 3. Transversus abdominis. Arrows point to the transversely oriented muscle fibers that lie deep to the rectus abdominis. (Copyright © Primal Pictures Ltd., www.primalpictures.com)

It is generally agreed upon that all of the spine muscles may play a role in ensuring spinal stability (8, 21, 27) during high-level activity, such as lifting heavy weights or competitive sports. In the healthy spine, the trunk musculature functions to control and initiate movement, respond to loading and postural perturbations, provide stiffness, minimize aberrant movements, and provide a stable base for activity. While all muscles of the trunk play a role in stability to some degree, certain muscles have a more specialized function than others. Stabilization of the lumbar spine is achieved through muscles classified as either having local (deep and intrinsic) or global stabilizing function. The local spinal stabilizers have received considerable attention in the literature due to their ability to prevent movement

outside the spine's neutral zone. Additionally, research indicates that local stabilization abilities are suppressed following an episode of LBP, specifying the need to address these muscles. In the authors' experience, athletes invariably receive some form of spinal stabilization as part of their conditioning routines; however, the focus is often on the large global stabilizers and deficient in the area of local stabilization.

The global stabilizers include the rectus abdominis, spinal extensors, external obliques, quadratus lumborum, and psoas muscles. The global stabilizers function in response to voluntary effort during the initiation of spinal movement and during challenging activities that require a stiff spine.

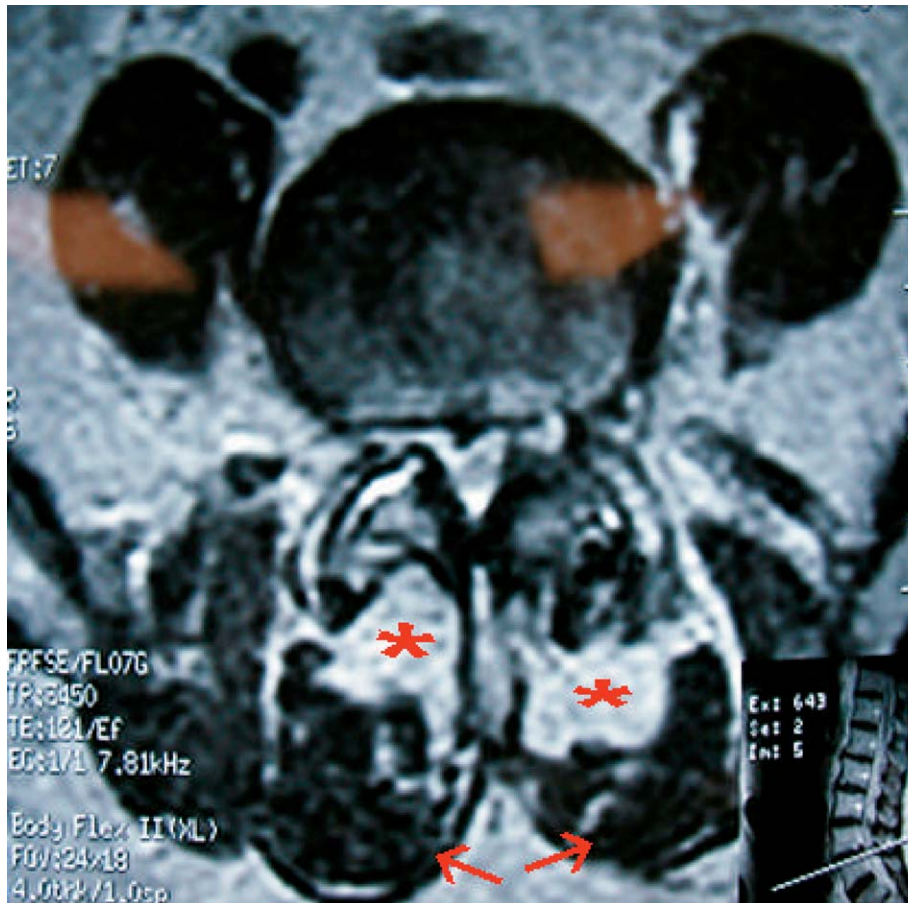


Figure 4. Fatty infiltration (white regions identified by *) of the multifidus in an individual with chronic low back pain. The multifidus (*) is located deep to the erector spinae (arrows).

Much attention in the literature has been focused on the spinal extensors, particularly the erector spinae (2, 5, 7, 24, 62), which will be the primary global stabilizer lending itself to discussion in this manuscript. The spinal extensors serve to extend the lumbar spine, maintain the natural lordosis, and stabilize the spine in the closed packed position during lifting and other activities requiring lumbar stabilization. Evidence suggests that there is decreased endurance and measurable atrophy of the spinal extensors in the LBP population (10, 35, 39, 54). Weakness of the spinal extensors relative to the spinal flexors increases an individual's risk for developing LBP (45). In addition, a decreased reflex response of the extensors in reaction to movement when compared to

the flexors may predispose one to injury (9). Lastly, evidence suggests that increasing the strength of the spinal extensors may decrease the likelihood of developing LBP (5, 24, 51).

The local stabilizers' function is to provide a stable base in preparation or anticipation of trunk and extremity movements. The local stabilizers fulfill the role of stabilizing the spine when the integrity of the spine's neutral zone is challenged. In the healthy spine, muscle contraction of the local stabilizers is automatic and precipitated by movement of the extremities or trunk, unlike the injured spine whereas the activation is suppressed or delayed. The local stabilizers include the multifidus (Mult), transversus abdominis (TrA), and internal obliques.

The Mult is a deep intrinsic spinal muscle that will maintain posture, extend, and rotate the spine. Additionally, the Mult contracts in anticipation of trunk and extremity movement to provide a stable base. In the healthy lumbar spine, the Mult provides stabilization locally by minimizing movement of the spinal column and maintaining the theoretical neutral zone (55, 66, 67, 76). Evidence suggests that the Mult undergoes pathological changes following an episode of LBP, such as suppressed activation, atrophy, fatty infiltration (Figure 4), and weakness (11, 30, 29, 38, 47, 60, 77).

The TrA functions to flatten and compress the abdominal wall. The TrA is invariably activated in anticipation of trunk and extremity movement to provide stability of the lumbar spine (31–34) similar to the Mult. Weakness or delayed activation of the TrA may directly affect local spinal stabilization as a result. Research has reported reduced activation ability of the TrA in the LBP population (16, 31, 33, 34) as well as decreased recurrence rates of LBP on those who are able to restore their ability to contract the TrA (28).

Among clinicians and strength and conditioning specialists, it is generally agreed upon that all of the muscles surrounding the spine provide stabilization to some degree during physical activity. While the authors agree that a comprehensive approach to spinal stabilization and conditioning is necessary in the athletic population, specific muscles require attention in the athlete with LBP. The erector spinae, Mult, and TrA have received much attention in the literature due to their stabilizing abilities and associated deficits following an episode of LBP. Therefore, these select muscles will be the focus of this discussion.

Stabilization Program

Individuals with LBP will recover at varying time frames depending upon the nature of their injury, diagnosis, and ability to recover without aggravation of



Figure 5. (a) Supine abdominal draw exercise with natural lordosis using a pressure cuff to monitor positioning. (b) Supine abdominal draw exercise with natural lordosis and simultaneous upper and lower extremity flexion.

their condition. Due to the competitive nature of athletics, known risk factors for LBP, and prevalence of LBP, spinal exercises are invariably incorporated into both training and rehabilitation programs.

The proposed stabilization program includes 3 progressive stages. The program focuses on the local stabilization musculature in phase one, with progression into the final stage, which incorporates the global stabilizers. Progression through the 3 stages is dependent upon the athlete's abilities, pain level, and stage of injury. In cases where the stabilization program is used for prevention, the athlete may progress on a timeline based on their abilities to master the exercises. Individual progression, however, will vary as the initial tasks may require a few sessions in some individuals due to suppressed muscle activity associated with LBP.

This program is recommended as part of a comprehensive individualized conditioning program. It should be performed daily during stage 1 since neural activation is essential to mastering the required tasks. The program can then be decreased to 3 times a week during stage 3 since the athlete will typically return

to pre-morbid activities at this time and will be performing the routine as part of a comprehensive program.

Stage 1

All participants begin the program with stage 1, which includes 3 progressive exercises. An individual's level of conditioning will not directly influence their ability to activate the local musculature following an episode of LBP; therefore, stage 1 is of primary importance. The initial goal of stage 1 is to activate the local stabilizing muscles without compensation by the large global stabilizers. This stage requires neural activation and muscle coordination. The final goal of stage 1 involves maintaining a co-contraction of the local stabilizers while performing rapid alternating extremity movements in the sagittal plane.

Exercise 1 (Figure 5a) is referred to as the supine abdominal draw. The athlete is asked to lie on their back with their hips and knees flexed 45 degrees, assuming a natural lordosis of the lumbar spine. A blood pressure cuff inflated to 30–40 mm hg is placed under their spine. Once in position, the athlete is asked to draw their abdomen in and up. The athlete is asked not to hold their breath and to maintain the natural lor-

dosis by avoiding the desire to flatten the back during this task. The read-out on the pressure cuff should remain relatively constant throughout the exercise. Flattening the back and not maintaining the desired lordosis will result in a rise in the pressure read-out, which is then used as a signal for the athlete to resume the lordotic position. This exercise activates both the TrA by drawing in the abdomen and the Mult by maintaining the lordosis. This exercise is held for 10 repetitions of 30 second durations. Once mastered the co-contraction performed in this exercise is utilized for the progressions in the remainder of the exercise program.

Exercise 2 involves maintaining the co-contraction of the TrA and Mult while performing rapid alternating arm flexion for a duration of 1 minute for 3–5 sets. Research has identified a feed-forward anticipatory contraction of the TrA and Mult in response to upper and lower extremity movements (32–34). Once upper extremity (UE) motions are tolerated, the athlete is instructed to perform rapid alternating hip flexion of approximately 6–12" while maintaining a co-contraction of the TrA and Mult. Once the athlete is able to maintain a co-contraction of the TrA and Mult during

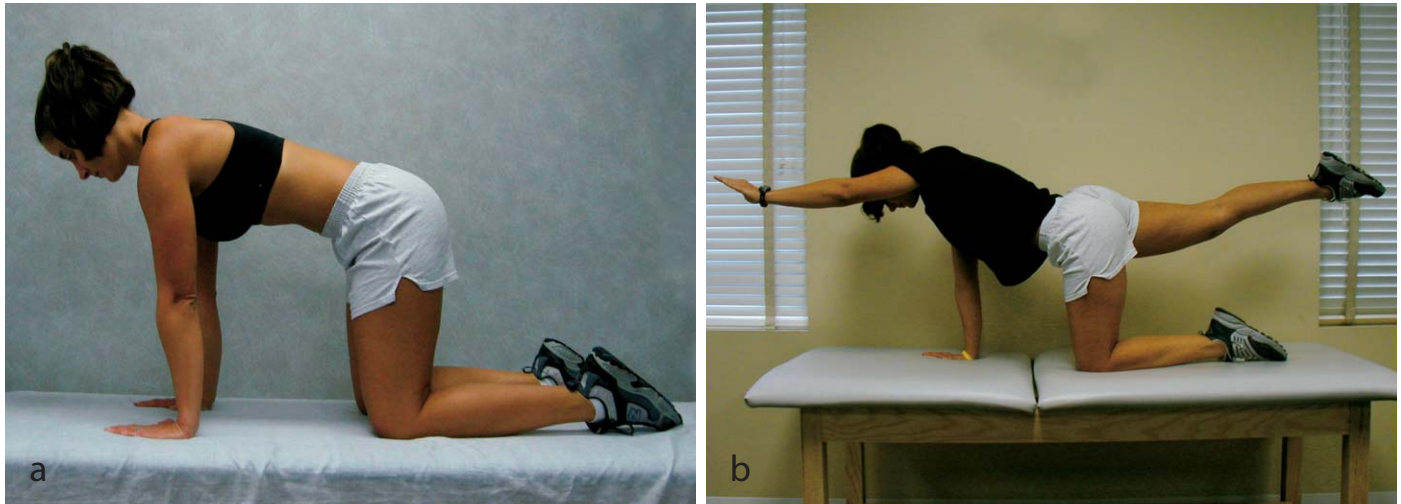


Figure 6. (a) Quadrupedal position with co-contraction of the transversus abdominis and multifidus. (b) Quadrupedal position with co-contraction and simultaneous upper and lower extremity extension.

movements of the upper and lower extremities, the task is mastered and the athlete can advance to the next exercise.

Exercise 3 (Figure 5b) requires the athlete to maintain the co-contraction and natural lordosis while rapidly alternating the arms and hips into flexion while lying on their back. The procedure involves simultaneously raising the left arm and right lower extremity (LE) followed by raising the right UE and left LE. This is carried out for 3 sets of 20 repetitions or durations of 1 minute for 3–5 repetitions. The athlete can advance to stage 2 once they demonstrate the ability to maintain the co-contraction of the TrA and Mult while performing extremity movements in the supine position and without complaints of pain.

Stage 2

The progression to stage 2 involves exercises that require a co-contraction of the TrA and Mult during the assumption of additional positions, with the added recruitment of the erector spinae, shoulder, and hip extensor musculature.

Exercise 1 (Figure 6a) requires the individual to assume a quadrupedal position while maintaining a co-contraction of the local stabilizers. The individual is

asked to raise the arms forward into flexion alternating rhythmically from the right to the left arm. Once this is mastered, LEs are brought into relative hip extension while maintaining the local co-contraction.

Exercise 2 (Figure 6b) involves alternately raising their upper and lower extremities simultaneously (right arm with left leg, then left arm and right leg) while maintaining the co-contraction of the TrA and Mult. When performed according to the recommendations outlined, this exercise will activate both the local and global stabilizers. The exercise is typically performed for 3 sets of 20 repetitions or 10 10-second holds. The individual should demonstrate the ability to maintain a steady position with minimal sway while strictly holding a co-contraction of the TrA and Mult prior to advancing to the next exercise.

Exercise 3 involves performing the same exercise described in exercise 2 with the addition of ankle cuff weights. Ankle cuff weights will challenge the stabilizing musculature by increasing muscle activation when raising the leg, as well as through the relative imbalance of weight between the upper and lower extremities.

Stage 3

The final stage requires the participant to maintain the abdominal draw and natural lordosis co-contraction during the performance of exercises designed to recruit global stabilizers. The 3 exercises described in stage 3 are considered to be of equal challenge and may be performed as tolerated by the individual using principles of exercise progression.

Exercise 1 (Figure 7) requires the individual to assume the prone position while lying flat on a table. The participant first assumes the abdominal draw, then begins extending the spine off the mat to their end-range limit of extension while maintaining the abdominal draw. The feet are not stabilized as this exercise is designed to be performed in a controlled manner, primarily seeking the effort of the spinal extensors. This exercise may be advanced by adding pillows under the waist to increase the range of motion of the exercise, by extending the arms overhead, or by adding cuff weights to the wrists while holding the arms overhead.

Exercise 2 (Figure 8) requires the individual to perform the “side bridge”



Figure 7. Prone trunk extension with abdominal draw.



Figure 8. Side bridge exercise while maintaining abdominal draw.

exercise (48) traditionally used to strengthen the quadratus lumborum. Participants are required to lie on their side with the legs extended. The exercise requires the individual to lift

their hips off the table to a level where their body is straight and supported by their weight-bearing arm and feet. Individuals are instructed to maintain the TrA-Mult co-contrac-

tion and hold the position for a duration of 10 seconds for a total of 10 repetitions. This exercise is performed on both the right and left sides. To advance this exercise, the participant is instructed to repetitively reach their arm straight out to the front (Figure 9a) and then toward the ceiling (Figure 9b) while maintaining the side bridge position with a TrA-Mult co-contraction.

Exercise 3 (Figure 10) requires the individual to stand on an unstable surface, such as a balance board, which will recruit the stabilization musculature through postural perturbations. While standing on the unstable surface, the participant is instructed to assume a position of slight knee and hip flexion while maintaining a co-contraction of the local stabilizers. During the exercise, the individual is instructed to rapidly alternate arm flexion while maintaining their balance and position. This exercise is advanced by using dumbbells during the arm movement. This exercise is usually performed for a duration of 1 minute for 2–5 repetitions. As the exercise is mastered the weights may be increased and the eyes may be closed to further challenge the local stabilizers.



Figure 9. (a) Side bridge exercise with abdominal draw and horizontal upper extremity movement. (b) Side bridge exercise with abdominal draw and horizontal upper extremity movement.



Figure 10. Standing stabilization on an unsteady surface maintaining co-contraction of multifidus and transversus abdominis with alternating upper extremity flexion.

strength and conditioning specialist. While intervention is often sport specific and based on the treatment provider's acumen, it is common practice to recommend general stabilization exercises. Although general stabilization exercises are useful as a means of spinal conditioning, exercises specifically designed to challenge and activate the local stabilizers are of primary importance due to the documented pathological changes in these muscles following or associated with LBP. Furthermore, evidence has substantiated the preventative benefits associated with training the local stabilizers and the spinal extensors.

Spinal stabilization exercises, when performed according to the progressions recommended in this manuscript, require no special equipment or space, may be progressed based on the individual's ability and learning curve, and are safe and applicable for the more common low back conditions experienced by athletes. Lastly, the stabilization exercises recommended may be prescribed as part of a general conditioning program considering the known risk for LBP associated with certain sports.

Key Points

- Evidence in the literature has identified the importance of local stabilization exercises for the management of LBP.
- The spinal extensors, Mult, and TrA are adversely affected following an episode of LBP.
- LBP is associated with reduced TrA and Mult activation.
- LBP is associated with atrophy of the Mult and spinal extensor musculature.
- Training the TrA and Mult reduces the recurrence of LBP. ♦

References

1. ALARANTA, H., H. HURRI, M. HELIOVAARA, A. SOUKKA, AND R. HARJU. Non-dynamometric trunk performance tests: Reliability and normative

Conclusion

LBP is a common condition that may affect athletes at a greater prevalence than the general population. Individu-

als involved in program design for the athlete with a current or a past history of LBP often include the physician, physical therapist, athletic trainer, and

- data. *Scand. J. Rehabil. Med.* 26:211–215. 1994.
2. ALARANTA, H., S. LUOTO, M. HELIOVAARA, AND H. HURRI. Static back endurance and the risk of low-back pain. *Clin. Biomech.* 10:323–324. 1995.
 3. ALEXANDER, M.J. Biomechanical aspects of lumbar spine injuries in athletes: A review. *Can. J. Appl. Sport Sci.* 10:1–20. 1985.
 4. BERGMARK, A. Stability of the lumbar spine. A study in mechanical engineering. *Acta. Orthopaedica Scandinavica.* 230:S20–S24. 1989.
 5. BIERENG-SORENSEN, F. Physical measurements as risk factors for low-back trouble over a one year period. *Spine.* 9:106–119. 1984.
 6. BONO, C.M. Low-back pain in athletes. *J. Bone Joint. Surg. Am.* 86:382–396. 2004.
 7. CARPENTER, D.M., AND B.W. NELSON. Low back strengthening for the prevention and treatment of low back pain. *Med. Sci. Sports Exerc.* 31:18–24. 1999.
 8. CHOLEWICKI, J., AND S.M. MCGILL. Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain. *Clin. Biomech. (Bristol, Avon).* 11:1–15. 1996.
 9. CHOLEWICKI, J., S.P. SILFIES, R.A. SHAH, H.S. GREENE, N.P. REEVES, K. ALVI, AND B. GOLDBERG. Delayed trunk muscle reflex responses increases the risk of low back injuries. *Spine.* 30:2614–2620. 2005.
 10. COOPER, R.G., W.F. ST CLAIR, AND M.I. JAYSON. Radiographic demonstration of paraspinal muscle wasting in patients with chronic low back pain. *Br. J. Rheumatol.* 31:389–394. 1992.
 11. DANNEELS, L.A., G.G. VANDERSTRAETEN, D.C. CAMBIER, E.E. WITVROUW, J. BORGIOIS, W. DANKAERTS, AND H.J. DECUYPER. Effects of three different training modalities on the cross sectional area of the lumbar multifidus muscle in patients with chronic low back pain. *Br. J. Sports Med.* 35:186–191. 2001.
 12. DE GIROLAMO, G. Epidemiology and social costs of low back pain and fibromyalgia. *Clin. J. Pain.* 7(Suppl. 1):S1–S7. 1991.
 13. DELITTO, A., R.E. ERHARD, AND R.W. BOWLING. A treatment based classification approach to low back syndrome: Identifying and staging patients for conservative treatment. *Phys. Ther.* 75:470–489. 1995.
 14. DONELSON, R., C. APRILL, R. MEDCALE, AND W. GRANT. A prospective study of centralization of lumbar and referred pain: A predictor of symptomatic discs and anular competence. *Spine.* 22:1115–1122. 1997.
 15. ENGELHARDT, M., I. REUTER, J. FREIWALD, T. BOHME, AND A. HALBSGUTH. Spondylolysis and spondylolisthesis and sports. *Orthopade.* 26:755–759. 1997.
 16. FERREIRA, P.H., M.L. FERREIRA, AND P.W. HODGES. Changes in recruitment of the abdominal muscles in people with low back pain. Ultrasound measurement of muscle activity. *Spine.* 29:2560–2566. 2004.
 17. FERREIRA, P.H., M.L. FERREIRA, C.G. MAHER, R.D. HERBERT, AND K. REFSHAUGE. Specific stabilization exercise for spinal and pelvic pain: A systematic review. *Aust. J. Physiother.* 52:79–88. 2006.
 18. FLICKER, P.L., J.L. FLECKSTEIN, K. FERRY, J. PAYNE, C. WARD, T. MAYER, R.W. PARKEY, AND R.M. PESHOCK. Lumbar muscle usage in chronic low back pain. Magnetic resonance image evaluation. *Spine.* 18:582–586. 1993.
 19. FOSTER, N., K. THOMPSON, D. BAXTER, AND J.M. ALLEN. Management of nonspecific low back pain by physiotherapists in Britain and Ireland. *Spine.* 24:1332–1342. 1999.
 20. FRYMOYER, J.W., AND W.L. CATSBARIL. An overview of the incidences and costs of low back pain. *Orthop. Clin. North Am.* 2:263–271. 1991.
 21. GARDNER-MORSE, M.G., I.A. STOKES, AND J.P. LAIBLE. Role of muscles in lumbar spine stability in maximum extensor efforts. *J. Orthop. Res.* 13:802–808. 1995.
 22. GARRY, J.P., AND J. MCSHANE. Lumbar spondylolysis in adolescent athletes. *J. Fam. Pract.* 47:145–149. 1998.
 23. GERBINO, P.G., AND P.A. D’HEMECOURT. Does football cause an increase in degenerative disease of the lumbar spine? *Curr. Sports Med. Rep.* 1:47–51. 2002.
 24. GUNDEWALL, B., M. LILJEQVIST, AND T. HANSSON. Primary prevention of back symptoms and absence from work. A prospective randomized study among hospital employees. *Spine.* 18:587–94. 1993.
 25. HALVORSEN, T.M., S. NILSSON, AND P.H. NAKSTAD. Stress fractures. Spondylolysis and spondylolisthesis of the lumbar vertebrae among young athletes with back pain. *Tidsskr. Nor. Laegeforen.* 116:1999–2001. 1996.
 26. HARVEY, J., AND S. TANNER. Low back pain in young athletes. A practical approach. *Sports Med.* 12:394–406. 1991.
 27. HICKS, G.E., J.M. FRITZ, A. DELITTO, AND S.M. MCGILL. Preliminary development of a clinical prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. *Arch. Phys. Med. Rehabil.* 86:1753–1762. 2005.
 28. HIDES, J.A., G.A. JULL, AND C.A. RICHARDSON. Long-term effects of specific stabilizing exercises for first-episode low back pain. *Spine.* 26:E243–E248. 2001.
 29. HIDES, J.A., C.A. RICHARDSON, AND G.A. JULL. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine.* 21:2763–2769. 1996.
 30. HIDES, J.A., M.J. STOKES, M. SAIDE, G.A. JULL, AND D.H. COOPER. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine.* 19:165–172. 1994.
 31. HODGES, P.W., AND C.A. RICHARDSON. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transverse abdominis. *Spine.* 21:2640–2650. 1996.
 32. HODGES, P.W., AND C.A. RICHARDSON. Contraction of the abdominal

- muscles associated with movement of the lower limb. *Phys. Ther.* 77:132–141. 1997.
33. HODGES, P.W., AND C.A. RICHARDSON. Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. *J. Spinal. Disord.* 11:46–56. 1998.
 34. HODGES, P.W., AND C.A. RICHARDSON. Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. *Arch. Phys. Med. Rehabil.* 80:1005–1012. 1999
 35. HULTMAN, G., M. NORDIN, H. SARASTE, AND H. OHLSEN. Body composition, endurance, strength, cross-sectional area, and density of MM erector spinae in men with and without low back pain. *J. Spinal Disord.* 6:114–123. 1993.
 36. IWAMOTO, J., H. ABE, Y. TSUKIMURA, AND K. WAKANO. Relationship between radiographic abnormalities of lumbar spine and incidence of low back pain in high school and college football players: A prospective study. *Am. J. Sports Med.* 32:781–786. 2004.
 37. IWAMOTO, J., H. ABE, Y. TSUKIMURA, AND K. WAKANO. Relationship between radiographic abnormalities of lumbar spine and incidence of low back pain in high school rugby players: A prospective study. *Scand. J. Med. Sci. Sports.* 15:163–168. 2005.
 38. KADER, D.F., D. WARDLAW, AND F.W. SMITH. Correlation between the MRI changes in the lumbar multifidus muscles and leg pain. *Clin. Radiol.* 55:145–149. 2000.
 39. KANKAANPAA, M., S. TAIMELA, D. LAAKSONEN, O. HANNINEN, AND O. AIRAKSINEN. Back and hip extensor fatigability in chronic low back pain patients and controls. *Arch. Phys. Med. Rehabil.* 79:412–417. 1998.
 40. KEENE, J.S. Low back pain in the athlete. From spondylogenic injury during recreation or competition. *Postgrad. Med.* 74:209–213, 217. 1983.
 41. KEENE, J.S., AND D.S. DRUMMOND. Mechanical back pain in the athlete. *Compr. Ther.* 11:7–14. 1985.
 42. KENDALL, F.P., AND E.K. MCCREARY. *Muscles Testing and Function With Posture and Pain* (5th ed). Baltimore, MD: Lippincott Williams & Wilkins, 2005. pp. 165–197.
 43. KRAMER J. *Intervertebral Disk Diseases* (2nd ed.). New York, NY: Thieme Medical Publishers, 1990. pp. 134.
 44. KUJALA, U.M., J.J. SALMINEN, S. TAIMELA, A. OKSANEN, AND L. JAAKKOLA. Subject characteristics and low back pain in young athletes and non-athletes. *Med. Sci. Sports Exerc.* 24:627–632.1992.
 45. LEE, J.H., Y. HOSHINO, K. NAKAMURA, Y. KARIYA, K. SAITA, AND K. ITO. Trunk muscle weakness as a risk factor for low back pain. A 5-year prospective study. *Spine.* 24:54–57. 1999.
 46. LONG, A., R. DONELSON, AND T. FUNG. Does it matter which exercise? A randomized control trial of exercise for low back pain. *Spine.* 29:2593–2602. 2004.
 47. MATTILLA, M., M. HURME, H. ALARANTA, L. PALJARVI, H. KALIMO, B. FALCK, M. LEHTO, S. EINOLA, AND M. JARVINEN. The multifidus muscle in patients with lumbar disc herniation. A histochemical and morphometric analysis of intraoperative biopsies. *Spine.* 11:732–738. 1986.
 48. MCGILL, S.M., A. CHILDS, AND C. LIEBSON. Endurance times for low back stabilization exercises: Clinical targets for testing and training from a normal database. *Arch. Phys. Med. Rehabil.* 80:941–944. 1999.
 49. MCKENZIE R.A., AND S. MAY. *The Lumbar Spine: Mechanical Diagnosis and Therapy* (2nd ed.). Waikanae, New Zealand: Spinal Publications New Zealand Ltd, 2003.
 50. MCTIMONEY, C.A., AND L.J. MICHELI. Current evaluation and management of spondylolysis and spondylolisthesis. *Curr. Sports Med. Rep.* 2:41–46. 2003.
 51. MOONEY, V., M. KRON, P. RUMMERFIELD, AND B. HOLMES. The effect of workplace based strengthening on low back injury rates: A case study in the strip mining industry. *J. Occup. Rehab.* 5:157–167. 1995.
 52. NACHEMSON, A. Advances in low-back pain. *Clin. Orthop.* 200:266–278. 1985.
 53. NORKIN, C.C., AND P.K. LEVANGE. *Joint Structure and Function. A Comprehensive Analysis* (2nd ed.). Philadelphia: F.A. Davis Company, 1992. pp.125–162.
 54. ODDSSON, L.I., J.E. GIPHART, R.J. BUIJS, S.H. ROY, H.P. TAYLOR, AND C.J. DE LUCA. Development of new protocols and analysis procedures for the assessment of LBP by surface EMG techniques. *J. Rehabil. Res. Dev.* 34:415–26. 1997.
 55. O’SULLIVAN, P., G.D. PHYTY, L.T. TWOMEY, AND G.T. ALLISON. Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine.* 22: 2959–2967. 1997.
 56. O’SULLIVAN, P.B., L. TWOMEY, AND G.T. ALLISON. Altered abdominal muscle recruitment in patients with chronic back pain following a specific exercise intervention. *JOSPT.* 27:114–124. 1998.
 57. PANJABI, M. The stabilizing system of the spine. Part I function, dysfunction, adaptation, and enhancement. *J. Spinal Disord.* 5:383–389. 1992.
 58. PRENTICE W.E. *Rehabilitation Techniques in Sports Medicine*. St. Louis, MO: Times Mirror/Mosby College Publishing, 1990. pp. 47, 151,164–168.
 59. RALSTON, S. AND M. WEIR. Suspecting lumbar spondylolysis in adolescent low back pain. *Clin. Pediatr. (Phila.)* 37:287–293. 1998.
 60. RANTANEN, J., M. HURME, B. FALCK, H. ALARANTA, F. NYKVST, M. LEHTO, S. EINOLA, AND H. KALIMO. The lumbar multifidus muscle five years after surgery for a lumbar intervertebral disc herniation. *Spine.* 18:568–74. 1993.
 61. RICHARDSON, R.A., C.J. SNIJDERS, J.A. HIDES, L. DAMEN, M.S. PAS, AND J. STORM. The relationship between the transverse abdominis muscles,

- sacroiliac joint mechanics, and low back pain. *Spine*. 27:399–405. 2002.
62. RISSANEN, A., M. HELIOVAARA, H. ALARANTA, S. TAIMELA, E. MALKIA, P. KNEKT, A. REUNANEN, AND A. AROMAA. Does good trunk extensor performance protect against back related disability? *J. Rehabil. Med.* 34:62–66. 2002.
 63. RISSER, W.L. Musculoskeletal injuries caused by weight training. Guidelines for prevention. *Clin. Pediatr.* 29:305–310.1990.
 64. ROSSI, F., AND S. DRAGONI. Lumbar spondylosis and sports. The radiological findings and statistical considerations. *Radiol. Med. (Torino)*. 87:397–400. 1994.
 65. SCHMIDT, C.O., AND T. KOHLMANN. What do we know about the symptoms of back pain? Epidemiological results on prevalence, incidence, progression and risk factors. *Z. Orthop. Ihre Grenzgeb.*143:292–298. 2005.
 66. SOLOMONOW, M., B.H. ZHOU, R.V. BARATTA, Y. LU, AND M. HARRIS. Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 1. Loss of reflexive muscular stabilization. *Spine*. 24:2426–2434. 1999.
 67. SOLOMONOW, M., B.H. ZHOU, M. HARRIS, Y. LU, AND R.V. BARATTA. The ligamento-muscular stabilizing system of the spine. *Spine*. 23:2552–2562. 1998.
 68. SPITZER, W.O. Scientific approach to the assessment and management of activity-related spinal disorders. A monograph for clinicians. Report of the Quebec Task Force on Spinal Disorders. *Spine*. 12:s1–s59. 1987.
 69. SPITZER, W.O. Approach to the problem. *Spine*.12:s9–s11.1987.
 70. STANDAERT, C.J., S.A. HERRING, B. HALPERN, AND O. KING. Spondylolysis. *Phys. Med. Rehabil. Clin. N. Am.* 11:785–803. 2000.
 71. TRAINOR, T.J., AND M.A. TRAINOR. Etiology of low back pain in athletes. *Curr. Sports Med. Rep.* 3:41–46. 2004.
 72. TROUP, J.D., J.W. MARTIN, AND D.C. LLOYD. Back pain in industry: A prospective survey. *Spine*.6:61–69.1981.
 73. VAN DEN HOOGEN, H.J.M., B.W. KOES, J.T.M. VAN EIJEK, L.M. BOUTER, AND W. DEVILLE. On the course of low back pain in general practice: A one year follow-up study. *Ann. Rheum. Dis.* 57:13–19. 1998.
 74. VON KORFF, M. Studying the natural history of back pain. *Spine*. 19:s2041–s2046. 1994.
 75. WADDELL, G. A new clinical model for the treatment of low back pain. *Spine*. 12:632–644. 1987.
 76. WILKE, H.J., S. WOLF, L.E. CLAES, M. ARAND, AND A. WIESEND. Stability increase of the lumbar spine with different muscle groups. A biomechanical in vitro study. *Spine*. 20:192–198. 1995.
 77. YOSHIHARA, K., Y. SHIRAI, Y. NAKAYAMA, AND S. UESAKA. Histochemical changes in the multifidus muscle in patients with lumbar intervertebral disc herniation. *Spine*. 26:622–626. 2001.
 78. YOUNG, S., AND C.N. APRILL. Characteristics of a mechanical assessment for chronic lumbar facet joint pain. *J. Manual Manipulative. Ther.* 8:78–84. 2000.
 79. YOUNG, S., C. APRILL, AND M. LASLETT. Correlation of clinical examination characteristics with three sources of chronic low back pain. *Spine. J.* 3:460–465. 2003.
 80. YOUNG, S., M. LASLETT, C.N. APRILL, AND C. KELLY. The sacroiliac joint: Comparing physical examination and diagnostic block arthrography. *J. Orthop. Sports Phys. Ther.* 30:A34. 2000.

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Kolber

Morey J. Kolber is an assistant professor at Nova Southeastern University in Ft. Lauderdale, Florida.



Beekhuizen

Kristina Beekhuizen is an assistant professor at Nova Southeastern University in Ft. Lauderdale, Florida.