The case for cumulative trauma in low back disorders

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There has been significant debate in recent years over the existence of cumulative trauma as a source of low back pain. Some contend that all low back disorders are idiopathic and constitute a normal life experience. Others argue that some of these disorders are rooted in the exposure to risk factors that contribute to progressive “wear and tear” of the spine structures and tissues. There is no preponderance of direct evidence supporting one side of the argument or the other. Critics of the cumulative trauma argument claim that because no direct evidence exists to support the theory, low back pain must be a result of natural aging or must be genetically determined. Studies that directly test cumulative trauma hypotheses would require long-term, difficult and potentially unethical study designs to fit the traditional randomized control trial convention valued by the medical community. However, significant insight can be gained by examining the systematic pattern of evidence available from a large body of literature exploring the elements of work-related musculoskeletal disorders.

The pattern begins by noting trends in the epidemiologic literature that suggest cumulative trauma is a common element found in many reports of low back pain associated with the workplace [1–4]. This observational literature suggests that cumulative exposure might explain at least part of the low back pain picture. However, epidemiologic studies are limited in that they are often not specific enough in their measures to assess the precise dose–response trends associated with work. Most epidemiologic studies of work-related factors simply observe the presence or absence of a potential risk factor without noting the specific quantitative level of exposure, thereby making it difficult to understand at what level the risk factor begins to contribute to risk. In addition, it is difficult to identify interactions between risk factors unless the study is designed to be sensitive to these interactions. Hence, although the epidemiologic evidence can suggest which factors may be significant individually, it is unable to explain how these factors may interact with other potential risk factors. Nonetheless, these studies do suggest that excessive cumulative exposure to musculoskeletal loads can be considered a potential risk factor, because occupations in which there is cumulative exposure to repetitive biomechanical loading have been shown to increase risk.

A more compelling argument of the role of cumulative trauma can be built if the pattern of evidence includes biological plausibility to support epidemiologic finding. At the heart of the logic of cumulative trauma plausibility is the relationship between loads imposed on a structure and the tolerance of that structure. This concept is at the core of biomechanical analyses and suggests that when the loads experienced by a structure exceed the tolerance of a structure, damage occurs, whereas if the imposed load magnitude is below that of the structural tolerance, the loading is safe. In classical mechanical terms “damage” would indicate structural change, which has been demonstrated to occur in the spine for human and animal models as a result of cumulative trauma [5]. In biomedical terms this relationship could be expanded to tissue reactions that exceed a tolerance and can initiate a pain-producing sequence of events rather than traditional mechanical “damage.” Exceeding a pain-producing sequence tolerance can mean that the loads imposed on a structure are sufficient to initiate a pain process, such as stimulation of a nociceptor, release of a pro-inflammatory agent, reduction of blood supply to a tissue, rupture of a muscle, cellular changes or a host of other mechanisms that might lead to pain [6]. The point is that all of these mechanisms are initiated through biomechanical loading in excess of a tissue’s tolerance.

The tolerance of a tissue can be exceeded in two ways. Either the load can increase or the tolerance can decrease. The cumulative trauma concept assumes a time dimension. In other words, the tolerance level can change over time. Tolerances may change for a variety of reasons. We all accept the idea that age will decrease the tolerance of most biological materials. However, cumulative trauma and adaptation are also at work. Materials science has taught us that materials fatigue during repetitive loading and are subject to damage. For example, repeated bending of a piece of metal (eg, a paper clip) will change the tensile properties of the metal, the metal will heat up, become more brittle and...
eventually break at the point where the forces are concentrated in the structure. Similar processes are at work for biological materials. Hip joints wear down with excessive use over time, and athletes often complain of shin splints with excessive exercise. There is also evidence that cadaveric spines are compromised at low levels of force when exposed to repeated loading [5,7]. One significant difference between cadavers and living tissues is the ability of the living tissue to respond and adapt to loads. Wolff’s law states that exposure to loads make a tissue or structure stronger. However, adaptation has limits. Stress-strain relationships suggest that adaptation increases tissue strength up to a point, and beyond that point failure occurs. Body builders are well aware of these concepts. They build muscles by stressing the tissue and then allowing the muscle to rest for at least 24 hours. Adaptation ensues, and muscle bulk and bone mass increase. However, with some types of repetitive occupational tasks, significant rest may occur only to a limited degree between work shifts and on weekends only if overtime or recreational activities do not interfere with the rest. As is the case with work that consistently exceeds the adaptation process limit, the tissue tolerance could decrease quickly, making it more susceptible to injury. Therefore, cumulative trauma concepts are simply an extension of accepted concepts of how biological tissue functions.

The pattern of evidence associated with cumulative trauma must also consider the characteristics unique to the individual in response to mechanical loading. These characteristics may mediate the load-tolerance relationship over time. Age, conditioning, genetics, lifestyle habits, psychological state, personality and the current state of tissue degeneration can all influence the rate at which tissue tolerances change over time. These influences on the load-tolerance relationship have all been well documented in the literature [4] but also require further study before they are fully understood.

Workplace factors have been found to influence both the loading and tolerance of the tissues through cumulative loading or adaptation reactions. Factors include physical factors, psychosocial factors and organizational factors. Traditionally, it has been the physical workplace factors that have been explored and associated with increased tissue loading, particularly when the biomechanical characteristics of the work were properly and specifically addressed [4]. There continues to be controversy as to the contributions of psychosocial factors and organizational factors. Some have argued that increases in low back pain reporting can be explained through work dissatisfaction, organizational factors or the availability of compensation [8]. However, most studies have not considered the explanatory power of these factors relative to the load-tolerance relationship. Some of the classic psychosocial studies, when reexamined, have been found to explain a very small percentage of variability in low back pain reporting [9]. Analyses have shown that when biomechanical evaluations are considered along with psychosocial evaluations, the explanatory power associated with the psychosocial studies is greatly reduced [10]. More recent findings have shown that psychosocial factors have an interactive effect with biomechanical loading [11] and that individual factors, such as personality, can explain much of the variation in the magnitude of the loading forces experienced across individuals [12].

Collectively, this pattern of evidence suggests that no single factor fully explains the presence or absence of cumulative trauma and its association with low back pain. Traditionally, the literature has taken the approach of examining single risk factors in isolation in trying to explain back pain. Large bodies of literature exist that argue for the influence of each type of risk factor independently. However, if we consider the components of the system, it is likely that physical factors, individual characteristics, organizational factors, and psychosocial factors all influence the load-tolerance relationship that is at the core of cumulative trauma. The evidence suggests that cumulative exposure to loads when combined with other risk factors can contribute to low back disorders above and beyond the influence of aging or genetics alone. Therefore, instead of asking whether cumulative trauma exists, the question shifts to, how big a role can cumulative exposure to biomechanical loads play in the causation of low back disorders when considered in context along with the effects of the other risk factors?

The pattern of evidence suggests that we must consider how the system behaves in order to appreciate the influence of cumulative trauma in the etiology of low back pain. Instead of continuing to explore low back pain causality within the confines of specific disciplines (eg, biomechanics, psychosocial, physiology, genetics, and so forth), we must more fully explore the interactions between these disciplines as proposed in Fig. 1. The pattern of evidence suggests that the explanatory power inherent in the interaction between these disciplines may very well overpower the influence of any main effects. Thus, the answer to the aforementioned question depends on the strength of the interactions between the risk factors. The research community has already begun to quantify the degree of interaction between many of these risk factors, yet much more work is required.
before we fully understand these interactions. These interactions represent the current research “gaps” as well as opportunities for future research direction.

Therefore, when considering the pattern of evidence for cumulative loading and low back pain, all the components of plausibility are in place and a picture emerges that logically supports the influence of occupationally related cumulative exposure to loads as one pathway in the etiology of low back disorder.

References