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Commentary

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Intervertebral Disc and Physical Exercise: An Evidence-Based Remark

João Pinheiro, MD, PhD and Pedro Figueiredo, MD*

Faculty of Medicine, Physical and Rehabilitation Medicine, University of Coimbra, Coimbra, Portugal

Summary

Back pain is a frequent complaint in elite athletes. The intervertebral disc (DIV) is a preponderant element in the programming of movement and posture in humans. The homeostasis of DIV tissues is maintained through a close balance between matrix synthesis and its degradation, but physical exercise can introduce dramatic biological changes. AAs comment on the impact of physical exercise on the quality and integrity of the intervertebral disc.

Keyword

Intervertebral Spine Disc, Exercise, Injury, Prevention

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Intervertebral disc (IVD) is a main element in the evolution of human movement and posture. It gives stability, distributes axial load and has proprioceptive function. Usually the sum of 23 IVD corresponds to 20 to 33% of the spine length.

IVD is a heterogeneous structure formed by a fibrous annulus (type I collagen) and a nucleus pulpous (collagen type II, aggrecan, elastin, water and chondrocytes-like cells). Regarding innervation, IVD has afferent mechanoreceptors in the outer annulus as well as in the posterior and anterior longitudinal ligaments [1].

The homeostasis of the IVD tissues is maintained through an accurate balance between matrix synthesis and degradation, but physical exercise can introduce biological and structural changes which can modify IVD functions [2].

IVD pathology is common with exercise, being reported moderate and severe injuries in 52% of the Olympic athletes [3]. Thus back pain is a common complaint in elite athletes. A demanding training volume in athletes and a low activity level volume in individuals is related with a high prevalence of disc pathology [4].

Low-impact and moderate physical exercise is a good stimulus for an adequate nutrition of fibrous annulus and nucleus pulpous [5]. Various elements such as oxygen and lactate move to IVD (via diffusion and pumping action), due to the mechanical stresses produced by the movement and the optimal degree of disc hydration depends also on this condition [6].

There is little knowledge regarding the anabolic benefits of exercise in IVD and spinal tissues [7]. Regular walking or low-demand running improves IVD composition, promoting better hydration and increasing the amount of proteoglycans), contributing to disc hypertrophy. On the contrary high impact activities, static and rotational forces, flexion with compression, torsion, damage of vertebral end-plate and static standing may deteriorate the quality of disc materials [2,7,8].

In the pulpous nucleus there is a loss of hydration and vascularization, reduction of cellularity and degradation of the extracellular matrix. In fibrous annulus there is a rapid increase in the number of nociceptive fibres relative to proprioceptive [4]. Degeneration is characterized by reduced disc height and the difficulty in distinguishing the separation) between the fibrous annulus and the nucleus pulpous. Catabolic cellular activity also determines low disc pressure and conditions in the pulpous nucleus for high shear forces.

Decreased proteoglycan content in the pulpous nucleus is considered a major factor affecting the dynamic viscoelastic properties of the entire disc. Fibrous annulus fissures resulting from demanding physical exercise and degenerative process will thus condition instability in the spine (lesional cascade proposed by Kirkcaldy-Willis) [9].

***Corresponding author:** João Pinheiro, MD, PhD, Faculty of Medicine, Physical and Rehabilitation Medicine, Sports Medicine, University of Coimbra, Coimbra, Portugal

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However, it is not just the disc and chondral bone that are involved in degeneration. Fat infiltration (FI) and volume reduction have been described as characteristic changes that occur in muscle degeneration.

The degree of intramuscular fat infiltration seems to affect the trunk extensor muscles more, evolving from lower to upper levels [10].

Spinal pain can modify central muscle activation, with increased latency, reduced muscle strength production, and deterioration in coordination and proprioception [11].

Individuals with chronic low back pain had lower acuity to detect changes in trunk position and demonstrated significantly greater trunk repositioning errors during back flexion compared with pain-free individuals [12]. Adaptations in central movement control can thus justify loss of core control and especially increase muscle co-contractions, promoting increased motor fatigue [13-15].

This reduction in proprioceptive spinal input in cortical sensorimotor circuits may be associated with neuroplasticity changes.

This cortical reorganization may explain the persistent changes in motor control and the loss of functionality and performance associated with the maintenance of low back pain [10] as well as maintaining biomechanical and load anomalies in the spinal tissues, predisposing factors for IVD and facet degeneration [14,15].

These brain changes reduce the diversity of motor behaviours and modify learning abilities. This reduction of proprioceptive feedback may also challenge the learning process of new skills by increasing uncertainty and increasing the threat related to exposition to sports gesture [16].

Conclusions

Back pain is common in exercise and sports. Certain type of exercise may cause aggression to IVD and other spinal tissues, particularly deteriorating proprioceptive afferences. There is evidence that a certain type of exercise can be anabolic, promoting IVD regeneration and muscle function. It is important to avoid nociceptive stimuli during the exercise considering that the pain experience will modify the sensitivity and motor control. Chronic or recurrent pain promotes cortical neuroplasticity, altering motor and behaviour control, motor and reducing the effectiveness of learning new skills.

This condition will also contribute to the maintenance of biomechanical deterioration of IVD, other spinal tissues and technical gesture.

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