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What is This?

The Complex Spine: The Multidimensional System of Causal Pathways for Low-Back Disorders

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Objective: The aim of this study was to examine the logic behind the knowledge of low-back problem causal pathways.

Background: Low-back pain and low-back disorders (LBDs) continue to represent the major musculoskeletal risk problem in the workplace, with the prevalence and costs of such disorders increasing over time. In recent years, there has been much criticism of the ability of ergonomics methods to control the risk of LBDs.

Method: Logical assessment of the systems logic associated with our understanding and prevention of LBDs.

Results: Current spine loading as well as spine tolerance research efforts are bringing the field to the point where there is a better systems understanding of the inextricable link between the musculoskeletal system and the cognitive system. Loading is influenced by both the physical environment factors as well as mental demands, whereas tolerances are defined by both physical tissue tolerance and biochemically based tissue sensitivities to pain. However, the logic used in many low-back risk assessment tools may be overly simplistic, given what is understood about causal pathways. Current tools typically assess only load or position in a very cursory manner.

Conclusion: Efforts must work toward satisfying both the physical environment and the cognitive environment for the worker if one is to reliably lower the risk of low-back problems.

Application: This systems representation of LBD development may serve as a guide to identify gaps in our understanding of LBDs.

Keywords: health and medical systems, biomechanics, anthropometry, work physiology, aging and individual differences, attentional processes

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INTRODUCTION

Low-back pain (LBP) and low-back disorders (LBDs) continue to represent an extremely challenging problem to workers around the globe. Recent U.S.-based findings indicate that up to 80% of the population will suffer LBP at some time during their life (Luo, Pietrobon, Liu, & Hey, 2004). As a nation, the United States alone spends more than \$90 billion per year treating back problems (Luo et al., 2004), about the same amount of money spent to treat cancer in the United States. These problems represent the second-most-common symptom-related reason for physician visits and the second-greatest cause of disability within America (Deyo & Weinstein, 2001; McGill et al., 2003). On a yearly basis, between 15% and 29% of Americans report back pain (Deyo & Weinstein, 2001), and LBDs result in more than 100 million lost workdays per year (Atlas, Wasiak, van den Ancker, Webster, & Pransky, 2004). All of these LBP and LBD issues create an unbalanced stress on the American health care system, with expenditures estimated to be 60% greater for those with LBPs compared with other illnesses (Deyo & Weinstein, 2001). Thus, the incidence and costs associated with LBPs represent reason for significant concern in modern society.

During the early part of this century, a comprehensive literature review by the National Research Council (NRC) and the Institute for Medicine (IOM) (2001) concluded that LBDs were indeed linked to work exposure, with between 11% and 80% of the occurrences of LBP being attributed to physical work factors. However, in more recent years, a series of reviews by Wai and colleagues (Wai, Roffey, Bishop, Kwon, & Dagenais, 2010a, 2010b, 2010c) and a series of papers by Roffey and associates (Roffey, Wai, Bishop, Kwon, & Dagenais, 2010a, 2010b, 2010c, 2010d, 2010e) have contended that there was no association between

work exposure and LBP. Still others claim that these reviews contained serious flaws in their methodology. For example, professor Stuart McGill (2011) has noted, "Another review of nonspecific back pain related to nonspecific activity guarantees a null finding" (p. 365). In fact, the NRC and IOM review noted earlier observed that the more specifically the nature of the work exposure is documented, the better the ability to observe an association with risk. Unfortunately for workers, critics of ergonomics have continued to challenge the ability of ergonomic interventions to control LBP and LBDs.

The alternative to ergonomic-based prevention of LBDs would be treatment after the LBD has occurred. However, treatment has proven less than effective. For example, surgical success rates for simple discectomies have been shown to be 42.6%, compared with 32.4% for nonoperative treatments (Weinstein et al., 2006). Another recent study notes a 629% increase in Medicare expenditures for epidural injections, a 423% increase in opioids, a 307% increase in magnetic resonance imaging, and a 200% increase in spinal fusion surgeries in recent years without any population-level improvements in LBP patient outcomes or disability rates (Deyo, Mirza, Turner, & Martin, 2009). These findings suggest a need for a better understanding of LBD causality for both prevention and treatment purposes.

These statistics beg the question, Do ergonomists understand LBD causality well enough to prevent LBD caused by work exposure? As a discipline, there is a plethora of assessment and interventions available that attempt to control LBD incidence. However, researchers have produced few validation papers that examine how well tools for low-back risk assessment match LBP reporting, and the few validation papers that do exist indicate a range in effectiveness of these tools (Marras, Fine, Ferguson, & Waters, 1999; Waters et al., 1999).

One can hypothesize that perhaps some of the ergonomic tools used for the assessment of LBDs are fundamentally missing some of the fidelity needed to understand and control the problem. Perhaps the tools are too simple and not sophisticated enough to do the job they are intended to do. After all, Einstein (2012)

suggested that people should "make everything simple as possible, but not simpler." Have we made things too simple?

To assess this contention, I will review the various mechanisms that the literature suggests can lead to LBD and consider how well the ergonomic assessment tools map onto these logic pathways.

THE PREMISE (ASSUMPTIONS)

LBDs and LBP are initiated by stimulation of pain-sensing tissues. For the purposes of this discussion (and there is good reason to believe), I assume that the source of this stimulation is always force or load. However, the relationship between the tissue load and the tissue tolerance can take many forms and can vary within and between individuals. It is imperative to understand these relationships if one is to understand LBD causality, risk, and intervention.

This premise suggests that all LBDs have a biomechanical basis in some way, shape, or form. However, biomechanical loading can be triggered by physical work factors, individual factors, or as psychosocial and organizational factors (NRC & IOM, 2001).

The relationship between tissue load and tissue tolerance defines the risk of experiencing an LBD. When work activities impose peak tissue loads that are below the tissue tolerance, the work is considered safe. When the tissue loading exceeds the tissue tolerance, then risk is present at least for some portion of the populations. However, one must be aware that both the imposed tissue load and tissue tolerance are both distributions. Hence, the degree to which these distributions overlap describes the risk of LBDs to the population at large.

TISSUE TOLERANCES

Traditionally, tissue tolerance has been viewed in terms of mechanical tolerance. Specifically, lumbar spine compression limits defined in terms of cadaver endplate microfracture limits have formed the "gold standard" for tissue tolerance in many earlier models (Chaffin, Andersson, & Martin, 2006; National Institute for Occupational Safety and Health, 1981). However, although many of the assessment tools consider 3,400 N as a lower limit for risk,

researchers have known for decades that the tolerance to endplate microfractures in cadaveric specimens spans a vast range of less than 3 KN to more than 8 KN (Jager & Luttmann, 1991, 1999). Furthermore, it is also known that cadaveric tissue is quite different from in vivo tissue in its failure characteristics. Although these findings may be the best available data for making tolerance limits, further research should be encouraged to better understand these limits in living people.

More recently, researchers have recognized that, for some work situations, risk to the lumbar spine may be driven by shear tolerance limits that are believed to be substantially lower than those for compression (Marras, 2008). Unfortunately, fewer studies have attempted to define the spine tolerance limit in both the anterior-posterior and lateral directions. To make matters worse, many of the assessment tools that are commonly used for work assessment are unable to accurately assess the shear forces imposed on the lumbar spine during work. Thus, there is a need to better define disc load tolerance in response to shear as well as tolerance in response to multidimensional (coupled) loading.

Some of the ergonomics tools have used muscle strength tolerance to define the limits for when workers are at risk of overexertion during manual materials handling tasks (Chaffin & Baker, 1970; Snook & Ciriello, 1991). However, these muscle strength limits do not document the yield strength of the muscle fibers. Instead, they are based on the amount of strength that one will voluntarily exert when asked. Here again, few studies have attempted to determine the validity of this tolerance threshold in its relationship to LBDs.

Although basic logic considers the tolerance limit to be more or less a constant, one knows in reality that these tolerance mechanical limits can change over time because of various factors, including age, tissue degeneration, cumulative loading, conditioning, genetics, and muscular compensation triggered by pain. Figure 1 illustrates a conceptual relationship among these factors. This figure indicates that some of these factors decrease tolerance over time, whereas others could increase tolerance

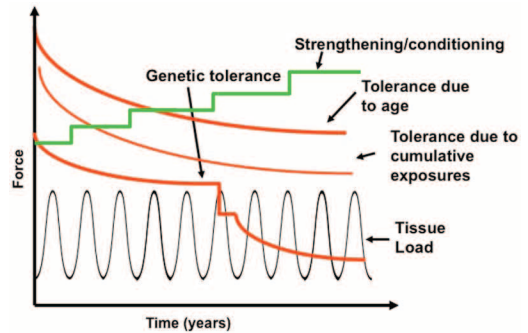


Figure 1. Conceptual relationship between various factors that influence material tolerance over time.

over time. It is important to remember that this figure represents general relationships. Although it is not known how much these factors vary between individuals, it is believed that every individual would have his or her own unique mix of these tolerances, yielding a personalized individual tolerance over time. Describing the time-dependent behavior of these material tolerances should represent an important avenue of exploration for future research endeavors.

The concept of assessing risk by way of a mechanical load versus mechanical tolerance has been an appealing concept, since it can account for many factors known to influence the risk of LBD. However, it is also known that a relatively small percentage of LBP reports are associated with tissue damage via magnetic resonance or computed radiography imaging (Deyo & Weinstein, 2001; Nachemson, 1985). It appears that people experience pain prior to observable tissue damage within the spine tissues. One can think of this relationship relative to time as a lowering of a pain tolerance threshold that occurs prior to a lowering of a tissue tolerance threshold (Figure 2).

If one follows the trail of pain from the spinal tissue to the brain, one knows that nociceptors are pain-sensitive cells that respond to mechanical stress as well as chemical insult. The spinal system contains many nociceptors. They are abundant in most tissues except for the nucleus, inner two thirds of the annulus, and some of the spinal ligaments. The spine's nociceptors have their own tolerance thresholds for activation.

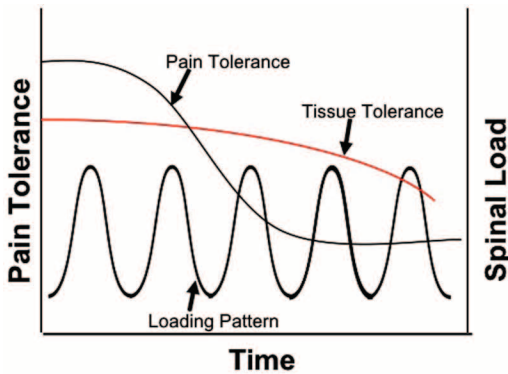


Figure 2. Conceptual relationship between pain tolerance and tissue mechanical tolerance over time (adapted from Marras, 2008).

However, this threshold can be lowered by an inflammatory response of the tissues. When a tissue becomes inflamed, the nociceptors are activated more easily (increased sensitivity), and thus, pain is perceived at a much lower level of force stimulus (Marras, 2008).

Biochemical reactions to tissue loading could provide a pathway for tissue inflammation and tissue sensitivity. It is known that a host of proinflammatory cytokines (interleukin-1, interleukin-8, interleukin-10, TNF α , etc.) when upregulated can enhance the inflammation and sensitivity of the tissue and, thus, initiate the pain pathway at lower stimulus levels. Recent studies have shown that cytokines followed by tissue damage can occur according to a dose-response pattern when one performs lifting tasks (Yang, Marras, & Best, 2011), thus suggesting the importance of ergonomic controls. In addition, there also appears to be a mental stress–personality interaction associated with cytokine response to spine loading (Splittstoesser, 2012). Hence, it appears that in addition to the individualized mechanical spine tissue tolerances, there are also individualized biochemical tissue tolerances that should be considered to truly appreciate the load tolerance nature of the LBP.

The upregulation of proinflammatory cytokines can increase the complexity of the risk picture via a systems response. Evidence indicates that even though one tissue is responsible for up-regulating a proinflammatory cytokine,

the biochemical response is systemic (Barr & Barbe, 2004). Proinflammatory mediators can be released into the bloodstream and increase the nociceptors sensitivity in tissues other than those responsible for the up-regulation of the proinflammatory mediator. For example, a muscle overexertion may increase the release of a proinflammatory cytokine that may, then, increase the sensitivity of the facet joint of a vertebrae.

Finally, one needs to realize that tolerance is a dynamic process. It has been well established that pain responses can be imprinted within the brain through a process (central set) similar to muscle memory. Even if the stimulus for LBP is no longer present, if the pattern of pain response has been ingrained into a portion of the brain, the sensation of pain can still occur (Marras, 2008). It is also known that pain chronicity can progress from peripheral (at the site of the force stimulus), to neuropathic (increased sensitization due to biochemical responses), to central (within the brain) (Marras, 2008). Hence, when a worker begins to experience back pain, it is important to break the pattern of pain reinforcement before the pain response becomes established centrally. Here again, this process can be individualized and this process may explain why one worker reports back pain caused by work whereas another worker with similar anthropometry and job responsibilities does not.

In summary, although researchers have a basic understanding of how mechanical tissue tolerance responds to work, the factors influencing pain perception tolerance are much less understood. A better understanding of how work and nonwork factors influence the mechanical and biochemical tolerance of the spinal tissues is needed.

TISSUE LOADING

Most ergonomic efforts have attempted to uncover how spine tissues are loaded during work. In the past 40 years, these efforts have evolved from static single-equivalent-muscle models (Chaffin, 1969), to static multiple-muscle models (Schultz & Andersson, 1981), to static biologically assisted models (McGill & Norman, 1986), to static stability models

TABLE 1: Characteristics of Various Spine Modeling Efforts That Can Be Used to Understand Precise Spine Loading

Spine Model Group	Location	Model Type	Static/Dynamic	Spine Geometry	Muscle Geometry	Muscle Function	Validation
McGill et al.	Lumbar/whole body	Rigid linked segment/stability	Dynamic	Literature generic	Literature data	Distributed EMG/optimization	Moment matching
Shirazi-Adl et al.	Lumbar/whole body	Rigid linked segment/stability	Static/quasistatic	CT simplified	Literature data	Optimization/inverse dynamics	Literature, EMG
van Dieen & Kingma	Lumbar	Rigid linked segment	Dynamic	Literature generic	Literature data	Distributed EMG/optimization	Moment matching, literature
Marras et al.	Lumbar/whole body	Hybrid FEM and rigid linked segment	Dynamic	CT/MRI subject specific	Subject-specific MRI	EMG	Moment matching/upright MRI

Note. EMG = electromyography; FEM = finite element method.

(Cholewicki & McGill, 1996; Granata & England, 2006), to dynamic biologically assisted models (Arjmand, Gagnon, Plamondon, Shirazi-Adl, & Lariviere, 2010; Gagnon, Arjmand, Plamondon, Shirazi-Adl, & Lariviere, 2011; Granata & Marras, 1993; Marras & Sommerich, 1991; McGill, 2004; van Dieen & Kingma, 2005).

With each advancing generation of models come better fidelity and increased precision. Most of the advanced models employ multiple muscle inputs and sophisticated modeling techniques in an attempt to develop person-specific assessments of spinal loading during work. There are four major groups around the globe that are leading the way in understanding spine loading. Table 1 summarizes the approaches of these various groups. As can be seen through this table, there are several efforts that are approaching the ability to build and use spine models that are specific to an individual worker.

As an example of such a model, Figure 3 shows an example of my group's biomechanical model of the spine. This model includes a specific individual's spine geometry, individual disc loads throughout the lumbar spine, actual muscle forces (derived via electromyogram), and ligament and bone contact forces. In this

example, a lumbar motion monitor (LMM) has also been included within the model in an attempt to reveal how changes in trunk kinematics (documented in field studies) influence the tissue loading throughout the spine.

Researchers have used these advanced models extensively to better understand the typical as well as atypical spine loading variability among the worker population associated with all types of work factors. Examples of how researchers have employed these models to better understand loading associated with the work factors of lifting (Arjmand, Plamondon, Shirazi-Adl, Lariviere, & Parnianpour, 2011; Marras, Knapik, & Ferguson, 2009), repetition and workday length (Marras et al., 2006), pushing (Knapik & Marras, 2009), and carrying (Schibye, Sogaard, Martinsen, & Klausen, 2001) are abundant in the literature.

COMMON RISK PATHWAYS

These high-fidelity models have also been employed to help researchers understand how nonphysical risk factors can contribute to LBDs. In several studies in the past decade, researchers have examined the manner in which psychosocial, psychological, personality, and physical load can interact to influence spine

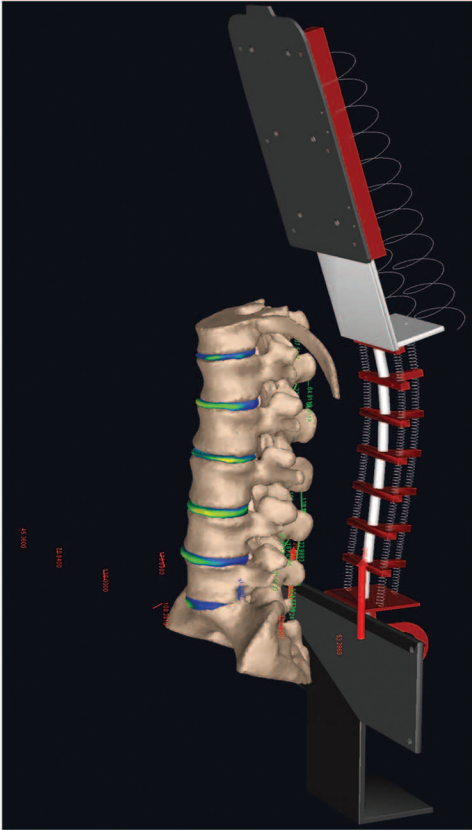


Figure 3. Personalized model of the lumbar spine used to assess work-related kinematics as measured by a lumbar motion monitor (LMM).

loading that would contribute to the risk of LBDs. Davis and colleagues (Davis, Marras, Heaney, Waters, & Gupta, 2002), as well as Marras and associates (Marras, Davis, Heaney, Maronitis, & Allread, 2000), have shown that when certain personality types are exposed to negative psychosocial environments or demanding mental tasks while performing lifting tasks, the spine tissue loads increase significantly. In addition, similar increased tissue loadings were observed when those suffering from LBP lifted loads, as compared with asymptomatic individuals (Marras, Ferguson, Burr, Davis, & Gupta, 2004; McGill et al., 2003).

It appears that the common pathway among these various risk factors involves an increase in antagonistic coactivity among the muscle

supporting (and loading) the spine. When certain personality types are exposed to stressful situations (relative to their personality type), these individuals respond by greatly increasing their antagonistic muscle activity compared with individuals with the opposite personality type. This increased coactivity dramatically increases the magnitude as well as the direction of applied force on the spine. In addition, a recent study by Splittstoesser (2012) has shown that certain personality types, when exposed to mentally stressful Stroop tests, will increase their levels of the inflammatory enhancing biochemical interleukin-8. Thus, considering both the tolerance and the loading collectively, one sees that the loading increases as proinflammatory biochemical responses cause the pain tolerance to be reduced, thus putting the person at an increased risk of LBP. To more accurately conceptualize this process, one could adjust Figure 2 to indicate both an increase in the tissue loads over time as well as a significant drop in pain tolerance over time. These examples suggest a systems response that needs to be considered when trying to design workplaces that minimize the risk of LBDs.

FRONTIERS FOR THE CONTROL OF LBD RELATED TO WORK

This discussion has implicated the highly interdependent and systemic nature of LBD and LBP risk. I have shown that the multidimensional nature of work factors, both physical and mental, can greatly influence the loading of the spine tissues. I have also shown that researchers need to deepen their understanding of how tolerance limits vary throughout the lifespan of the worker because of both controllable (exposure) and noncontrollable (genetics) factors. Furthermore, I have also shown how these loading and tolerance factors exist in a delicate balance and how something as subtle as a certain personality matched with a psychosocially challenging situation can lead to increased risk for certain individuals. It should be clear that this is a call for greater understanding of the spine system behavior and not a call for worker discrimination.

These recent findings suggest that perhaps researchers have oversimplified risk through

many of the ergonomics assessment techniques. Although many of these models may be appropriate for certain individuals, ergonomists need to work toward assessment tools that can identify the population characteristics so that the tools apply to more of the population variance. Doing so will increase the sensitivity and specificity of these tools.

For interventions to be effective in the prevention of LBDs, researchers need to explore mechanisms that have the potential to affect both the load and the tolerance side of the LBD risk equation. Ergonomists have known for years that by simply adjusting the physical workspace loading exposure, work-related low-back injuries and illnesses are often not fully controlled. In addition, there is abundant evidence that participatory ergonomics greatly increases the effectiveness of the intervention.

What might be the causal pathways underpinning the participatory ergonomics approach? One could argue that participatory “factors” contribute a cognitive balance to the intervention that includes emotional, intellectual, and social environmental features. Given the subtleties associated with muscle recruitment patterns of tissue loading and the biochemical responses associated with pain perception tolerance, it is reasonable to expect that many of these participatory ergonomics factors serve a function to preserve a state of balance (or wellness) within the individual worker. This balance would then serve to minimize the tissue load (by mediating muscle recruitment patterns) as well as optimize the tissue tolerance (via biochemical pathways) and the resulting risk of LBD for an individual.

In conclusion, the risk of LBD and LBP can be intrinsically tied to the behavior of the biomechanical load tolerance system described throughout this article. The examples discussed in this article demonstrate that the body is not composed of an independent musculoskeletal system and an independent mental system. These systems are inextricably integrated and interactive. Thus, to control tissue loading and tissue tolerance, one must not only control the physical world to which the worker is exposed, but one must consider the worker’s perception of his or her environment in a systems fashion

to mediate both his or her biomechanical and biochemical responses. This principle is the basis of wellness. Hence, one needs to consider the social, occupational, spiritual, physical, intellectual, emotional, financial, mental, and medical aspects of the environment if one is to truly minimize the risk of low-back problems.

Although the treatment of all aspects of these environmental factors is outside the scope of this article, practitioners must begin to integrate a wellness approach into ergonomics approaches as well as understand that many of the low-back risk assessment tools are giving only a crude estimate of both physical and system-related LBD risk. A wellness approach has the advantage of addressing the total human system as opposed to just one component of the human condition. Given the integrated biomechanical-biochemical-cognitive-psychosocial nature of people, such a wellness approach to ergonomics should have the best chance of improving low-back health consistent with the current understanding of causality.

KEY POINTS

- Low-back disorders continue to represent a major challenge and cost to society.
- A better understanding of the underlying causal mechanisms is needed to improve prevention.
- Causal pathways might be better represented by adjusting load-tolerance models of risk to incorporate changes in tolerance over time.
- Tolerances need to include consideration of changes in tissue material properties over time as well as increases in pain sensitivity (biochemical response) over time.
- Tissue loading models need to be able to explain individual differences through personalized models to provide a better understanding of risk.
- Given this evolving view of risk, worker wellness is expected to play a key role in managing spine tissue loads as well as physical and biochemical tolerance components of risk.

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