Editorial

State-of-the-art research perspectives on musculoskeletal disorder causation and control: the need for an intergraded understanding of risk

In order to control musculoskeletal disorder (MSD) risk, it is important to understand how these disorders develop and manifest themselves in society. One could argue that without a thorough understanding of the root causes of a disorder we are only interpreting MSD symptoms in our efforts to reduce risk. However, when we understand how MSDs are “triggered” and understand their development, we have an opportunity to control the problem at the source and can potentially avoid the costly rehabilitative process.

Our knowledge of musculoskeletal disorder causation has progressed well over the years with in-depth understanding accelerating in the topic areas of epidemiology, the biomechanics of tissue loading, the biomechanics of tissue tolerance, physiological response to tissue loading, pain perception of the musculoskeletal system, individual and genetic influences on musculoskeletal disorder, psychosocial and organizational influences, and the role of primary and secondary interventions. However, MSDs are still the leading cause of occupational-related injuries. Hence, it appears we still have a long way to go before we have enough knowledge of MSD development so that we can eradicate these disorders from the workplace.

1. Need for definitions

One problem with MSD causality investigations is that we have been unable to adequately define many MSDs. Without operational definitions, the research surrounding these injuries and illnesses becomes nebulous. Systematic literature reviews can become problematic since different studies may be examining different aspects of MSDs. Much of this difficulty lies with the problem that many occupationally-related MSDs are simply labeled as functional somatic syndromes. These syndromes, such as repetitive stress injury and fibromyalgia have explicit and highly elaborated self-diagnoses, and their symptoms are often unresponsive to reassurance, explanation, and standard treatment of symptoms [1]. In addition, the climate surrounding many occupationally-related syndromes includes sensationalized media coverage, suspicion on the part of both the worker and employer, litigation, and a heavy reliance on simple causality explanations. In some studies, MSDs are defined as impairments and others as disabilities with no common definition that distinguishes between the two states. The challenge of many field-based studies is to control for these sources of potential bias so that they only consider medically relevant and quantifiable clinical end points. Unfortunately, it is obvious that some of these biases can be inherent to current end point definitions. Hence, there is a dire need to operationally define our occupationally-related MSD syndromes if the quality of field-based research is to grow beyond the current state of understanding.

2. Methodological considerations in reviewing the risk literature

Another major issue to consider when evaluating causality evidence and MSDs is the methodological issues associated with the data. While there are numerous methodological approaches to assessing scientific evidence, one must consider the benefits and limitations of the various approach criteria in order to appreciate the causality link. Criteria vary based upon the type of research investigated in that different methods apply to investigations of different aspects of MSDs.

Many of the methods for assessing evidence have been derived from epidemiologic studies. Three approaches to establishing a causal links have been employed to investigate the work relatedness of MSDs. The first, Hill’s Criteria, is common in epidemiologic research and has served as the basis for the other two approaches used to explore MSDs recently. Hill’s criteria include: 1) strength of association, 2) consistency between observational studies (replications), 3) specificity of effect from a factor, 4) temporality, 5) biological gradient or dose-response, 6) biological plausibility and coherence, and 7) experimental evidence and analogy with other known processes.

Second, the National Institute for Occupational Safety and Health (NIOSH) performed a review of the MSD
workplace causality evidence and modified these criteria [12]. They adopted Hill’s criteria 1–4 and added an exposure response relationship criteria as well as a coherence of evidence criteria.

Third, the National Research Council performed a literature review of MSD and workplace factors in 1999 [13]. They adopted the criteria of: 1) temporal ordering, 2) cause and effect covary, 3) temporal contiguity, 4) the size of the cause and effect are related, and 5) other plausible explanations for the observed effect are absent.

Two validity issues to consider in assessing causal relationships in MSD research involve internal and external validity [16]. Internal validity refers to the logical correctness of the conclusions. Examples of factors that can jeopardize internal validity include potential sources of confounding, problems with experimental control, uncontrolled variables, and experimental power issues. Specific issues to consider include: 1) use of random samples, 2) equating groups on all other factors, 3) loss of sample for follow-up, 4) compliance measured in each group, 5) other interventions, and 6) blinded effect measures.

External validity refers to the generalizability of the conclusions and is concerned with whether the effect can be generalized to populations, settings, or measures. Considerations of jeopardizing external validity include: 1) sample homogeneity, 2) adequacy of the control group, 3) relevant outcome measures, 4) follow-up period, and 5) generalizability to other similar groups and situations.

Collectively, the strength or quality of the evidence can be considered along a four level scale. Level 1 (strong evidence) is defined by multiple relevant, high quality, randomized clinical trials with consistent outcomes. Level 2 (moderate evidence) is characterized by one relevant, high quality randomized clinical trial in addition to one or more low quality randomized clinical trials or non-randomized controlled clinical trials with consistent outcomes. Level 3 (limited evidence) refers to one high quality randomized clinical trial or multiple low quality randomized clinical trials or non-randomized controlled clinical trials with consistent outcomes. Finally, level 4 (no evidence) is indicative of only one low quality randomized clinical trial or one non-randomized clinical trial, not relevant studies, or contradictory outcomes.

Intervention research also presents special challenges when considering causality. Several attempts to consider MSD interventions have been reported in the literature and at least one review [15] has suggested the following criteria for intervention assessment. First, the intervention was demonstrated useful through one or more scientifically acceptable controlled randomized trials. Second, the intervention was demonstrated useful through one or more scientifically acceptable non-randomized controlled studies (cohort or case-control studies). Third, the intervention is considered useful in current practice but without scientific proof of the effect. And, fourth, the intervention has not been demonstrated useful in the scientific literature and is not used in practice.

Most of the aforementioned criteria associated with strong causal inference are based upon a preponderance of scientific evidence. In the case of small focused biomedical issues that lend themselves to randomized controlled trials these criteria for causality interpretation are appropriate. However, in the case of work-related MSDs the situation is not so simple and these techniques are often unrealistic. For example, under real world conditions, when one introduces various interventions into the workplace, the “definitive” intervention study involving a randomized control experiment with double blinded conditions, is not possible. However, the inability to conduct such formalized studies in the workplace should not detract from the causality issue.

An alternative to viewing the preponderance of evidence has been successfully used by the National Research Council [14] to explore the MSD causality issue. In this effort, the scientific judgment was based upon piecing together evidence based upon a series of studies using various study designs. In this approach it was important to conceptualize the literature in a broader sense and understand how the pieces of evidence may fit together. In other words, this approach considers the pattern of evidence associated with the body of knowledge. This provides an opportunity to expand the breadth and depth of the evidentiary base. Given the complexity of MSDs, this appears to be a feasible approach to understanding MSD causality. The state-of-the-art research symposium exploring causality and MSDs has been constructed to examine this “pattern of evidence”. The papers that follow were designed to explore the evidence contributing to this pattern.

3. A comprehensive MDS causation model

Traditionally, the literature has investigated MSD causality via the traditional disciplines of biomechanics, psychology, psychophysics, psychosocial, physiology, genetics, organizational psychology, and rehabilitation. Each of these disciplines has studied MSD causality in isolation of other disciplines. The underlying assumption is that each discipline can fully explain causality and each discipline was treated as if it were mutually exclusive and exhaustive of the other disciplines. Hence, the body of knowledge has progressed along research “silos” where we have in-depth knowledge along given research tracks that are defined by the boundaries of the discipline. However, a wealth of knowledge has been amassed within each of these research silos. How can they all be correct if they are indeed mutually exclusive and exhaustive? The answer is: they can’t be.
Perhaps instead of observing MSDs through the myopic lens of each discipline, we need to begin to view MSD causality as a system so that we might appreciate the pattern of evidence that has emerged. Recently, the National Research Council [13,14] has suggested a conceptual model of how the different avenues of research may be interrelated. Fig. 1 suggests how the various potential risk factors associated with these various research disciplines may be interrelated.

The premise underlying this conceptual model is that any MSD must have a biologically plausible foundation. In this model, the biomechanical load-tolerance relationship represents the underpinning or root cause of a MSD event. McGill [11] proposed that injuries and disorders are initiated when a biomechanical load imposed on a tissue exceeds the tissue tolerance. This situation can occur when loads become excessive, as when heavy objects are lifted, or when the tolerance has decreased, such as occurs through aging or cumulative trauma.

The model represented in Fig. 1 also shows that the load-tolerance relationship might also explain the causal pathways associated with the various risk factors that have been reported in the various research disciplines or silos. For example, it is evident that physical work factors can influence the magnitude and nature of the loading occurring on the musculoskeletal system. However, similar processes might be at play with psychosocial factors and organizational factors. These factors could conceivably cause muscle recruitment patterns to change and increase loading on musculoskeletal tissues. In addition, tolerance may be affected by individual genetic and psychological factors as well as through previous loading history (CTD or adaptation). Each of these factors can either lower or raise the tolerance to loading and can, therefore, influence this load-tolerance relationship.

Fig. 1 also indicates how psychological factors may play a role in MSD causality. The diagram indicates that the load-tolerance relationship initiates a sequence of events relating to potential MSD pain symptom perception and reporting. This sequence of events indicates that reporting and perception can be influenced by a multitude of psychological and perceptual factors. However, these reports and perceptions are often the root source of information for some research disciplines (i.e. epidemiology). Hence, whereas, the field of biomechanics derives its findings from the actual load-tolerance relationship, observational disciplines such as epidemiology derive their findings from derivatives of this load-tolerance relationship (reporting) which might be influenced by personal factors, motivations, perceptions, and altered pain thresholds. Therefore, this conceptual model suggests that the different disciplines are really looking at the same injury causality process, but these different disciplines are simply observing different aspects of the process.

Conceptualizing the MSD causality process as indicated in Fig. 1 also suggests promising avenues for accelerating our understanding of causality. The conceptual model indicates that the load-tolerance relationship can be influenced by many factors. However, there is a void in the literature in that we do not know how many of the factors outside of the biomechanics silo influence the load-tolerance relationship. Hence, it is the contention of this treatise that the future of research into LBD causality lies in understanding the interactions between biomechanics and these other research silos.

4. The significance of interactions in exploring causality

Recently, a few studies have begun to explore how the interactions between these research silos might impact MSD risk. One example, offered to show how this interaction might occur, involves a recent investigation to describe how the body responds mechanically, to diverse non-physical influences [10]. The psychosocial literature had reported for years that psychosocial influences were an important factor for LBD risk [2–5,7–9,17]. However, the causal mechanism was poorly understood. Some speculated that poor psychosocial environments would create an environment where workers were more likely to report injury and illness. However, studies in our laboratory have been able to demonstrate a complex biomechanical pathway through which psychosocial risk factors act. Our study [10] asked subjects to perform standard lifting tasks under psychosocially stressed and unstressed conditions. In this study, psychosocial stress was defined as the interaction of the experimental subject with the experimenter. Under the unstressed conditions, the experimenters were friendly and interactive with the subjects during the lifting exercises. During the stressed conditions, the experimenters...
were terse and appeared agitated. Even though the same exact physical exertions were performed under both conditions, the stressed conditions resulted in greater spine loadings (26–70%) which were traced to greater trunk muscle coactivity. Hence a direct interaction between psychosocial stress and biomechanical response of the musculoskeletal system was identified that might explain why psychosocial factors increase MSD risk.

Further analysis of the data revealed that individuals responded in dramatically different fashions in response to the stress conditions. Individual responses were tied to individual personality characteristics that interacted strongly with psychosocial stress. In particular, those subjects who were classified as introverts via the Myers–Briggs personality inventory experienced a 14% increase in spine compression and a 27% increase in lateral spine shear under the stressed conditions compared with their extravert counterparts who experienced far less increases in spinal load (4–6%) under the stressed condition. Similarly the “intuitive” personally trait was associated with much greater spine loading (10–25%) responses under the stressed conditions compared to negligible increases in spine loading experienced by the “sensor” counterparts. Hence, this study has shown that interactions between both individual factors (personality traits) and psychosocial stress are able to explain much of the variability in subject response when performing physically demanding tasks.

In a similar manner, a more recent study has been able to show how the degree of mental processing and pacing required during a physical task interact to strongly influence biomechanical loading of the spine [6]. In this study subjects were asked to perform lifting tasks under fast and slow pacing conditions while the mental processing requirements were altered. Mental processing consisted of high or low decision making tasks as well as varying levels of object placement complexity. Pacing by itself did not have much of an influence on spinal loading. However, when task complexity was great and high levels of mental processing were required, these factors interacted with pacing to increase spinal loading by 25–65%. Here again, the mechanism of loading was traced to significant increases in muscle coactivation.

5. Conclusions

These studies demonstrate that a rich and logical pattern of evidence can be established for MSD causation if researchers are willing to reach beyond the traditional boundaries in each field of study. Most scientific advances are occurring at the intersection of scientific disciplines and interdisciplinary investigations have enriched our body of knowledge. The field of MSD causality is no different. We expect that significant “field moving” leaps in knowledge will occur as we investigate the interactions between the various traditional disciplines that have explored MSDs.

The following papers represent an attempt to present the diverse perspectives relative to MSD causality within the various disciplines represented in the conceptual model shown in Fig. 1. These papers are a result of a “State-of-the-Art Research Symposium: Perspectives on Musculoskeletal Disorder Causation and Control” held in Columbus, Ohio on May 21 and 22, 2003. It is our hope that by exposing researchers to the various scientific perspectives associated with the variety of disciplines believed to impact MSDs, interactions between the traditional scientific disciplines will be enhanced.

W.S. Marras,
Biodynamics Laboratory, The Ohio State University,
Columbus, Ohio, USA


References


William S. Marras holds the Honda Endowed Chair in Transportation in the Department of Industrial, Welding and Systems Engineering at the Ohio State University. He is also the director of the Biodynamics Laboratory and holds joint appointments in the Departments of Physical Medicine, and Biomedical Engineering. Professor Marras is also the co-director of the Ohio State University Institute for Ergonomics. Dr. Marras received his Ph.D. in Bioengineering and Ergonomics from Wayne State University in Detroit, Michigan. His research centers around industrial biomechanics issues. Specifically, his research includes workplace biomechanical epidemiologic studies, laboratory biomechanics studies, mathematical modeling, and clinical studies of the back and wrist. His findings have been published in over 150 refereed journal articles and numerous book chapters. He also holds several patents including one for the lumbar motion monitor (LMM). Professor Marras has been selected by the National Academy of Sciences to serve on several committees investigating causality and musculoskeletal disorder. His work has also attracted national as well as international recognition. He is a two time winner (1993 and 2002) of the prestigious Swedish Volvo Award for Low Back Pain Research as well as Austria’s Vienna Award for Physical Medicine and recently won the Liberty Mutual Prize for Injury Prevention Research.