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FASCIA SCIENCE AND CLINICAL APPLICATIONS: SELECTIVE REVIEW

Acute repetitive lumbar syndrome: A multi-component insight into the disorder

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Summary *Purpose:* Repetitive Lumbar Injury (RLI) is common in individuals engaged in long term performance of repetitive occupational/sports activities with the spine. The triggering source of the disorder, tissues involved in the failure and biomechanical, neuromuscular, and biological processes active in the initiation and development of the disorder, are not known. The purpose is, therefore, to test, using *in-vivo* feline model and healthy human subjects, the hypothesis that RLI due to prolonged exposure to repetitive lumbar flexion–extension is triggered by an acute inflammation in the viscoelastic tissues and is characterized by lingering residual creep, pronounced changes in neuromuscular control and transient changes in lumbar stability. This report, therefore, is a summary of a lengthy research program consisting of multiple projects.
Methods: A series of experimental data was obtained from *in-vivo* feline groups and normal humans subjected to prolonged cyclic lumbar flexion–extension at high and low loads, high and low velocities, few and many repetitions, as well as short and long in-between rest periods, while recording lumbar displacement and multifidi EMG. Neutrophil and cytokines expression analysis were performed on the dissected feline supraspinous ligaments before loading (control) and 7 h post-loading. A comprehensive, time based model was designed to represent the creep, motor control, tissue biology and stability derived from the experimental data.
Results: Prolonged cyclic loading induced creep in the spine, reduced muscular activity, triggered spasms and reduced stability followed, several hours later, by acute inflammation/tissue degradation, muscular hyperexcitability and hyperstability. Fast movement, high loads, many repetitions and short rest periods, triggered the full disorder, whereas low velocities, low loads, long rest and few repetitions, triggered only minor but statistically significant pro-inflammatory tissue degradation and significantly reduced stability.

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Conclusion: Viscoelastic tissue failure via inflammation is the source of RLI and is also the process which governs the mechanical and neuromuscular characteristic symptoms of the disorder. The experimental data validates the hypothesis and provides insights into the development of potential treatments and prevention.

Introduction

Repetitive Lumbar Injury (RLI), also known as Cumulative Trauma Disorder (CTD), is diagnosed with pain, weakness, limited range of motion and stiffness/spasms in the muscles associated with the respective joint. Common diagnostic procedures produce negative indications for deficits such as prolapsed disc, facet impingement, vertebral fracture, stenosis, etc., potentially placing RLI as a major contributor to the non-specific back pain category. The epidemiological evaluation suggested a strong relationship between repetitive occupational/sports activities over long periods and RLI. In specific, activities under high load magnitudes, large number of repetitions, and high rates of motion were identified as risk factors (Hoogendoorn et al., 2000; Marras, 2000; Orchard et al., 2009; Punnett and Wegeman, 2004; Silverstein et al., 1986; Smoljanovic et al., 2009). Biomechanical and physiological validation of the observational statistics (epidemiology) is missing, as well as the etiology of the development of RLI (e.g., what tissues failed, failure mode, neurological and biological pathways and interactions). Viscoelastic tissues' tolerance to load and the associated strain over many repetitions was suspected to be the source of the disorder, but experimental validation was not available (Kumar, 2001, 2008).

It is hypothesized that repetitive/cyclic anterior lumbar flexion strains the posterior viscoelastic tissues (ligaments, facet capsule, discs, fascia) and induces creep (McGill and Brown, 1992; Sanchez-Zuriaga et al., 2010; Solomonow et al., 1999, 2000) and micro-fractures in their collagen fibers (Woo et al., 1981, 1982, 1999; Fung et al., 2009) with parallel changes in reflexive neuromuscular function (Stubbs et al., 1998). An acute inflammation is triggered as the micro-damage exceeds a certain threshold (Leadbetter, 1990) simultaneously with spasms, reflexive hyperexcitability of the muscles (Williams et al., 2000) and changes in stability. Continuous exposure of the lumbar spine to repetitive activities can eventually convert the acute inflammation into a chronic inflammation, collagen fibers degeneration into fibrous tissue and permanent disability (Leadbetter, 1990).

Repeated bending activities in daily living appear to change both structure (ligaments, discs) and function (protective spinal reflexes)

If ignored, repeated bending is not just painful, it appears to trigger detrimental structural changes.

The objective of this paper is to provide a systematic integration of the various components active in the hypothesis (creep, neuromuscular control, stability, inflammation/tissue biology) obtained from a long series of experimental research projects in animal models (Williams et al., 2000; Eversull et al., 2001; Solomonow et al., 2003b, 2008, 2011; Claude et al., 2003; Hoops et al., 2007; Le et al., 2007; Lu et al., 2004, 2008; Navar et al., 2006; Youssef et al., 2008; Le et al., 2009; Ben-Masaud et al., 2009; King et al., 2009; D'Ambrosia et al., 2010; Pinski et al., 2010) and healthy humans (Solomonow et al., 2003a; Olson et al., 2004, 2006, 2009; Li et al., 2007) in order to provide a comprehensive model of the multi-factorial etiology of RLI, offer validating support for the acute phase of the hypothesis and allow the potential developments of science based methods for its treatment and prevention.

Methods

Animal experiments

In-vivo feline groups were used as the model for studying the various aspects of RLI and its relationship to load magnitudes, duration of loading, rest duration in-between loading periods, number of repetitions and movement velocity (or frequency) of the lumbar flexion–extension. Feline anaesthetized with chloralose is the classical model in neurophysiology of movement and was approved by the NIH-OLAR and the local IACUC. The preparations were supported by standard respiration, hydration and monitoring during the experiments to maintain proper body functions and metabolism. Exact details of the set up and loading are rather lengthy and provided in the separate publications on the various aspects of the loading (Williams et al., 2000; Eversull et al., 2001; Solomonow et al., 2001, 2003a, 2008, 2011; Claude et al., 2003; Hoops et al., 2007; Le et al., 2007; Lu et al., 2004, 2008; Navar et al., 2006; Youssef et al., 2008; Le et al., 2009; Ben-Masaud et al., 2009; King et al., 2009; D'Ambrosia et al., 2010; Pinski et al., 2010) and healthy humans (Solomonow et al., 2003b; Olson et al., 2004, 2006, 2009; Li et al., 2007).

In general, anesthetized preparations were set up in a fixture that allowed loading of the lumbar spine in flexion–extension within the physiological range under various load magnitudes, load durations, number of repetitions, intermittent rest periods and different loading rates/