[EDITORIAL]

When It Comes to Editor-in-Chief Dr Guy G. Simoneau, Thanks Isn't Enough—It's More About Inspiration

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Sometimes in our profession, you get really, really lucky and find a person whose intellect, work ethic, and unselfish commitment to helping others truly reinforce all that is good about physical therapy. *Leadership* and *vision* are terms that get thrown around a lot these days, but when we think of *Journal of Orthopaedic & Sports Physical Therapy* (*JOSPT*) Editor-in-Chief (EIC) Guy G. Simoneau, PT, PhD, FAPTA, ***inspiration*** is probably the best descriptor. Inspiration to perform solid research. Inspiration to mentor young, emerging clinicians and researchers. Inspiration to advance the field and be the best therapist you can be.

After 15.5 years of guiding the *JOSPT* through its amazing progress, Dr Simoneau is stepping down as EIC, handing editorial leadership of the *Journal* to the very talented and able Clare L. Ardern, PT, PhD,[3] beginning with the July 2019 issue. In this editorial, we, the JOSPT/Movement Science Media Board of Directors, summarize some of Dr Simoneau's many achievements and humbly express our gratitude and that of the Editorial Board and publishing team for his overwhelming devotion to the *JOSPT* for nearly 2 decades.

To gain some perspective on Dr Simoneau's impact on advancing the field of orthopaedic and sports physical therapy, it is helpful to go back 40 years to the origins of the *JOSPT*. In 1978, there was almost no evidence base to support physical therapy interventions for people with orthopaedic and sports-related conditions. Customary spine care was often the modality-based "4 to the back." Following anterior cruciate ligament reconstruction, patients were typically immobilized for 8 weeks, and many people believed that weightlifting was bad for you. The term *evidence-based practice* had not yet been proposed,[5] and there were not many opportunities for physical therapists to obtain high-quality information. A few advanced masters programs for physical therapy were emerging, but there were almost no PhD opportunities. The idea of an entry-level professional doctorate was considered naïve and irresponsible.

In 1979, a great leap forward occurred when 2 physical therapists from Wisconsin, George J. Davies and James A. Gould, created the *Journal of Orthopaedic & Sports Physical Therapy* to be a forum for communication based on dissemination of "the knowledgeable application of the most recent advances in medical research and clinical treatment to facilitate and improve health care delivery to

Dr Guy Simoneau.

the patient with a musculoskeletal condition."[4,8] The fundamental mission of the *JOSPT* was "to further the understanding of basic sciences as applied to musculoskeletal conditions and to promote justification of clinical procedures in orthopaedics and sports medicine," that is, to start to build the evidence base for orthopaedic and sports physical therapy and to make this information available to practicing physical therapists, physicians, and other health care providers.

Over the next 23 years, under the guidance of 4 editors, *JOSPT* advanced from a quarterly to a monthly publication and continued to gain respect. By 2002, the *JOSPT* was established as the "go-to" journal of its founding American Physical Therapy Association bodies, the Orthopaedic and Sports Physical Therapy Sections, which then consisted of approximately 17 500 members. It was encouraging that the *JOSPT* also served about 1800 institutional and individual subscribers from about 40 countries. However, most of the research the *Journal* published came from the United States, and the *JOSPT* had no international partners or meaningful presence on the worldwide web.

In 2002, Dr Guy G. Simoneau was a very promising young researcher. He had graduated from the University of Montreal in 1982 with a degree in physical therapy, and in 1984 was awarded a masters degree in physical education with a sports medicine emphasis, from the University of Illinois at Urbana-Champaign while also working toward his athletic training certification. After several years of clinical practice and teaching at Russell Sage College in Troy, NY, he became a graduate research assistant at The Pennsylvania State University, where he developed his skills by coordinating running injury and orthopaedic patient clinics and conducting research on fall prevention among elderly patients with lower extremity diabetic neuropathies. In 1992, he received his PhD at Penn State after successfully defending his dissertation, titled "The Effects of Diabetic Distal Symmetrical Peripheral Neuropathy on Static Posture."

Shortly afterward, Dr Simoneau joined the physical therapy faculty at Marquette University in Milwaukee, WI. At that time, Dr Simoneau had a great opportunity to focus on his own line of research at an outstanding university. Here, however, is the part that really shows Guy's character and high personal integrity: rather than pursue his own recognition, he accepted the position as EIC of the *JOSPT* and dramatically reduced his own research so that he could unselfishly mentor and facilitate the achievements of others. Importantly, while some journal editors place themselves at the front and bask in recognition, Guy's philosophy was based on a willingness to let others have the limelight and credit. This philosophy worked. Over the next 17 years, countless authors, editorial board members, and clinicians succeeded and were empowered and grew as professionals, while Guy tirelessly provided encouragement and constructive feedback.

So here is where the *Journal* is so many years later under Guy's leadership. After being conceived around a dinner table in La Crosse, WI 40 years ago, the *JOSPT*'s current impact factor—a record 3.090, with a 5-year factor of 4.061—ranks it as one of the world's leading musculoskeletal and sports-related journals. It is now the international go-to resource for orthopaedic and sports physical therapy. Currently, more than half of the papers published in the *JOSPT* come from countries other than the United States, while its content is delivered monthly in print and continuously online to 23 000-plus American Physical Therapy Association Orthopaedic and Sports Physical Therapy Academy members, as well as more than 11 500 institutional and additional individual subscribers located in the United States and around the world. Thirty-five international organizations representing 25 countries have partnered with JOSPT to provide access to JOSPT content to their individual members. JOSPT's highly acclaimed website[7] supports more than 155 000 sessions each month from nearly 91500 unique users, who come to www.jospt.org from approximately 177 countries. It is hard to imagine this success without the diligent, enthusiastic, and unselfish work of Dr Simoneau.

Think of the extraordinary changes in health care research that Dr Simoneau has so successfully navigated in his 15.5 years as *JOSPT*'s EIC. The astonishing development of web-based data sets and knowledge transfer, huge advances in statistical modeling and data appraisal, dramatic changes in care delivery, and the proliferation of competing journals—some of which rely upon predatory business models[2]—have created challenges, but also great possibilities, that Dr Simoneau and *JOSPT*'s outstanding editorial and publishing teams have turned into opportunities leading to achievement.

Our field has had sensational and unparalleled knowledge growth in the last 17 years that has been spearheaded, disseminated, and appraised in the more than 1300 original research papers published in the *JOSPT* during that time. In addition to the continued publication of highly relevant clinical trials, many, many other impactful studies have been distributed to relevant audiences. These studies include advances in meta-research leading to clinical practice guidelines,[1] and innovative and important work regarding pain science, imaging, biomechanics, and telerehabilitation, all of which have been facilitated and refined by Guy's leadership. The result is a major contribution to the robust body of evidence that has formed the basis for today's physical therapist practicing in a direct-access environment.

During Guy's tenure as EIC, so many of us have been privileged to work with him. He has never given less than 100% to the *JOSPT*, nor has he ever compromised the *Journal*'s integrity in any editorial decision. He has always been available to any author and always provided mentorship through difficult manuscript review and editing processes. His work has always been done unselfishly, with dignity and professionalism. It has always been inspirational. A quote from the ancient Chinese philosopher, Lao-Tzu, sums up Guy's quiet mentorship most succinctly: "A leader is best when people barely know he exists … when his work is done, his aim fulfilled, they will all say: we did it ourselves."[6]

In recognition of all that he has done for the *JOSPT*, the Board of Directors is pleased to announce its plan to honor Dr Simoneau's devoted service in 2 important ways: first, to acknowledge him as Editor-in-Chief Emeritus on the *JOSPT* masthead from the July 2019 issue on, and second, to rename the JOSPT Excellence in Research Award, an award he established 16 years ago, the "Guy G. Simoneau–JOSPT Excellence in Research Award."

"Thank you" will never be enough; Guy's inspiration will last a lifetime, and on behalf of all involved in *JOSPT*, we intend with these honors to ensure that.

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[RESEARCH REPORT]

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Model Simulations Challenge Reductionist Research Approaches to Studying Chronic Low Back Pain

Low back pain (LBP) is a multifactorial problem associated with many biological, psychological, and social factors.[8,21,28,30] In most cases, the exact causes underlying LBP are unknown; hence, the term *nonspecific LBP* is often used. This nonspecificity makes selecting the appropriate treatment challenging for clinicians. Therefore, much of the current research efforts are directed toward identifying specific causal factors underlying the clinical presentation of LBP or toward subclassifying patients with specific characteristics (a collection of factors that determine the nature of an individual's LBP) to formulate the appropriate intervention strategies addressing these specific factors (hereafter referred to as "factors contributing to LBP"). This approach is based on the rationale that when more is known about the etiology of LBP, the treatment can be more specific in addressing the factors contributing to LBP and result in better outcomes. Subsequently, randomized clinical trials (RCTs) are conducted to evaluate whether such matching between factors contributing to LBP and treatment leads to improved outcomes compared to other treatments, standard care, or sham treatment.

BACKGROUND: Traditionally, low back pain (LBP) is studied using a reductionist approach, in which the factors contributing to the clinical presentation of LBP are studied in isolation to identify the primary pathology or condition linked to LBP. We argue that reductionism may not be suitable for studying LBP, considering the complex, multifactorial nature of this condition.

OBJECTIVES: To quantify the likelihood of successfully subclassifying patients with LBP and effectively targeting treatment based on a single dominant factor contributing to LBP.

METHODS: Both analytical and numerical simulations (Monte Carlo) of 1 million patients with LBP were performed. Several factors contributing to LBP were randomly assigned to each individual. The following outcome measures were computed, as a function of the number of factors: the percentage of individuals who could be subclassified by identifying a single factor exceeding a certain threshold, and the average reduction in LBP when treatment eliminates the largest contributing factor versus a multimodal treatment that eliminates a number of the randomly selected factors.

RESULTS: With an increasing number of factors, the probability of subclassifying an individual to a subgroup based on a single factor tends toward zero. A multimodal treatment arbitrarily addressing any 2 or more factors was more effective than diagnosing and treating a single factor that maximally contributed to LBP.

CONCLUSION: Results suggest that reductionism is not appropriate for subclassifying patients with LBP or for targeting treatment. The use of reductionist approaches may explain some of the challenges when creating LBP classification systems and designing effective treatment interventions. *J Orthop Sports Phys Ther 2019;49(6):477-481. Epub 15 May 2019. doi:10.2519/jospt.2019.8791*

KEY WORDS: *classification, Monte Carlo simulation, randomized clinical trials, risk factors, subgrouping*

The above-outlined strategy in LBP research is termed a *reductionist* approach in the parlance of systems science.[1] In the reductionist approach, the system is broken down into smaller parts to isolate and study them comprehensively. The reductionist approach is well suited for containable diseases, such as local infection. However, reductionism is less helpful when the problem is multifactorial and where interactions between biological subsystems exist.[1] These features make the behavior of a complex system difficult to predict, even when the behavior of its parts is well defined.[1] For example, studying motor control in patients with LBP is a reductionist approach that evaluates the pathomechanics of neuromuscular control in isolation from other biologi-

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cal, psychological, and social factors to identify the primary pathology or condition linked to LBP. A natural extension of this approach is the development of intervention strategies attempting to correct those pathomechanics.[27]

Reductionism is not inherently wrong, as it allows for the identification of parts of the system (eg, factors associated with LBP) and has been useful for establishing factors associated with patient presentations (phenotypes), an important part of patient care. The problem lies in the assumption that information about individual parts is sufficient to explain the behavior of the entire system. In the example of studying motor control using a reductionist approach, the assumption is that other biological, psychological, and social factors have minimal or no influence on motor control. Perhaps in some patients this may be the case, but the evidence suggests that motor control interventions are not superior to other interventions in the management of patients with LBP,[27] which raises questions about other factors and interactions involved.

In contrast to reductionism, a systems approach takes the entire system into consideration when describing its behavior and identifying interdependence between its subsystems.[1] Attempts toward such an approach have been made with conceptual, structural equation, or collaborative modeling to account for a number of factors contributing to LBP simultaneously.[5,6,9,18,25] Yet, research in LBP lags substantially behind systems biology, which rapidly progressed in recent years with its effective application of systems science.[4,14] There is a critical lack of knowledge regarding the number of factors and their interactions needed to adequately represent LBP, which in turn, limits the ability to target them through treatment modalities. As spine research evolves, the trend points toward more complexity, with more subsystems and their interactions requiring consideration.[6,11]

There have been more than 1000 RCTs published evaluating various interventions for LBP, such as manual therapy, massage, acupuncture, dry needling, physical therapy, and specific exercise.[15] Unfortunately, this literature collectively shows low to moderate effects and practically no differences between various interventions.[3] More importantly, to date, "no classification system is supported by sufficient evidence to recommend implementation into clinical practice."[2,7,13] Even a triage based on various clinical prediction rules has not led to better outcomes.[13] One possible explanation for the lack of success in documenting large positive treatment outcomes could be the reductionist approach, typically applied in LBP research, whereby unimodal intervention strategies targeting the dominant factor believed to be contributing to LBP are compared and studied in RCTs. While this approach has its place in research, considering the extreme biological complexity of the spine system, the multifactorial nature of LBP, and interactions among these factors,[21,30] an approach that addresses these issues simultaneously is needed to advance LBP research and the development of more effective intervention strategies.

The goal of this study was to highlight the challenges of studying a complex condition using reductionist approaches. Specifically, using analytical and numerical simulations, we quantified the likelihood of correctly identifying the dominant factor contributing to LBP and of effectively treating LBP by modifying such a dominant factor. The following 2 hypotheses were tested: (1) when dealing with a large number of factors contributing to LBP, it is not possible to identify subgroups effectively based on the dominant factor; and (2) on a population scale, providing a number of treatments targeting any 2 or more factors is more effective than identifying and treating a single factor that maximally contributes to LBP. If these hypotheses are true, perhaps a different research method, based on a systems approach,[1] could lead to the development of more effective intervention strategies for LBP.

METHODS

We performed both analytical and numerical simulations (Monte Carlo) of a large population (n = 1 million) with LBP. Factors contributing to LBP for each individual were uniformly distributed random variates (U_1, U_2, U_3, ... U_k) between 0 and 1. For each individual, each factor U_i was normalized by dividing it by the sum of k factors to create a fraction contribution to LBP; ie, the total pain/disability effect of 1 is: $(X_1 + X_2 + ... + X_k) = 1$ (**FIGURE 1**). For example, for 3 factors (k = 3), a person with LBP may have normalized factors such as $X_1 = 0.3$, $X_2 = 0.1$, and $X_3 = 0.6$. This means that factor X_1 contributes 30%, factor X_2 contributes 10%, and factor X_3 contributes 60% to the overall presentation of LBP, totaling 100%. To test the 2 hypotheses, we calculated (1) the percentage of individuals who could be subclassified by identifying a single normalized factor (X_i) exceeding a certain threshold θ (where $\theta = 0.2$, 0.3, 0.4), and (2) the average reduction in pain/disability when the largest factor contributing to LBP is identified and eliminated with the targeted unimodal treatment, versus a number of treatments (multimodal treatment) eliminating a number of randomly selected factors.

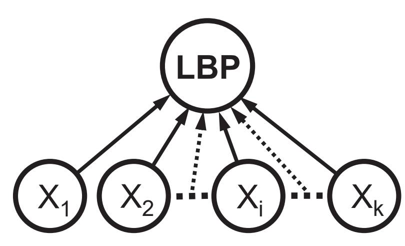

FIGURE 1. A schematic of the multifactorial, uniformly distributed model of LBP used in this study. All factors contributing to the clinical presentation of LBP were independent. Note that the sum of all factors (X_i) contributing to LBP is equal to 1 in every case simulated. Abbreviation: LBP, low back pain.

The analytical derivation and calculation of the hypothesized values are presented in the **APPENDIX** (available at www.jospt.org). To validate these analytical calculations, a numerical model simulation (Monte Carlo) was performed twice (macro feature in Excel 2010; Microsoft Corporation, Redmond, WA) by seeding an array with 1 million random variables between 0 and 1 and calculating the values derived analytically.

RESULTS

The maximum differences between any analytically derived values and the 2 simulation results were 5.28×10^{-4} and 4.75×10^{-4}. These small differences indicate excellent agreement between the 2 methods, validating the analytical approach.

With an increasing number of factors, the probability of a single factor exceeding a certain threshold ($X_i>\theta$) tends toward zero (**FIGURE 2**). In our model, this result represents the diminishing likelihood of classifying an individual to a subgroup of patients with LBP based on a single factor reaching some set threshold of contribution to the overall LBP (**FIGURE 2**). Even with a low threshold of $\theta = 0.2$ (accounting for 20% of LBP symptoms), less than 1% of the LBP population can be subclassified when the number of factors exceeds 11.

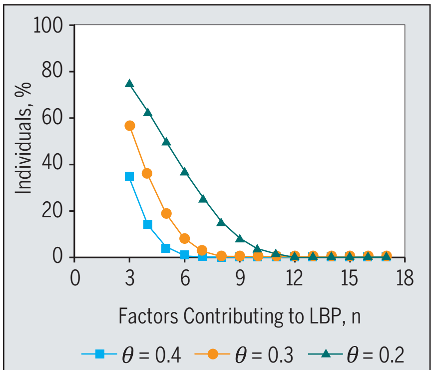

FIGURE 2. Percentage of individuals with LBP who can be subclassified, based on a single factor exceeding a certain threshold θ. Abbreviation: LBP, low back pain.

On average, in a multifactorial model, the sum of any 2 or more factors is greater than the largest factor identified in each individual (**FIGURE 3**). This simulation result illustrates that a multimodal intervention addressing any 2 or more factors will likely be more effective in the population of patients with LBP than diagnosing and treating a single dominant factor that maximally contributes to LBP in each individual.

DISCUSSION

The results from our analytical and numerical simulations of a multifactorial presentation of LBP are consistent with the data reported in the literature. With respect to the first hypothesis, our results show that with an increasing number of factors contributing to LBP, there is a diminishing likelihood of classifying an individual to a subgroup of patients based on the dominant factor. This could explain why attempts to identify subgroups of patients who would respond more favorably to a particular treatment have not yet been successful or reproduced.[2,7,13,22,23] Our simulations suggest that such a result would be expected if LBP were a large multifactorial problem. Reductionist research approaches, focusing at most on a few dominant factors contributing to LBP, are not able to address the entire complexity of this condition or document meaningful impact of interventions targeting those dominant factors. This scenario can be further complicated if many different mechanisms and factors interact and overlap, rendering the presence of pure subgroups rare.[16]

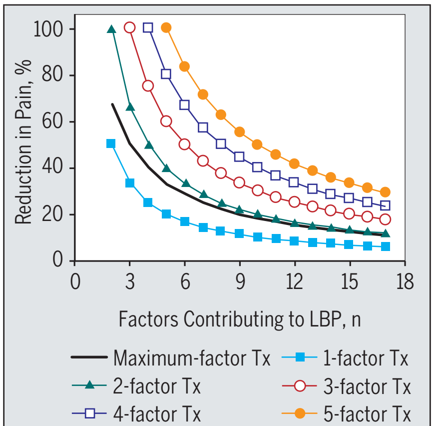

FIGURE 3. The average reduction in LBP when a given unimodal treatment eliminates the single largest contributing factor to LBP (maximum-factor Tx; solid black line), compared to a multimodal treatment eliminating a number of arbitrarily selected factors (1-factor Tx, 2-factor Tx, etc; lines with symbols). These results are plotted for different scenarios (number of total factors contributing to LBP) on the horizontal axis. Note that 100% reduction in LBP is expected when the number of factors targeted by a treatment is the same as the total number of factors contributing to LBP. Abbreviations: LBP, low back pain; Tx, treatment.

Based on the number of existing baseline predictors and the variance in outcomes they explain, Mistry et al[19] concluded that it is unlikely we can identify a single strong moderator of LBP treatment effects. None of the RCTs they reviewed were powered sufficiently to identify differential subgroup effects, and appropriately powered studies would be practically unrealistic.[19] To circumvent this problem, Patel et al[23] pooled data from 19 back pain trials that provided a data set of 9328 patients. Yet they, too, did not find any subgroups that would benefit from specific treatment, and, more importantly, they calculated that such an approach to identifying patients would not be cost-effective.

Our simulations are consistent with such findings. With only 12 factors contributing to LBP, only 0.5% of the LBP population could be subclassified based on a single factor and treated to achieve a minimal clinically important difference of a 20% reduction in pain[20] ($\theta = 0.2$) (**FIGURE 2**). What if there were 21 factors,[24] 69 factors,[8] or more[6]? Our simulation results indicate that this percentage would be 10^{-6} and 10^{-36} for 21 and 69 factors, respectively. Even if such an RCT could be conducted, it would likely have little clinical relevance.

With respect to the second hypothesis, it appears that multidisciplinary (ie, multimodal) rehabilitation strategies consistently show better results when compared to any single approach.[12] Likewise, our simulations suggest that when dealing with a multifactorial problem, it is more effective to treat several factors than to try to diagnose and treat the single dominant factor that contributes the most to LBP in each individual. Perhaps future research efforts should focus on designing effective multimodal, integrative, and adaptive approaches to the management of LBP.[17] As the management of patients with LBP continues to progress toward personalized medicine, multimodal treatment sequence, timing, and interaction effects will need to be considered.

There are, however, instances in the literature where the combination of 2 treatment approaches (eg, physical therapy and cognitive behavioral therapy[29]) was not superior to a unimodal treatment (physical therapy). One possibility in this example is that a single treatment modality (physical therapy) affected several factors contributing to LBP,[31] including those targeted by the cognitive behavioral therapy. In our model, such a situation could be simulated by a comparison of a single treatment targeting several factors contributing to LBP versus the same number of unimodal treatments targeting a single factor. Both interventions would show the same effectiveness in such a comparison. Alternatively, in the above example, the psychological factors targeted by cognitive behavioral therapy might not have been important factors contributing to LBP in these patients.

Several assumptions determine the behavior of this model simulation. The assumption having probably the biggest effect on the results was that various factors contributing to LBP are uniformly distributed across the population with LBP. That is, all factors have the same probability of being present in each individual, and there is no factor occurring more frequently in the LBP population. If some factors were occurring more frequently, it would have been easier to identify a cluster of patients with these factors. We submit, however, that in reality the distribution of factors contributing to LBP might be closer to uniform, because the studies thus far have failed to identify a dominant modifiable factor or subgroup of patients with LBP.[13] The simulated treatments were unrealistic because they completely eliminated the targeted factors contributing to LBP in every case. Most likely, the real treatment effects would have been much smaller, because interventions for LBP are not 100% effective, and not all individuals respond to them.

Another assumption that impacted the model results was that the model was unstructured (**FIGURE 1**). Such a model assumes that each factor is independent and directly linked to LBP, which is unlikely to be the case. However, we purposefully chose such a model, given that it represents the common factorial analyses used in LBP research. The addition of interactions between the factors, which could represent serial, parallel, and feedback connections, would make the model more complex, strengthening the argument that reductionist approaches are not appropriate to study the complex phenomena represented by such a model.

CONCLUSION

Research to identify the factors, or group of factors, that contribute to LBP and to understand the efficacy of individual treatment interventions is necessary but not sufficient to address the LBP problem effectively. As demonstrated by our unstructured multifactorial model of LBP, simply identifying components within the model and not the structure of the model (ie, the interactions between these components) is not likely to lead to robust classification or better treatment effects.

To advance LBP research, more sophisticated modeling methods that consider the structure of the system being studied[9,18] and possibly the dynamics of the system[1] (LBP symptoms and treatment effects are not static and change with time) are needed. Future research should involve a paradigm shift toward a systems approach, which allows for integration of knowledge in a more systematic and effective way.[26] A systems approach has been specifically developed to address complexity and successfully implemented in engineering. Such an approach appears to be well suited for studying medical conditions that are multifactorial in nature.[1] ◉

KEY POINTS

FINDINGS: With an increasing number of factors contributing to low back pain (LBP), the probability of finding subgroups of patients, based on a single factor exceeding a certain threshold, tends toward zero. Arbitrarily applying treatments addressing any 2 or more factors was more effective in the simulated population of patients with LBP than diagnosing and treating a single factor that maximally contributed to LBP in each individual.

IMPLICATIONS: A reductionist approach aimed at identifying 1 or a few dominant factors contributing to LBP, or subclassifying patients based on those factors, will likely not result in the discovery of strong modifiers of treatment effects. The simulations suggest that multimodal management of LBP will likely be more effective than unimodal treatment.

CAUTION: The main assumptions influencing the specific numerical results were that factors contributing to LBP were uniformly distributed and that there were no interactions among them. While these assumptions affect the complexity of the modeled LBP problem, the simulation trends will likely hold for more complex models.

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APPENDIX

HYPOTHESIS 1

To estimate how many people can be subclassified based on identifying a single factor exceeding a certain threshold θ, we needed to calculate the probability (P) of a factor $X_1>\theta$ in the population with LBP. The following derivation is a consequence of the sampling distribution of a large number of uniform variates on the unit interval (0, 1).

Let U_1, U_2, ... be independent, random variables uniformly distributed on the unit interval (0, 1). Let θ be a given number: $0<\theta<1$. Let $S_{k+1} = U_1 + \ldots + U_{k+1}$, $X_i = U_i/S_{k+1}$, $1\leq i\leq k + 1$.

We start with a set of cases where $X_1>\theta$:

$$\{X_1 > \theta\} = \{U_1 > \theta S_{k+1}\} = \{(1-\theta)U_1 > \theta(U_2 + \cdots + U_k)\} = \{(1-\theta)U_1 > \theta S_k\} =$$

$$= \{(1-\theta)U_1 > \theta S_k\} = \left\{S_k < \frac{(1-\theta)}{\theta}U_1\right\}$$

Therefore, probability of the event $\{X_1>\theta\}$ is:

$$P(X_1 > \theta) = P\left(S_k < \frac{(1-\theta)}{\theta}U_1\right) = E\left[P\left(S_k \middle|< \frac{(1-\theta)}{\theta}u \middle| u\right)\right] = \int_0^1 P\left(S_k < \frac{(1-\theta)}{\theta}u\right)du$$

where u has uniform distribution on (0,1). For large k, the distribution of S_k is approximately normal, with mean $k/2$ and standard deviation $\sqrt{k/12}$ (Irwin-Hall distribution).[10] Therefore,

$$P(X_1 > \theta) \approx \int_0^1 \Phi\left(\frac{1-\theta}{\theta}u, \frac{k}{2}, \sqrt{\frac{k}{12}}\right)du \to 0 \text{ as } k \to \infty$$

in which $\Phi(z, \mu, \sigma)$ denotes the cumulative distribution function of the normal variate, with mean μ and standard deviation σ.

HYPOTHESIS 2

To address hypothesis 2, we must estimate the expected value of the sum of m factors $E(X_1 + X_2 + \ldots + X_m)$ and the expected value of the maximum factor $E(X_{max})$. Based on the same Irwin-Hall distribution,[10]

$$E(S_{k+1}) = \frac{k+1}{2}$$

and

$$E\left(\frac{U_1}{S_{k+1}}\right) = E\left(\frac{U_2}{S_{k+1}}\right) = \cdots = E\left(\frac{U_{k+1}}{S_{k+1}}\right) = \frac{1}{k+1}E\left(\frac{S_{k+1}}{S_{k+1}}\right) = \frac{1}{k+1}$$

APPENDIX

Therefore,

$$E(X_1 + \cdots + X_m) = E\left(\frac{U_1 + \cdots + U_m}{S_{k+1}}\right) = mE\left(\frac{U_1}{S_{k+1}}\right) = \frac{m}{k+1}$$

The expected value of the maximum factor is a ratio of 2 random variables, and to the first-order approximation is

$$E(X_{max}) = E\left(\frac{\max U_1}{S_{k+1}}\right) \approx \frac{E(\max U_i)}{E(S_{k+1})} = \frac{2}{k+1} E(\max U_i) =$$

$$= \frac{2}{k+1} \int_0^1 u(k+1)u^k du = \frac{2(k+1)}{k+1} \int_0^1 u^{(k+1)} du = 2\int_0^1 u^{(k+1)} du = \frac{2}{k+2} \rightarrow 0 \text{ as } k \rightarrow \infty$$

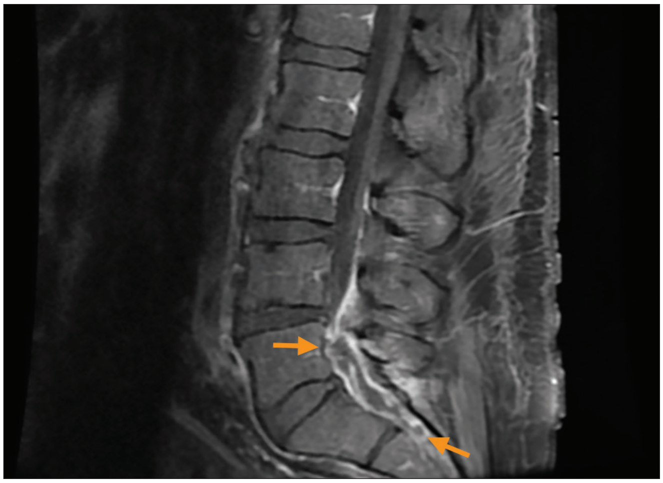

FIGURE 1. Sagittal, T1-weighted, fat-saturated, postcontrast magnetic resonance image slightly left of midline demonstrates the rim-enhancing epidural abscess extending from L5 to S2 (arrows).

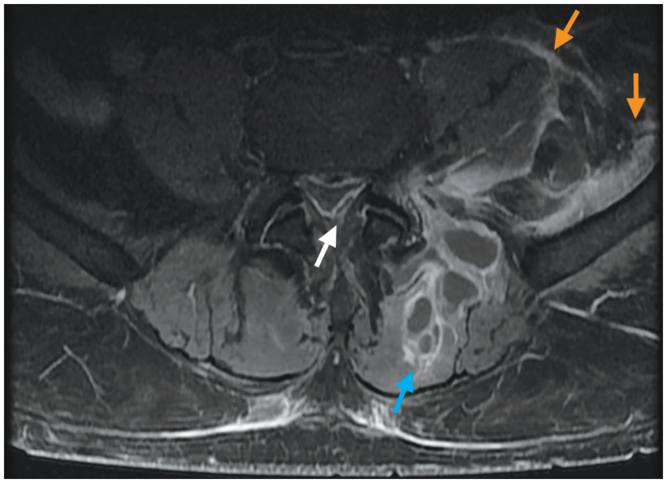

FIGURE 2. Axial, T1-weighted, fat-saturated, postcontrast magnetic resonance image at the L5 level demonstrates the multiloculated, rim-enhancing left paraspinal muscle abscess (blue arrow). Adjacent enhancement of the left psoas and iliacus muscles is consistent with myositis or intramuscular abscesses (orange arrows). The thin rim-enhancing epidural abscess is partially visualized (white arrow).

Epidural and Paraspinal Abscess Presenting as Acute Low Back Pain

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A 56-YEAR-OLD MAN SOUGHT CARE from a primary care physician (PCP) 5 days after lifting a heavy box produced acute, isolated lumbar pain. The PCP diagnosed a lumbar disc herniation and referred the patient to physical therapy without diagnostic imaging. Four days later, during the physical therapist's examination, the pain was more severe and in the prior 48 hours had spread into the left flank and lower abdomen, with new onset of fatigue, malaise, chills, and night sweats.[2] During the physical exam, the patient had nondermatomal left anterior thigh and leg paresthesia with normal myotome and reflex assessments. Severe pain significantly limited all lumbar spine motions and active and passive left hip flexion to less than 90°. Extreme tenderness to palpation was present in the left lower abdominal quadrant and low back. The patient was afebrile and without etiology for infection or systemic disease.

Due to the presence of multiple red flags and the recent change in status, the patient was transferred to the emergency department for further evaluation. Magnetic resonance imaging revealed a spinal epidural abscess from L5 to S2, with an associated multiloculated abscess within the adjacent left paraspinal muscles (**FIGURES 1** and **2**; **FIGURE 3**, available at www.jospt.org). Blood culture identified a methicillin-susceptible *Staphylococcus aureus* pathogen. Image-guided drainage of the paraspinal abscess and initiation of intravenous oxacillin and oral rifampin antibiotic treatment occurred during a 10-day hospital admission. The patient continued antibiotic treatment for 6 months and experienced complete recovery. Suspicion of a nonmechanical source of pain prompted early diagnosis and averted potential neurological consequences.[1] ◉ *J Orthop Sports Phys Ther 2019;49(6):482. doi:10.2519/jospt.2019.8456*

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[CLINICAL COMMENTARY]

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Convergence and Divergence of Exercise-Based Approaches That Incorporate Motor Control for the Management of Low Back Pain

SYNOPSIS: Many approaches for low back pain (LBP) management focus on modifying motor control, which refers to motor, sensory, and central processes for control of posture and movement. A common assumption across approaches is that the way an individual loads the spine by typical postures, movements, and muscle activation strategies contributes to LBP symptom onset, persistence, and recovery. However, there are also divergent features from one approach to another. This commentary presents key principles of 4 clinical physical therapy approaches, including how each incorporates motor control in LBP management, the convergence and divergence of these approaches, and how they interface with medical LBP management. The approaches considered are movement system impairment syndromes of the lumbar spine, Mechanical Diagnosis and Therapy, motor control training, and the integrated systems model. These were selected to represent the diversity of applications, including approaches using motor control as a central or an adjunct feature, and approaches that are evidence based or evidence informed. This identification of areas of convergence and divergence of approaches is designed to clarify the key aspects of each approach and thereby serve as a guide for the clinician and to provide a platform for considering a hybrid approach tailored to the individual patient. *J Orthop Sports Phys Ther 2019;49(6):437-452. Epub 15 May 2019. doi:10.2519/jospt.2019.8451*

KEY WORDS: *clinical perspectives, low back pain, motor control, spinal control*

Many physical approaches to managing low back pain (LBP) include exercise that aims to change motor control. In this context, motor control refers to motor, sensory, and central processes involved in control of posture and movement. Although different approaches share the underlying assumption that the manner in which individuals use their body and load their tissues is related to the development and maintenance of their conditions, there are differences in how motor control is assessed and trained, as well as differences in proposed mechanisms for its efficacy. This commentary aims to describe how motor control is used in 4 clinical approaches commonly used in physical therapy, and to consider areas of convergence and divergence between these approaches and how these approaches interface with nonsurgical medical management of patients with LBP.

Clinical Approaches That Focus on Motor Control

The clinical approaches included in this

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commentary are movement system impairment (MSI) syndromes of the lumbar spine, Mechanical Diagnosis and Therapy (MDT), motor control training (MCT), and the integrated systems model (ISM). These were selected with the objective of including approaches with some diversity of underlying concepts, that consider motor control as a central (MSI, MCT, ISM) versus an adjunct feature (MDT), and that are evidence based (MSI, MDT, MCT) versus evidence informed (ISM). Below is an overview of the key features of each approach, including concepts, assessment, treatment, and key research evidence.

MSI Syndromes of the Lumbar Spine

Underlying Concepts The movement system consists of physiological organ systems that interact to produce movement of the body and its parts (**FIGURE 1**). Movement system impairment syndromes are one set of classifications of patients with musculoskeletal pain and comprise the neuromusculoskeletal components of the system. The theoretical construct of MSI syndromes is depicted in the kinesiopathologic model,[67,116,149] which proposes how movement induces pathology (**FIGURE 2**).

In this model, the main inducers of movement impairments are the repeated movements and sustained alignments of everyday activities. The changes in tissues associated with repetition of activities are proposed to induce movement impairments. Studies have demonstrated that rotation-related sports induce movement impairments in individuals with LBP.[13,38,143,146,148,156] Indirect support for a link between daily activities and the problem is provided by evidence that correction of movement impairments during these activities significantly reduces symptoms for 1 year.[146] The characteristics of specific tissue, movement, and alignment changes are proposed to vary because of intrinsic personal characteristics and extrinsic factors, such as the type and intensity of activities. According to the model, the result of these tissue adaptations is a joint that moves more readily in a specific direction (ie, flexion, extension, rotation) than in other directions and more readily than another joint with a similar movement direction,[82] thus becoming the path of least resistance for movement.

The model proposes that the major determinants of the path of least resistance that cause a joint to move too readily are (1) joint relative flexibility (intrajoint and interjoint),[119,125] (2) relative stiffness (passive tension of muscle and connective tissue),[35,67,150] and (3) motor performance and learning.[95,96,147,151] The predisposition for a joint to move more readily in a specific direction, only a few degrees different in patients with LBP than in controls,[119,125] suggests the presence of accessory-motion hypermobility that induces microtrauma that becomes macrotrauma over time.

There are several sources of evidence for the change in joint flexibility contributing to a low threshold for motion. First, patients present with similar types of lumbar motion, for example, rotation, across different clinical tests involving movement of the trunk and lower extremities in a variety of positions.[35,144] Second, the range of lumbar/lumbopelvic motion most often varies with the movement of one lower extremity relative to the other, supporting variation in the flexibility of the joint.[144] Third, motion-capture studies have shown that patients with LBP initiate lumbar/lumbopelvic movement within a few degrees of initiating limb motion and a few seconds earlier than individuals without LBP.[95,119,125] Most studies evaluated knee flexion and hip lateral and medial rotation in the prone position.[68-70] The early onset of motion and occurrence with movements of the trunk and lower extremities in a variety of positions support the concept of intrinsic changes in joint flexibility.

Additional support is derived from studies that demonstrate that patients classified as "extension-rotation" have greater lumbopelvic rotation with hip

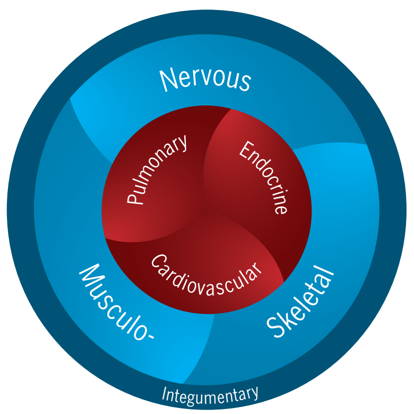

FIGURE 1. Human movement system. Reproduced with permission from Washington University in St Louis Program in Physical Therapy, licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International license. Based on a work at https://pt.wustl.edu/about-us/.

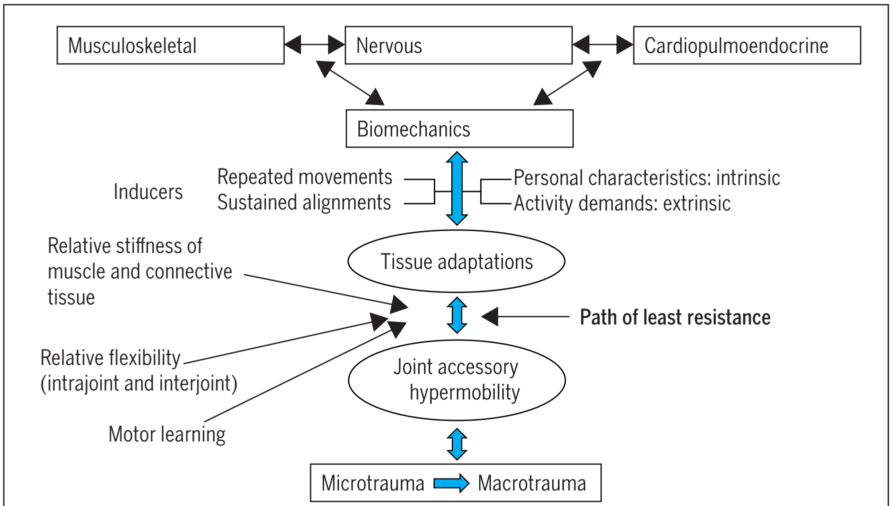

FIGURE 2. The kinesiopathologic model, a theoretical construct of movement system impairment syndromes.

lateral rotation in prone with one extremity than with the other.[144] These patients also demonstrate asymmetrical lateral trunk flexion.[35] This contrasts with patients classified as "rotation," who have symmetrical lumbopelvic rotation with both lower extremities and lateral trunk flexion.[35] Studies of lateral trunk flexion have shown that trunk passive elastic energy asymmetry is predicted by factors of sex and muscle in LBP, whereas in controls only sex is predictive.[34] Thus, muscle factors in LBP likely contribute to the greater imbalance in passive elastic energy. Although muscle and connective tissue can contribute,[34] intrinsic flexibility of the spine is also a factor.

Assessment Procedures Consistent with the model that a specific movement direction is problematic, the primary objective of the clinical examination is to identify the movement directions that elicit symptoms (the path of least resistance) and the contributing factors. The examination also identifies the associated movement impairment, such as excessive early lumbar flexion and limited hip flexion during forward bending. Then, the effect of the patient correcting the movement impairment on the symptoms is noted. Correction of the early lumbar motion has been shown to decrease symptoms.[96,145,151]

The systematic movement exam consists of tests performed in different positions: standing, supine, sidelying, prone, quadruped, and sitting. The tests involve movements of the extremities, primarily the lower extremity, and the trunk. The patient moves in the preferred manner while the symptoms and movement patterns are noted. Then, the movement is corrected, primarily by limiting any associated lumbar motion, and effects on symptoms are noted.[145,150-152,156] An important component of the exam and treatment involves instructing the patient in correct performance of basic mobility activities, as well as those during work and, if relevant, fitness or sports activities. These activities include how to roll, how to come to sitting when recumbent, during sit-to-stand, in a sitting position, when going up and down stairs, during gait, as well as when bending, returning to standing, and sidebending.[95,96,147,151] All these motions are assessed as part of the examination.

The reliability of clinicians performing the examination tests[40,134,150] and the validity of the classifications have been examined and are acceptable.[152] The reliability of examiners to classify patients has also been established (approximately 70% accuracy).[39,40,107,134] Alignment differences between patients with a specific lumbar classification and controls have been documented.[107,126] Other studies have documented that symptoms are elicited with movements of the spine and the extremities and that preventing lumbar motion during limb movements decreased or eliminated the symptoms.[96,145,150] Studies using motion capture have demonstrated that lumbopelvic motion occurs more readily during knee flexion and hip rotation in patients with LBP than in pain-free individuals.[119] A variety of other details related to variations in symptom behavior in men versus women and in the different classifications have also been examined.[33,70]

The validated classifications are based on the motion or alignment that provokes the patient's symptoms. The trunk/lower extremity movements that cause the offending movement are then eliminated or reduced to correct or prevent the offending spinal movement.[151]

The validated classifications are "lumbar extension" (greater lumbar extension in standing; symptom provocation: trunk/lower extremity movements causing lumbar extension; symptom elimination/reduction: alignment correction or prevention of extension), "extension-rotation" (symptom provocation: trunk/lower extremity movements causing extension and rotation; motions are asymmetrical; symptom elimination/reduction: correction of both movement directions), and "rotation" (symptom provocation: rotation or sidebending of the trunk/lumbopelvic rotation with rotation of both hips; symmetrical; symptom elimination/reduction: correction/prevention of lumbar motion).[152]

Intervention Outline During the examination that comprises basic mobility activities, many of which elicit symptoms, the patient is immediately instructed to correct the motion that usually reduces or eliminates the symptoms. The results of the examination identify the movement direction that most consistently elicits symptoms and the associated movement control impairments. The patient is informed of the movement direction and practices the movement correction. The major emphasis is placed on correcting basic daily activities and specifically on other types of activities that elicit symptoms, such as cooking or raking, as well as fitness or sports activities.

The patient is also instructed in specific exercises designed to correct the identified movement impairments. The exercises aim to prevent the offending lumbar motion while moving the trunk and lower extremities. Most often, this involves improved lumbopelvic control by contracting the abdominal muscles and improved extensibility of the hip muscles by elongation of the muscles while preventing lumbopelvic motion.

Evidence of Efficacy A recent randomized controlled trial (RCT) has supported that teaching the patient to keep the spine in neutral during basic mobility and fitness activities reduced symptoms for 6 months after 6 weekly visits consisting primarily of performance training.[146] At 1 year, the symptoms remained significantly lower than at the initiation of treatment. Subsequent RCTs of patients with chronic LBP have shown greater efficacy for symptom reduction by correcting movement and alignment impairments by motor skill training according to the MSI approach than by using strength and flexibility exercises.[147] Research has also demonstrated that patients adhere to training of functional activities significantly more often and for longer than they do to strength/flexibility exercises.[142,146]

Mechanical Diagnosis and Therapy

Underlying Concepts The MDT paradigm is unique in this commentary in that treatment is entirely based on the findings of a mechanical examination of the behavior of the pain source for each patient. Mechanical Diagnosis and Therapy is typically not considered a motor control approach, yet MDT considers posture correction and control to be essential features of recovery and prevention for every patient with a directional preference. The type of correction is determined by establishing the patient's directional preference associated with pain relief during the initial assessment. The performance of matching directional exercises is the key component of treatment, along with similar directional postural modifications. For most, that involves establishing and maintaining a lumbar lordosis and avoiding spinal positions associated with symptom provocation, such as prolonged spinal loading in lumbar flexion.[157] Experiencing the relationship between relief of pain and an erect sitting posture can be sufficiently motivating for most patients to learn to modify their sitting posture to prevent pain from returning.[157] In the MDT approach, patients perform their assigned directional exercise and practice the desired pain-relieving/preventative posture, which then creates a new postural habit that helps prevent the return of their pain.

Assessment Procedures Assessment begins by focusing on mechanical elements in each patient's history and with a dynamic examination (**FIGURE 3**) that mechanically and systematically loads and tests the tissues considered to be the patient's pain source, to determine which familiar patterns of pain response occur as a result.

If the clinical findings/pain response patterns reveal a "directional preference" (a single direction of repeated end-range spinal loading that achieves lasting pain relief) and "pain centralization" (change of pain location toward the spine from the periphery), then this is interpreted to indicate that the patient's pain source is reversible or correctable, as well as reveals the means by which it can be reversed or corrected. This information guides the treatment and is unobtainable by other forms of clinical examination or imaging technology. Research indicates that these 2 clinical findings (**FIGURE 4**) can be elicited in 70% to 91% of patients with acute LBP and in 50% of those with chronic LBP.[17,19,20,29,77,84,89,90,121,131,155]

Numerous studies[31,32,80,81,112,127,155,158] have reported strong interexaminer reliability across clinicians possessing the credentialed level of MDT training provided by the McKenzie Institute International.

Intervention Outline The goals of MDT are to identify mechanical spinal loading strategies that eliminate pain, then implement these strategies to restore each individual's ability to function at home, work, and during recreation. An additional goal is to teach patients successful prophylactic strategies to avoid recurrences and the need for further medical care. Published data support the achievement of those goals for the subgroup that has a directional preference and centralization.

Most patients can achieve these recoveries independently after being taught individualized self-management and preventive strategies.

Evidence of Efficacy Numerous observational cohort studies,[17,19,20,29,77,84,89,90,121,131,155] RCTs,[9,10,30,36,82,89,109,118] and systematic reviews[15,98,132] have reported that patients in whom a directional preference and/or pain centralization is elicited achieved better outcomes when treated with exercises that matched their disorder's directional preference, coupled with appropriate posture modifications, compared with other forms of treatment. The interexaminer reliability of the MDT assessment findings and patient classification—validated by improved patient report of pain reduction and improvement in functional outcomes using self-management strategies—along with the high prevalence rate for directional

FIGURE 3. A "press-up" is a prone end-range lumbar extension test that, when done repeatedly, will often centralize and/or abolish axial low back pain or any variation, such as referred pain or sciatica.

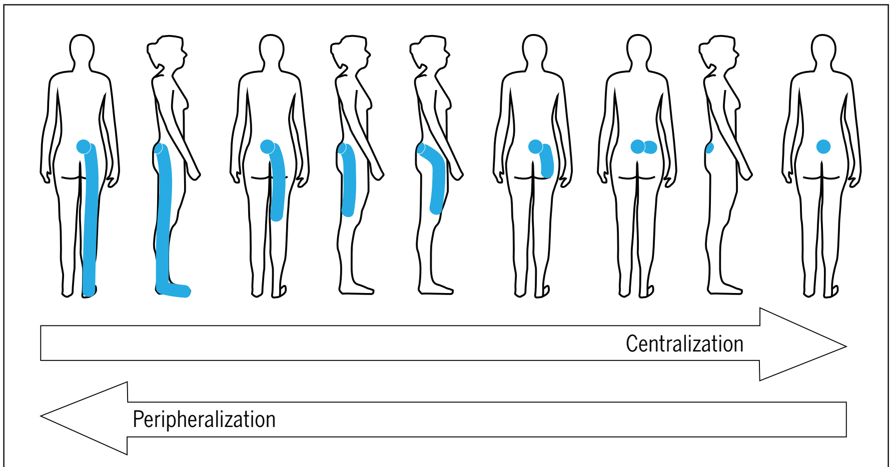

FIGURE 4. Pain "centralizes" when it is intentionally caused to retreat back toward the lumbar midline from its most distal location. It "peripheralizes" when it spreads farther away from the lumbar midline. Reprinted with permission from Donelson R. Is your client's back pain "rapidly reversible"? Improving low back care at its foundation. *Prof Case Manag*. 2008;13:87-96. https://doi.org/10.1097/01.PCAMA.0000314179.09285.5a

preference, supports this examination as a valuable component of evaluation for patients who seek care for LBP. Mechanical Diagnosis and Therapy is typically not considered a motor control approach, yet MDT considers posture correction and control to be essential features of recovery and prevention for every patient with a directional preference. In that context, motor control could be viewed as an adjunct feature of MDT treatment.

Motor Control Training

Underlying Concepts True to the complexity of motor control, MCT encompasses many aspects. It considers sensory and motor aspects of spine function, and each individual's management program is tailored to features considered to be "suboptimal" on assessment. The basic premise of MCT is that, for many individuals, inputs from the spine and/or related tissues (including nociceptive) contribute to maintenance of symptoms secondary to suboptimal loading by person-specific features of alignment, movement, and muscle activation. Motor control training aims to identify and modify the suboptimal features of motor control, with integration into function.

Considerable research has identified motor control features that differ between pain-free individuals and those with a variety of presentations of LBP. Most features are highly variable between individuals. Some examples include compromised muscle structure (eg, atrophy, fatty infiltration) and activation or contraction of muscles (eg, the multifidus[1,55,93,154] or transversus abdominis[26,55]), augmented muscle activation or contraction (eg, the obliquus externus abdominis,[58] obliquus internus abdominis,[44,46,54,72] or erector spinae[2,97]), modified postures,[16] and modified movement features (eg, augmented trunk stiffness,[56] smaller preparatory trunk movements[101]).

Motor control training aims to identify candidate features that might be relevant for the individual's pattern of symptom presentation. It is presumed that not all features will be relevant for the patient and not all individuals with a specific feature will develop symptoms. Motor control training includes therapeutic exercise to modify specific motor control features for a broad, multidimensional view incorporating psychosocial aspects of LBP (**FIGURE 5**). It is important to recognize that MCT considers the potential relevance of both "upregulation" (ie, increased/augmented activation) and "downregulation" (ie, decreased/compromised activation) of muscles. Increased/augmented activation of muscles, particularly those that are more superficial, is common. Laboratory studies reported a universal response of increased muscle activity when exposed to a noxious input, but with a pattern that was unique to each individual.[58]

There are numerous clinical examples. In response to low-load axial loading tasks (25% of body weight), individuals with LBP have greater activation of the obliquus internus abdominis than pain-free controls.[46,53,54] This has been interpreted as a strategy to enhance protection,[65] but could also be related to features such as habitual postures.[16] An MCT program reduced excessive contraction,[46] along with reducing LBP. This can be achieved within a session.[135] The contrasting observation of decreased/compromised muscle activation is also common and may be concurrent with increased activation of other muscles. There is substantial evidence of decreased[26] or delayed[63,93] activation and reduced ability to voluntarily contract muscles.[43,154] There are many mechanisms that could explain compromised activation. These include reflex inhibition[50,60] and other changes at many levels of the nervous system.[65] Activation of deep muscles such as the multifidus is also compromised by changes in structure such as atrophy[55] and fat/connective tissue accumulation,[61,83] which might be secondary to reduced activation or other mechanisms such as a local inflammatory dysregulation.[73] If downregulation of muscles such as the multifidus and transversus abdominis is identified, then the MCT program includes strategies to augment contraction in patients with acute[50] and with chronic[43,154] LBP. Programs that have included this component have decreased the recurrence of episodes of LBP[47] and improved pain/function.[117] It is a common misinterpretation that MCT aims to "upregulate" or increase muscle activity/cocontraction to restrict motion via a unidimensional focus on activation of specific muscles. This is not correct. Instead, the target should be the appropriate balance between movement and stiffness, as required by the task and the individual.[57]

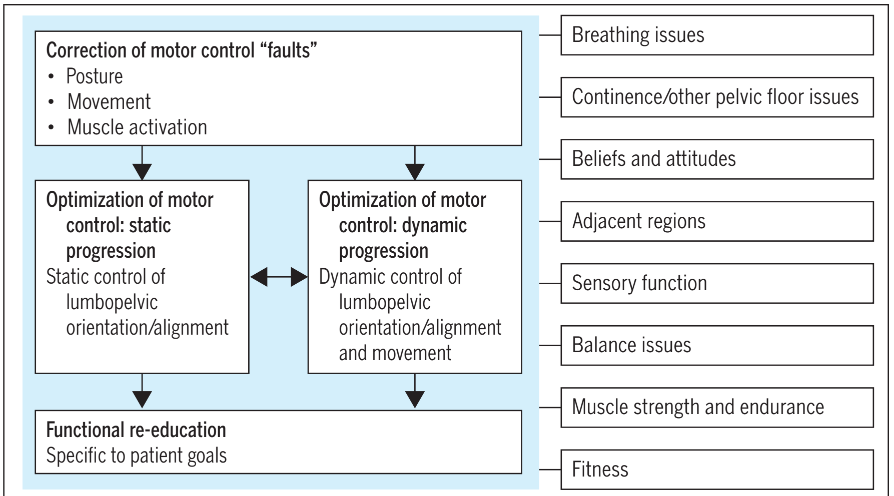

FIGURE 5. Motor control training approach.

Biomechanical/mechanical principles that are considered in program design include the following.

1. A controlled lumbopelvic unit is important for function,[100,139] requiring a balance between movement and stiffness[56,79] achieved through appropriately coordinated activation of the complex array of trunk muscles.[58,140]
2. Maintenance of a "neutral" lumbar spine position (ie, mid-range position with alignment of the trunk relative to gravity, controlled spinal curves, and frontal/transverse plane alignment) is important for sustained static positions.[14,99]
3. For many functions, movement should be initiated from the periphery (not the trunk) but should include the trunk to achieve full range.[119]
4. Adequate mobility and flexibility of adjacent joints and muscles attaching to the pelvis are required to maintain spine control during limb movement.[143]

Assessment Procedures Successful application of MCT principles relies on thorough assessment (including patient interview and physical examination); good communication skills; rapport with and an understanding of the patient, including his or her goals and concerns; and psychosocial context. Although these principles are common to several exercise approaches for LBP, tailoring the MCT treatment to the individual motor control features identified through assessment contrasts with many generalized exercise approaches. Multiple elements of assessment have been shown to have acceptable clinimetric properties.[110,128,133]

1. Assessment of trunk muscle control: assessment identifies features of muscle activation/contraction considered suboptimal (more or less activity/muscle contraction than expected for a task). Clinical muscle tests have been developed for specific trunk muscles that are commonly involved in LBP. These include deep muscles of the abdominal wall[42,43] and the paraspinal muscles, including the multifidus.[42,43] Ultrasound imaging can be used in clinical practice to measure the size and activation/function of trunk muscles.[128,133] Validity and reliability of this measurement method have been established; measures obtained by ultrasound imaging have been validated against measures obtained from magnetic resonance imaging[45,48,49,55] and intramuscular electromyography.[62]
2. Assessment of posture and movement: assessment is based on the identification of features that deviate from those considered ideal for a task and relevant for the patient's presentation. This is based on evidence from a broad base of research that shows person-specific postural attributes related to symptom profile,[16,23] relationships of postures and movements to modified muscle activation,[14] and that posture can be modified with exercise.[25] Tests utilized in MCT are drawn from multiple sources, including related motor control approaches (see Hodges et al[66] for review). Although reliability and validity of some tests have been established,[21,22] further research in this area is required.
3. Assessment of functional tasks: assessment of more complex functional tasks involves careful observation and relies on principles that are common across multiple motor control approaches (see Hodges et al[66] for review).
4. Assessment of broader dimensions of LBP: MCT incorporates, as required, consideration of many features that may determine the relevance of motor control for the patient's symptoms (eg, underlying pain mechanism) and features that may interact with the potential to achieve ideal control. These include a range of features that are related to motor control of the trunk and LBP psychosocial features,[11] breathing,[74,75] continence[124] and pelvic floor function,[111] adjacent joint function,[143] strength and endurance,[115] balance,[71] sensory function,[11] general fitness, etc.[66] Specific assessments used to evaluate these features vary and require further refinement.

Intervention Outline The following is an example of an MCT protocol.[53,66]

1. Optimization of muscle activation: individualized training targets the features identified in the assessment that suggest upregulation and/or downregulation of activity/contraction as required; that is, the training employs strategies to decrease overactive muscles and increase recruitment of muscles found to have demonstrable impairments on clinical muscle testing.[43,154] Training can include voluntary contraction of deeper trunk muscles to teach the skill of activating these muscles[138] for later integration into function, and reducing "overactivity" or increasing "underactivity" of more superficial muscles. The MCT approach to training lumbar paraspinal[135] and abdominal muscles[37] has been shown to induce immediate and sustained[136] changes in coordination of lumbar trunk muscle activation in recurrent LBP. Techniques to assist this phase include position change, feedback (eg, ultrasound imaging of muscle contraction) (**FIGURE 6**), relaxation strategies, imagery, and soft tissue techniques.

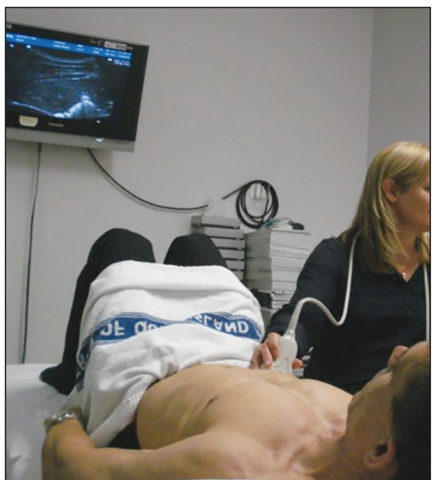

FIGURE 6. Ultrasound imaging can be used for detailed assessment and biofeedback of contraction of the deep trunk muscles, including the transversus abdominis and multifidus.

2. Optimization of posture and movement: features of spinal position that are considered suboptimal in the assessment and relevant for symptoms are corrected/trained. Among many options, this can include functional retraining in upright positions, with adjustment of spinal alignment; restoration and maintenance of normal patterns of respiration while exercising; dissociation of movement of the lumbar spine from that of the hip and thorax; practicing functional tasks such as sit-to-stand, with optimal spinal alignment and motion; and control of alignment and motion when challenged by unstable support.[66,76]
3. Functional integration and conditioning: this phase targets the patient's goals and can include exercises to achieve increased endurance of trunk muscles in functional activities and positions. Resistance can be added, with instruction to maintain spinal alignment when using weights. Flexible maintenance of spinal alignment in daily activities is encouraged, without causing rigidity or interfering with normal movement. Application of MCT according to these principles has decreased LBP and the occurrence of new injuries in several groups, including athletes.[51-53]
4. Broader dimensions of management: similar to other management approaches for patients with chronic LBP, MCT can be combined and integrated with other approaches, such as those that manage psychological features (eg, fear, catastrophizing, etc). For MCT, as for many other approaches, understanding pain processes, setting appropriate goals, providing reassurance (minimizing fear avoidance), and restoring pain-free normal movement are paramount.

Evidence for Efficacy Over the last 3 decades, changes to key recommendations in clinical practice guidelines for the management of LBP have placed greater emphasis on self-management and exercise programs targeting functional improvement.[28] A systematic review of 45 exercise trials (all forms of exercise) in patients with chronic LBP showed a modest benefit of exercise for nonspecific LBP, with greater efficacy than other conservative therapies.[120] Although effect sizes were modest, this finding should not be dismissed, because no intervention for LBP has a large effect when delivered in an RCT. Exercises classified as "coordination/stabilization" generally showed a positive effect. Another systematic review of 29 trials of MCT showed a clinically important effect compared with minimal intervention for chronic LBP,[117] but no superiority to other forms of exercise. Of note, early trials with large clinical effects applied MCT to specific patient groups in an individualized manner,[47,108,129] whereas most trials with modest effects have applied nonindividualized treatments to patients with nonspecific LBP. Individualization of treatment, which is now generally recommended, appears to be important. Several trials have shown that specific baseline features of motor control[27,137] and features of symptom presentation[94] are associated with better responses to treatment. These promising findings require further investigation.

Integrated Systems Model

Underlying Concepts The ISM[85,86,88] (**FIGURE 7**) is an evidence-informed (ie, founded on research findings, but not yet tested in RCTs), clinical-reasoning approach to organize knowledge from multiple fields of science and clinical practice for the nonsurgical care of individuals with disability and pain. This approach is compatible with the "regional interdependence model," a term used to describe clinical observations that regions of the body appear to be musculoskeletally

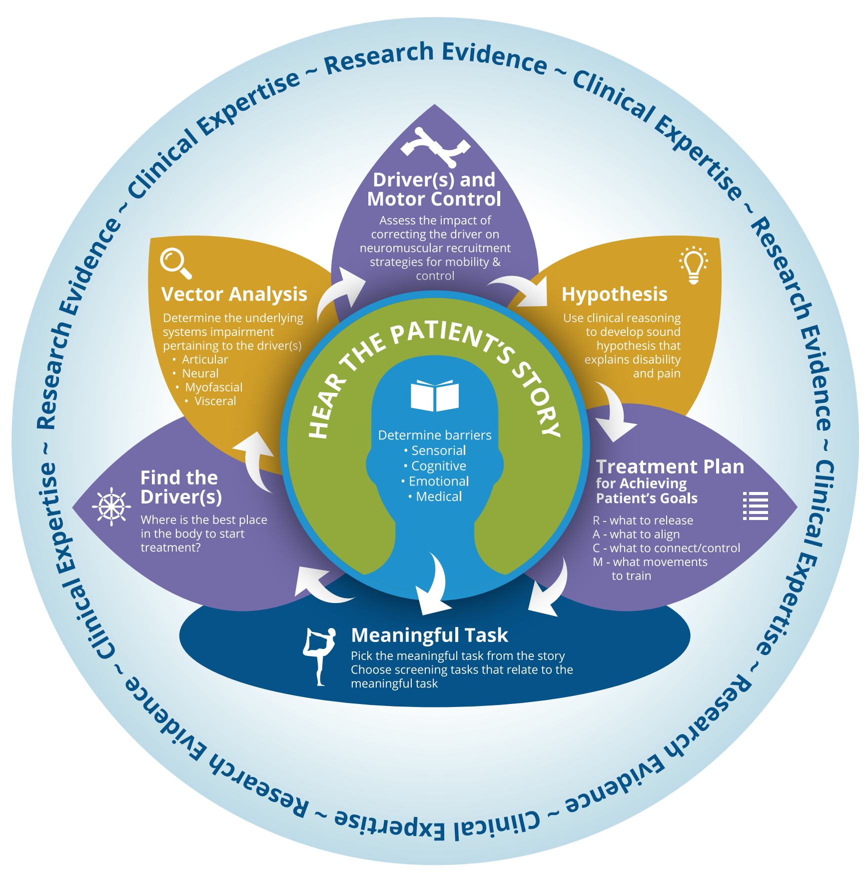

FIGURE 7. The integrated systems model. Reprinted with permission from Diane Lee.

linked, such that dysfunction in one body region could potentially lead to abnormal stresses to other body regions and subsequent development of dysfunction/pain in those regions.[130] Treating people with complex biopsychosocial problems requires an understanding of the relationship between, and the contribution of, various body regions and systems that ultimately manifest as cognitive, emotional, or sensorial dissonance. Collectively, this dissonance can be interpreted by the individual as threatening, and this is thought to have the potential to manifest as pain anywhere in the body, fear of movement, movement impairments, anxiety, breathing disorders, and/or incontinence.[3,5,12,64,103,123,141] Individuals with chronic LBP present with many of these features and have complex histories containing (1) multiple past high loads or accumulative traumas to areas of the body, many only partly resolved, (2) beliefs and cognitions that present barriers to recovery, and (3) poor lifestyle habits.

Ultimately, the ISM considers the impact that each system and body region has on function and performance of the whole body and person.

Assessment Procedures An ISM assessment begins with a patient interview to determine the contributions of the individual's sensations, thoughts, and beliefs to the clinical picture. Negative emotions and beliefs, or thoughts, are common in patients with complex LBP presentations and can be primary barriers to recovery.[113] The patient's goals are also determined through the patient interview, and these goals determine the tasks analyzed in the physical examination.[122] The tasks may not always relate to the location of pain. For example, evaluating the squat task and sitting posture is meaningful for someone who experiences LBP with sitting, but not relevant for an individual with LBP that intensifies with walking. An evaluation of strategies used for stepping forward and thoracic rotation, 2 requisite components of walking, is more meaningful for the latter individual.

The patient is asked to report any sensations evoked as the task is performed, while the clinician observes/palpates each region of the body and notes any areas with alignment, biomechanics, and/or control considered to be suboptimal. This requires an understanding of what is optimal for each body region for that task. Subsequently, manual or verbal cues are given to change the alignment, biomechanics, and/or control used for a body region, and the impact of this correction on the patient's experience, as well as any change in performance of other body regions, is noted. This is called "finding the driver," which refers to the region of the body that, when corrected, results in the best improvement in both the experience and performance of the task. For an individual with LBP, it may be the hip, foot, pelvis, thorax, neck, or a combination of corrections.[102,105,144] The low back is often the "victim" of suboptimal strategies for transferring loads through the trunk, regardless of whether the pain stage is acute or chronic.[92,93] The driver can change both within and between treatment sessions when the whole body is evaluated for each task. The driver informs the clinician where to focus treatment.

Further tests of the driver (the body region found to have the greatest impact on the function/performance of the meaningful task), such as active mobility/control and passive mobility/control, reveal the contribution of various system impairments (articular, neural, myofascial, and/or visceral) to determine individualized treatment, as no 2 patients have identical thoughts, beliefs, and system impairments culminating in their experience. These tests are directed to the driver (thoughts/beliefs, emotions, hip, pelvis, low back, thorax, foot, etc).

In summary, assessment using the ISM approach involves the following.

1. Choosing a relevant assessment task according to the patient's movement goals.
2. Analyzing how the patient performs the task, using observation and manual examination.
3. Correcting alignment, biomechanics, and/or control with manual examination and/or words/cues to assess the impact of changing performance and the impact of changes on other body regions.
4. Choosing to first treat the area of the body that has the greatest impact on performance of the task, regardless of the location of pain.

Intervention Outline Intervention is based on the findings of the clinical examination and a clinical-reasoning approach.[85,87] Intervention using the ISM approach may, therefore, involve a variety of treatment approaches based on different findings from different systems, such as treatments based on altered active control (including motor control[6,42,53-55,59,64,66]), passive mobility or passive control of joint structures[4] (eg, stress tests) or myofascial tissue, or neurodynamics of the nervous system.[106] The assessment findings direct the initial treatment, which is individualized according to the underlying system impairments impacting the body region.

Each treatment may include the following elements.

1. Education: to address negative thoughts/beliefs about pain[12,91] and manual therapy to mobilize any joints thought to be fibrotic or where mobility is reduced secondary to overactive muscles[6,104,114] or fascia.[8]
2. Motor control training[42,53-55,59,64,66,135,136]: to teach better recruitment strategies for neuromuscular support of joints for both static loading and movement, and to restore optimal recruitment of the transversus abdominis, deep multifidus, and pelvic floor muscles.
3. Movement training: to build strength, endurance, and capacity for the individual's movement goals.[24,129]

Evidence for Efficacy This approach is evidence informed, and, although aspects have been tested in trials, no RCT has yet tested the efficacy of the entire approach. The clinician's challenge is to decide which treatment is appropriate for the individual patient. The ISM aims to help clinicians use both the evidence and their experience to clinically reason the best way forward for individuals with disability or pain.

Convergence and Divergence of Consideration of Motor Control in the Management of LBP

Due to its diversity in presentation, LBP has been identified as a condition that may be amenable to subgrouping. Classification of patients to subgroups has been highlighted as a research priority for heterogeneous disorders such as LBP.[7,41] A major aim of subgrouping is to identify groups of individuals who may be more or less responsive to a specific treatment, based on certain presenting characteristics.[18] Evidence to support the potential benefits of identifying different subgroups of patients with LBP who will predictably respond to specific treatments comes from recent trials that show larger effect sizes for MCT in individuals with specific baseline features[27,94,137] and from the large clinical effects identified in early trials that applied MCT to specific groups of patients with LBP.[47,108,129]

While no single approach will solve the entire LBP problem, identifying subgroups of patients whose condition can be resolved by subgroup-specific treatments should be prioritized. Although application of motor control theory to LBP management varies, there is convergence. The **TABLE** summarizes key features considered by each motor control approach. Areas of convergence/similarity between approaches include the following.

1. All approaches incorporate detailed assessment (including patient interview and physical examination) to guide individualized treatment, but the elements addressed differ.
2. All approaches include clinical reasoning. Although some individual elements of the approaches may help some patients when used in isolation, effect sizes appear to be larger when treatment involves integrated use of multiple components in a clinical-reasoning framework, matched to individual patients.[94,153]
3. All approaches assume that tissue loading contributes to symptom maintenance.
4. Some aspects of treatment aim to optimize tissue loading.
5. Correction of posture/alignment is considered in all approaches, particularly with reference to maintenance of a specific alignment during sustained postures.
6. Careful and progressive instruction regarding how to appropriately limit lumbar motions and move appropriately at the hips during function is a common theme in most approaches.
7. Attention is placed on the patient-therapist alliance: the importance of identifying subgroups, understanding the patient's goals and expectations, use of appropriate communication skills, patient education, safety, self-care and patient independence, working together with the patient and the medical/multidisciplinary team, setting realistic goals, reassurance to minimize fear avoidance, understanding pain processes and their relevance, the importance of pain-free movement, and the need to promote LBP prevention.

There are also divergences between approaches.

1. Not all approaches have shown reliability in identifying subgroups that the approach can and cannot treat with predictive effectiveness.
2. Approaches differ somewhat in their primary focus, the most obvious being that MDT emphasizes evaluation of patterns of symptom response to a standardized group of repeated end-range spinal loading tests, whereas the MSI approach, MCT, and the ISM stress correcting alignment and movement patterns, but within different clinical frameworks.
3. Initial management differs. Mechanical Diagnosis and Therapy seeks to identify mechanical subgroups, and patients are taught to perform exercises based on this assessment; the MSI approach involves instructing the patient in alignment and movement correction; the ISM aims to "release and align"; and MCT enhances/reduces muscle activity and modifies alignment and movement as required.
4. Evidence for assessment and treatment differs. Although there are varying levels of evidence for assessment techniques and the efficacy of MDT, the MSI approach, and MCT, the ISM has not been tested, but some assessments and treatments included in the ISM approach have been studied.

The wrong question to ask is which approach is most effective. Rather, by identifying and validating subgroups, some patients can be more effectively treated with one approach than with another.[78] Further, patients often prefer the type of intervention they are willing to undertake and adhere to. Clinicians also have differing skill sets, levels and types of training, levels of expertise, and previous experiences. As LBP can be multifactorial, ideal management must first seek to reliably identify subgroups for which there are predictably effective treatments. Those validated subgroups will then inform the type of intervention needed to bring about improvement: mechanical, medication, motor control, psychosocial, injection, or even surgery. This may require integrating other health professionals who can advise on other forms of treatment (eg, appropriate medication). Ideally, those approaches would be complementary and enhance the response to physical and neuromuscular approaches.

Interface With Nonsurgical Medical Management

Subgrouping patients via movement patterns, posture, and provocative and symptom-relieving mechanical testing, such as the methods described above, is not only relevant for physical therapists, but also an important concept for health care providers of any profession managing patients with LBP. This consideration aids removal of the "non" from "nonspecific" LBP.

TABLE | **Features of the Approaches**

	Medical Approach	MDT	Movement Systems Approach	Motor Control Training	Integrated Systems Approach
Evidence					
Evidence for effectiveness for patients with acute LBP	?	Yes			
Evidence for effectiveness for patients with chronic LBP	?	Yes	Yes	Yes	
Demonstrated reliability and validity of assessments		Yes	Yes	Yes	
Treatment components related to motor control					
Treatment based on detailed physical examination	Yes	Yes	Yes	Yes	Yes
Spinal posture/alignment is assessed and trained	Yes	Yes	Yes	Yes	Yes
“Neutral spine” is a key feature			Yes	Yes	Yes
Movement is assessed and trained		Yes	Yes	Yes	Yes
Movement quality is a key feature	Yes		Yes	Yes	Yes
Muscle activation is assessed and trained				Yes	Yes
Aim for pain-free movement	Yes	Yes	Yes	Yes	Yes
Focus on importance of mechanical/biomechanical focus		Yes	Yes	Yes	
Body awareness is considered in assessment and treatment			Yes	Yes	Yes
Breathing pattern is assessed and trained			Yes	Yes	Yes
Mobility of adjacent areas is assessed and trained			Yes	Yes	Yes
Includes exercise that aims to integrate into function rehabilitation		Yes	Yes	Yes	Yes
Includes exercise to enhance muscle endurance				Yes	Yes
Includes exercise to enhance muscle strength			?	Yes	?
Biofeedback is used to guide motor control training			Yes	Yes	Yes
Additional aspects considered in design of treatment					
Patient interview provides information to guide treatment application	Yes	Yes	Yes	Yes	Yes
Identifies directional preference in response to mechanical loading		Yes			
Identification of “pain generators” is important		Yes			Yes
Whole-person assessment to identify the “driver” of the patient’s presentation		Yes			Yes
Approach considers patient’s lifestyle	Yes	Yes		Yes	Yes
Self-management is advocated	Yes	Yes	Yes	Yes	Yes
Aims to enhance prevention of further LBP episodes	Yes	Yes	Yes	Yes	Yes
Approach can be combined with other treatments	Yes	Yes	Yes	Yes	Yes
Approach is staged with guidance for progression of training	Yes	Yes		Yes	Yes
Adjunct treatments					
Considers injection of drugs	Yes	*			
Considers prescription of oral medication	Yes	*			
Psychosocial features are assessed and targeted with management	Yes	†		Yes	Yes
Training					
Approach requires specialized training	Yes	Yes	Yes	Yes	Yes
Credentialed training is available		Yes			Yes

Abbreviations: LBP, low back pain; MDT, Mechanical Diagnosis and Therapy.
**Studies argue that response to MDT assessment may aid this decision (van Helvoirt H, Apeldoorn AT, Knol DL, et al. Transforaminal epidural steroid injections influence Mechanical Diagnosis and Therapy (MDT) pain response classification in candidates for lumbar herniated disc surgery. J Back Musculoskelet Rehabil. 2016;29:351-359. https://doi.org/10.3233/BMR-160662).*
†Studies argue that psychological features may be improved by positive response to treatment (Takasaki H, Saiki T, Iwasada Y. McKenzie therapists adhere more to evidence-based guidelines and have a more biopsychosocial perspective on the management of patients with low back pain than general physical therapists in Japan. Open J Ther Rehabil. 2014;2:173-181. https://doi.org/10.4236/ojtr.2014.24023).

Identification of relevant motor control features or a specific response to a movement test can inform specific movements and corrective exercises, with a rapid response for some patients. Other patients may have a presentation complicated by features such as differences in pain processing, experience of intense pain, fear avoidance, and previous experiences that compromise their full participation in physical treatments. These patients may benefit from coordinating physical and medical treatments to fully accomplish recovery from an episode of LBP and establish a maintenance program and future self-management of LBP episodes. A coordinated interprofessional approach, including medical management, is required to achieve the best outcomes. The **TABLE** presents some of the interfaces between medical and motor control approaches.

At initial presentation, a thorough examination alludes to the potential benefit of combining medical and motor control interventions. The history gives insight regarding medical management that might be necessary as adjunct interventions to physical treatment. Features that may guide medical management include behavioral health (occupational health/psychological interventions), poor sleep (sleep education/medication), quality and distribution of pain recognized as neuropathic (medication), and recurrent soft tissue complaints (interventional procedures).

Some patients benefit from medication to manage symptoms and to enable performance of physical treatments to reach their potential. Decisions about the need for and type of medications[3] are influenced by the time course of LBP, the distribution and quality of pain, the underlying pain mechanism (eg, central, neuropathic, nociceptive), the nature of provocative activities, sleep interference, and the patient's beliefs, experiences, and expectations. A scheduled medication regime may accomplish adequate pain control for the patient to participate in an active physical therapeutic program.

Overall, it is critical for health care providers to understand and consider the relative importance of factors beyond motor control to optimize the treatment approach and achieve successful long-term patient outcomes.[5,139] The importance of standardizing the diagnostic/subgrouping process cannot be overemphasized, as that will inform treatment decision making in a multidisciplinary framework.

CONCLUSION

This commentary reviewed convergence and divergence in approaches to LBP management that include consideration of motor control. The element common to all approaches is the focus on the need to reliably identify membership or nonmembership in validated subgroups of patients who have been shown to respond to treatment that eliminates pain when possible, optimizes alignment, restores and maintains full lumbar motion, and ensures that adjoining body regions demonstrate full and free movement. This focus is applied during exercise as well as in activities of daily living, fitness, and sports. The major differences between approaches relate to the baseline examination methods and the patient-specific treatments used to eliminate pain while restoring optimal alignment and movement.

No evidence supports one treatment approach over another. However, the reliable identification of members of subgroups for which there are predictably effective subgroup-specific treatments begins the process of identifying standardized treatment for members of each subgroup. By identifying areas of convergence/divergence and acknowledging existing literature that validates subgroups, we hope these insights can provide guidance to clinicians regarding which approach will serve their patients best.

This information can also provide a platform for teams to work together to consider hybrid approaches tailored to the individual patient for a focused progression, based on presentation and response. Benefit can be gained by improved communication and increased collaboration between colleagues in multiple disciplines to manage aspects of the multifaceted presentation of LBP (eg, specialist psychological intervention), when needed, and to facilitate treatment approaches that include consideration of motor control (eg, appropriate analgesia). ◉

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[CLINICAL COMMENTARY]

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Hybrid Approach to Treatment Tailoring for Low Back Pain: A Proposed Model of Care

In a perfect world, a treatment for low back pain (LBP) would have a large effect size—that is, it would be effective for most patients—and could be applied simply and cheaply. Though such treatments have been identified for some conditions, few have been identified for LBP. When applied generically to individuals with LBP, exercise,[61] manual therapy,[12] psychology-based treatments,[21] pharmacological agents,[63] and surgery[29] have a small to no effect size. There are 3 options for progress: we can accept a small effect size and continue with nontargeted treatments; we can continue to search for the elusive treatment that will be effective for most patients; or we can accept that LBP is a complex condition and test methods with potential to optimize the allocation of treatments to improve the effect size.

Low back pain is a complex and heterogeneous condition that has considerable variation in its presentation and the underlying mechanisms of symptom development and progression. An enormous body of literature describes biological, psychological, and social features that explain some individual variation. In the biological domain, variation in tissue pathology,[53] tissue loading by strategies of motor control (posture/alignment, movement, and muscle activation),[62] pain neurobiology (eg, central and peripheral sensitization),[44] immune system responses,[34] changes in brain structure and behavior,[43] and so on have been implicated. In the psychological domain, there is equivalent diversity of presentation, with variation in features such as pain coping,[42] self-efficacy,[11] pain catastrophization,[54] fear avoidance,[4] kinesiophobia,[73] depression,[42] anxiety,[37] distress,[37] and pain behavior,[37] and all have different implications for treatment. The social domain is equally diverse, including features such as job satisfaction[28] and social support.[42]

From one perspective, this diversity invites great optimism, as many features are identifiable and potentially modifiable, providing potential for intervention tailoring. From another, such variation encourages considerable doubt, as comprehensive assessment across all domains would be time consuming, cumbersome, and unworkable. One path is to identify and test methods to simplify and target this decision-making process. Methods to target care have been presented, but none alone has yet achieved large effect

SYNOPSIS: Various approaches have been used to guide the treatment of low back pain. These approaches have been considered in isolation and often tested against each other. An alternative view is that a model of care that involves a hybrid approach may benefit patients with low back pain. This commentary considers the potential benefits of a sequentially applied hybrid approach for treatment tailoring to optimize resource allocation to those most likely to require comprehensive care, and then decision making toward treatment paths with the greatest potential for success. In a first step, a prognosis-based approach, such as the Subgroups for Targeted Treatment Back Screening Tool (STarT Back), identifies individuals likely to require greater resource allocation. Although a clear path is indicated toward simple and psychologically informed care for the low- and high-risk groups, respectively, there is limited guidance for the large medium-risk group. For that group, the hybrid model provides a stepwise path of additional methods to guide treatment selection. This includes subgrouping based on pain mechanism to guide priority domains for the next phase, which includes tailoring of psychological and movement-based approaches. Motor control approaches to exercise would be indicated for individuals with medium risk and a nociceptive pain mechanism, with treatment guided by detailed assessment via one of several paradigms. Psychologically informed treatments are tailored to those with medium risk and a predominantly central pain mechanism, guided by detailed assessment of psychosocial features. A hybrid approach to a model of care could simplify treatment selection and take advantage of the benefits of each method in a time- and cost-efficient manner. *J Orthop Sports Phys Ther 2019;49(6):453-463. Epub 13 Feb 2019. doi:10.2519/jospt.2019.8774*

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sizes. The objective of this commentary was to propose a model of care for LBP that includes a hybrid of several methods to target treatments. The proposed model of care includes stratification to guide the overall strategy and intensity of care, identification of the pain mechanism to guide physiological/psychological targets for management, and subgrouping based on movement and/or psychology to guide detailed components of care, when appropriate.

Models to Guide Treatment Allocation

Two primary models have emerged to guide allocation of treatment, and these have generally been considered in isolation. These models are broadly defined as (1) methods to stratify care based on prognosis (identification of prognostic variables), and (2) methods to allocate treatments based on subgroups expected to respond to specific treatments (identification of treatment moderator variables).[24] For the former, questionnaires such as the Subgroups for Targeted Treatment Back Screening Tool (STarT Back)[23] and the Örebro Musculoskeletal Pain Screening Questionnaire[38] have been used. For the latter, methods have been proposed to subgroup patients based on predicted response to specific treatments,[13] underlying pain mechanisms,[64] features of movement/posture/muscle activation,[27] and pathology/diagnosis.[53] Although some research shows that treatment targeting can reduce costs (STarT Back) and improve outcomes when specific groups are compared,[71] it has not yet achieved substantial gains when applied to a general LBP group. It is the premise of this commentary that although each alone has strengths and limitations, significant gains may be achieved by combining these treatments into a hybrid model of care. Consideration of this hybrid model requires understanding the separate approaches to treatment targeting.

Stratified Care for LBP One approach to simplify treatment selection involves stratification of patients into groups that require more or less comprehensive treatment, such that most treatment resources are allocated to those with greatest need. This is the basis of the STarT Back approach (**TABLE 1**).[23] This questionnaire is derived from initial work that identified features related to poor prognosis, with preference for simple assessments of potentially modifiable features.[23] Individuals allocated to the group with "low risk" for a poor prognosis receive minimal care, based on reassurance and advice regarding activity (**TABLE 1**). The group at "high risk" for a poor prognosis (primarily based on negative psychological features) receives intensive psychologically informed treatment. Individuals in the "medium-risk" group are allocated to multimodal physical therapy, which the scheme argues should be applied according to clinical practice guidelines[48,56] at the discretion of the treating clinician.

Care applied according to this approach is more cost-effective (greater mean health benefit of 0.039 additional quality-adjusted life-years), but effect sizes remain small.[25] This approach has potential to guide treatment to those who need it most, with some guidance for the type and amount of treatment, but there are limitations. Although the STarT Back tool's accuracy for prognosis has been challenged,[33] a greater concern is the limited clear guidance regarding decision making for the medium-risk group, whose members make up approximately 46% of patients.[25] For this group, the question remains: "What is the best way to guide tailoring of intervention?"

Treatment Subgroups in LBP Treatment tailoring to subgroups assumes that patients with similar presenting features can be identified and that treatments can be guided with a high probability of efficacy

TABLE 1 Suggested[25] Interventions Prescribed According to STarT Back Subgroups*

Subgroup	Treatment
Low risk: single session for reassurance and advice	Promotion of appropriate levels of activity, including return to work Reassurance to address concerns related to back pain and any resulting loss of function Address uncertainty about medication, further investigations, prognosis Video and book to reinforce messages Advice about local exercise venues and self-help groups
Medium risk: standardized physical therapy to address symptoms and function using evidence-based treatments	Restore function and target physical characteristics to reduce back-related disability Address moderate levels of psychological prognostic indicators Included interventions: advice and explanation, reassurance, education, exercise, manual therapy, and acupuncture Excluded interventions: bed rest, traction, massage, and electrotherapy
High risk: psychologically informed physical therapy to address physical symptoms and function and psychosocial obstacles to recovery	Cognitive behavioral principles to address unhelpful beliefs and behaviors Physical treatment modalities (exercise and manual therapy) integrated with psychologically informed techniques to provide a credible explanation for symptoms, reassurance, education, and collaborative goal setting Problem solving, pacing, graded activity, and relaxation Focus on low mood, anxiety, pain-related fear and catastrophizing Promotion of appropriate levels of activity, return to normal activities, and management of future back pain recurrences Address patient expectations about prognosis and implications for function Emphasis on self-management Advice about sleep and work Return-to-work plan

Abbreviation: STarT Back, Subgroups for Targeted Treatment Back Screening Tool.
**The initial clinical session for all groups included assessment of potential serious pathology (red flags); neurological examination (reflexes, sensation, and muscle power); symptom history, concerns, and treatment expectations; and a brief examination of back pain movements and a screen for hip pathology.*

to those subgroups. It is generally considered that subgroups must (1) be definable/identifiable, (2) have mutually exclusive categories, (3) have improved outcomes when treatments are applied according to subgroups, and (4) be simple to implement or have high benefit if implementation is more costly and/or complicated.[15]

Several methods to subgroup patients have been proposed. These emphasize the biological features of LBP, specifically physical features (eg, provocation or relief by specific movements), with varying degrees of validation and differences in underlying philosophy (**TABLE 2**). Treatment-based classification identifies individuals predicted to respond to 1 of 4 treatments.[13] Randomized clinical trials (RCTs) have shown that individuals allocated to a particular subgroup have better outcomes if they receive the aligned rather than the nonaligned treatment.[6] Mechanical Diagnosis and Therapy evaluates the response to repeated loading and uses this information to allocate patients to different subgroups.[46] Randomized clinical trials show better outcomes for matched treatments.[66] Movement system impairment proposes that pain is caused and maintained by suboptimal tissue loading from postures and movement patterns.[60] A recent RCT showed better outcomes with matched treatment than with general exercise.[70] Cognitive functional therapy began with identification of movement patterns in LBP[51] and has evolved to include increasing focus on behavioral psychology.[52] An RCT showed that treatments aligned to some subgroups are more effective than control interventions for specific presentations of LBP.[71] Motor control training involves individualization of treatment based on features identified in the assessment, using a clinical-reasoning approach.[27] Randomized clinical trials show that baseline clinical features can predict patients with greater response.[14,41,69]

Another biological feature used to subgroup patients involves identification of the underlying pain mechanism.[35,49,64,75] Despite some divergence in opinions, there is broad consensus that 3 primary mechanism classes underlie pain presentations: pain maintained by "nociceptive," "central," or "neuropathic" inputs. Key characteristics, presumed mechanisms, and potential differences in treatment are presented in **TABLE 3**. Although there may be overlap between pain classifications (eg, combined nociceptive and central sensitization mechanisms), most aim to identify the predominant mechanism.

Subgrouping approaches have also been proposed to consider differences in psychosocial features. These methods include subgrouping based on the West Haven-Yale Multidimensional Pain Inventory,[8,68] features such as fear avoidance and distress profiles,[3] and clusters based on latent class analysis.[57]

Limitations of Isolated Application of a Subgrouping Method From an optimistic perspective, the various subgrouping methods to assist clinical decision making regarding treatment planning provide movement toward treatment tailoring and away from the oversimplified view of LBP as a homogeneous condition. Numerous studies confirm that with sufficient training, clinicians can identify subgroups,[20] and some treatments are efficacious when matched to specific subgroups.[9] For example, for patients with pain provoked by postures/movements, tailoring treatment to modify specific features of posture/movement is effective.[71] Patients allocated to a subgroup respond better to a matched than to an unmatched intervention: the odds of a successful outcome among patients who were positive on a prediction rule and allocated the selected treatment were 60.8 (95% confidence interval [CI]: 5.2, 704.7) and only 2.4 (95% CI: 0.83, 6.9) for those who were negative on the prediction rule.[9] Patients who respond favorably to repeated loading respond better to matched than to unmatched intervention (standardized mean difference for reduction of back pain, 1.0; 95% CI: 0.6, 1.3).[66]

Yet, other studies show no benefit. Patients received no greater benefit from matched versus unmatched psychologically informed treatments.[3] From another perspective, stratification has reduced costs, but with small effect sizes. Thus, although subgrouping methods are identified as a research priority and several are promising, outcomes are not yet ideal for several reasons.

First, most methods fall short of consideration of the multidimensional nature of LBP. An increasingly diverse array of factors is linked to the development and persistence of pain. Many may be critical for LBP management but are not yet considered in subgrouping methods. Examples include sleep quality[67] and comorbidities.[19]

Second, and related to the first, most approaches are primarily monodimensional or place limited emphasis on issues outside the primary domain. Patients within a subgroup may be similar with respect to physical features of their presentation but differ in other domains (eg, psychosocial features, pain mechanism). For instance, patients in the treatment-based classification stabilization subgroup have fear-avoidance beliefs that range from very low to high.[32] Different treatment strategies may be required despite allocation to the same subgroup. This implies that patients require separate subgrouping for each domain. If subgroups are to be mutually exclusive, each combination would be a separate subgroup, multiplying the number of subgroups.

Third, a recent study that classified people according to multiple schemes observed that although some individuals are clearly aligned to the defined subgroups in a scheme, others are not because they have features of multiple groups.[30] For instance, overlap between subgroups is considerable when categorizing based on pain mechanism; features of central sensitization are common in most individuals with persistent pain, including those with nociceptive or neuropathic features. Perhaps it is neither possible nor necessary for groups to be mutually exclusive.

Fourth, although some subgroups have effective treatments (eg, directional

TABLE 2

Subgrouping/Treatment Allocation Methods That Include Consideration of Movement

Approach	Foundation	Treatment Allocation/Subgroups
Motor control training[27,59]	Clinical-reasoning approach that aims to train optimal control (balance between movement and stiffness) of the lumbopelvic region, primarily for individuals considered to have pain with ongoing nociceptive input. Training uses motor learning principles to address motor control features related to suboptimal tissue loading	Allocation of treatment based on assessment of • Posture/alignment • Movement • Muscle activation Consideration of • Breathing/pelvic floor function • Sensory function • Adjacent joints • Psychosocial features • Strength/endurance/cardiovascular fitness
Treatment-based classification[13]	Aims to allocate patients to subgroups based on predicted response to treatments	Specific exercise • Flexion • Extension • Lateral shift/sidegliding Manipulation Stabilization Traction
Mechanical Diagnosis and Therapy[46]	Aims to determine whether symptoms can be abolished or reduced through application of direction-specific, repeated lumbar spine movements or sustained postures. Syndromes differ by hypothesized explanation for symptoms/development	Derangement syndrome • Central and symmetrical • Unilateral and proximal to knee • Unilateral and distal to knee Dysfunction syndrome • Flexion • Extension • Lateral shift/sidegliding • Adherent nerve root Postural syndrome Other • Stenosis • Hip • Sacroiliac joint • Mechanically inconclusive • Spondylolisthesis • Chronic pain state
Movement system impairment[60]	Aims to identify the direction of alignment, stress, or spinal movement that elicits or increases symptoms based on the kinesiopathologic model, which hypothesizes that precision of joint movement is altered by repeated movements and prolonged postures associated with daily activities	• Rotation with extension • Rotation with flexion • Rotation • Extension • Flexion
Cognitive functional therapy[51,52]	Aims to identify underlying mechanisms that are considered to drive pain. Differentiation between specific and nonspecific conditions is based on radiological evidence. Differentiation is made between central (central sensitization) and peripheral (mechanical) pain mechanisms. For those with a peripheral pain mechanism, the relationship to movement is identified. Identifies psychosocial and/or lifestyle factors that contribute	Specific versus nonspecific Peripheral versus central pain mechanism Control disorder (pain provocation) • Multidirectional • Flexion • Lateral shift • Active extension • Passive extension Movement disorder (pain avoidance) • Flexion • Extension • Flexion with rotation/sidebending • Extension with rotation/sidebending Pelvic girdle pain • Form closure • Force closure

preference in Mechanical Diagnosis and Therapy[66]), others do not. Most methods include at least 1 subgroup with little guidance for treatment or poor prognosis.[30]

Taken together, these limitations suggest that a fresh approach is needed. A model of care for LBP based on a hybrid approach may be the solution.

Potential Benefit From Combining Approaches

The potential solution to many of these issues is to combine approaches into a single model of care. Some work has been done to this end, with some success. As an example, it is plausible that underlying pain mechanisms would influence the potential responsiveness to treatments that address movement/posture/muscle activation, as advocated by several subgrouping methods (**TABLE 2**). Movement-based treatments that aim to optimize tissue loading would be expected to have the most impact on pain maintained by an ongoing nociceptive input from suboptimal tissue loading. In contrast, when pain is maintained by central sensitization, there might still be gains from movement training—to provide healthy movement experience and to reinforce healthy behaviors—but specific modification of a movement/loading pattern would be less relevant. Thus, combined consideration of "motor control" and "pain mechanism" for treatment selection could improve treatment matching.

Preliminary evidence from 2 recent RCTs supports combined approaches.[41,71] In both trials, patients were managed with a movement-based approach to optimize tissue loading based on assessment, and both considered pain mechanism. One trial had a large effect (eg, improvement on the Oswestry Disability Index of 13.7 points; 95% CI: 11.4, 16.1 points) but only included patients with a clear relationship between pain and movements/postures.[71] The second trial did not select participants on the basis of pain mechanism, but baseline assessments were conducted for planned post hoc analysis of effect modification.[41] Although there was no difference in overall outcome between patients managed with tailored motor control training intervention and those

TABLE 3 Classification by Pain Mechanism

	Nociceptive	Neuropathic	Central/Central Sensitization
Definition[49,64,75]	Pain maintained by ongoing nociceptive input from the peripheral nociceptive neurons. May be provoked by mechanical loading (postures, movements, muscle activation), chemical, or thermal stimuli	Pain associated with a lesion or dysfunction of neural structures (central or peripheral)	Pain maintained by neurophysiological processes associated with amplification of neural signaling
Key features[64]	• Localized to a specific body region • Responds in a predictable manner to postures and movements • Provoked pain proportional to tissue loading • Usually intermittent and sharp	• History of nerve/neural injury or pathology • Pain provoked by movements and postures that compress/move/tension a nerve • Dermatomal distribution of pain • Pins and needles/numbness • Muscle weakness • Burning, shooting, electric-like pain	• Diffuse area of pain/tenderness • Inconsistent relationship to movement and postures • Intensity disproportionate to provoking posture/movement • Disproportionate to that expected from injury mechanism • Association with maladaptive psychological features
Questionnaires include	...	• pain*DETECT*[16] • ID Pain[55] • LANSS[2] • Neuropathic Pain Scale • Neuropathic Pain Questionnaire[36] • DN4[5]	• Central Sensitization Inventory[45] Assessments for psychological features • DASS-21[39] • FABQ • PCS[65] • CES-D[58]
Clinical examination	• Subjective examination of pain features • Response to tests of movement and posture	• Subjective examination of pain features • Tests to confirm nerve/neural pathology • Nerve conduction tests • Imaging • Neurological examination: reflexes, sensation, muscle strength • Neurodynamic tests: assess loading of neural tissues and their relationship to postures and movements	• Subjective examination of pain features • Quantitative sensory testing • Temporal summation • Conditioned pain modulation • Pain thresholds • Nociceptive withdrawal reflexes

Abbreviations: CES-D, Center for Epidemiologic Studies-Depression Scale; DASS-21, 21-item Depression, Anxiety and Stress Scales; DN4, Douleur Neuropathique 4; FABQ, Fear-Avoidance Beliefs Questionnaire; LANSS, Leeds assessment of neuropathic symptoms and signs; PCS, Pain Catastrophizing Scale.

managed with behavioral therapy (graded activity), when baseline features were considered, patients who scored high on a questionnaire regarding features that provoke and relieve pain responded better to motor control training, and those with a low score had a better outcome with graded activity.[41]

Taken together, these studies illustrate that a multicomponent (multistep) subgrouping approach might improve decision making and outcomes. Similar gains may be made from detailed assessment for individuals where it is warranted, based on other biological, psychological, and social features. Although one might argue that this consideration of multiple domains in treatment selection is simply good clinical reasoning, the alternative view is that formalizing the process into a model of care with structured decision steps would aid implementation, teaching, and consistency.

Proposed Model of Care Based on a Hybrid Subgrouping Approach

If no single approach provides the answers, one strategy would be to undertake a separate assessment of all domains and then disentangle the likely effective treatment plan. This is not feasible (in terms of time or resources), is unwarranted for many patients, and is too complex to implement. The alternative is to combine approaches into a stepwise model of care that includes an initial step to stratify individuals in order to allocate time and resources to those who are likely to require more intensive care (and simple care to the low-risk group), with several layers of assessment within the mid- and high-risk groups to provide more comprehensive decision making that combines features of multiple subgrouping methods in parallel and in series, and to guide treatment selection based on biological, psychological, and social features. The **FIGURE** presents such a proposed model of care to guide management of LBP.

Stratification to High, Medium, and Low Risk The first step involves identification of the risk profile to triage patients into low-, medium-, and high-risk groups. The STarT Back tool[23] provides an evidence-based model to undertake this step, although other options are available.[38]

Low-Risk Group: Treatment Path—Reassurance, Education, and Staying Active As advocated by the STarT Back tool, the low-risk group is managed with a brief intervention that includes advice and education to reassure patients that LBP is a "normal" part of life with a high likelihood of recovery (**TABLE 1**, **FIGURE**). Advice to stay/become active is provided, along with reinforcement of healthy behaviors. Care may be facilitated with the use of web resources tailored for this purpose, such as www.MyBackPain.org.au, which aims to empower individuals to make informed decisions about care and provides resources to engage in such aspects as pain coping skills training and treatment choices.

High-Risk Group: Treatment Path—Psychologically Informed Care Patients are allocated to the high-risk group based on psychosocial features that indicate unhealthy pain beliefs, attitudes, cognitions, and behaviors that must be addressed with treatment.[23] A comprehensive assessment of psychosocial features guides treatment (**TABLE 1**). Psychological treatments can include behavioral therapies (to modify behaviors), cognitive behavioral therapies (to address cognitions about pain), or acceptance-based therapies that encourage return to function despite pain.[72] Treatments have been specifically developed to address features such as fear avoidance,[21] pain coping skills,[1] and education regarding pain physiology/neurobiology.[47] Movement training would be relevant for this group as a component of physical activation to reinforce healthy behaviors, but with care regarding language to ensure that explanations do not contradict the objectives of the psychologically informed treatment (eg, "stabilize" and "protect" would reinforce the biomedical explanation for LBP). Consideration of pain mechanism (see following section) would inform whether nociceptive mechanisms remain relevant. In that case, modification of movement/posture/muscle activation may require consideration.

Medium-Risk Group—Detailed Assessment of Pain Mechanism to Guide Treatment Allocation to the medium-risk group indicates that detailed assessment is required to guide treatment. For this group, the potential benefits of hybrid subgrouping are most apparent. As a first step, clinical interpretation of the primary pain mechanism (nociceptive, neuropathic, or central sensitization) underlying the maintenance of pain is required (**TABLE 3**, **FIGURE**). This step provides guidance regarding which domains should be prioritized in assessment/treatment.

In the absence of a gold standard, the primary pain mechanism is identified based on clinical characteristics of pain. Work is progressing for tools to undertake this step.[49,64] Most advocate a combination of interview and questionnaires, with clinical examination of pain system function advocated by others (eg, quantitative sensory testing, temporal summation, conditioned pain modulation[18]). Questionnaires for identification of central sensitization[45] and neuropathic pain[16] have been developed. Although sensitive to detection of these pain mechanisms, they include questions related to features such as severity of pain that are not specific and are unlikely to aid differential diagnosis. This may explain the suspiciously high prevalence of neuropathic pain in some musculoskeletal conditions (eg, osteoarthritis[26]). The alternative interpretation is that these studies identify a group with more severe pain or features of central sensitization.

A difficulty with differentiation of pain mechanisms is that they overlap; for example, most individuals with prolonged nociceptive or neuropathic pain would have central sensitization. Thus, biologically, pain mechanism groupings are not mutually exclusive. This does not limit the utility of this approach; however, overlap between mechanisms would influence some elements of treatment selection.

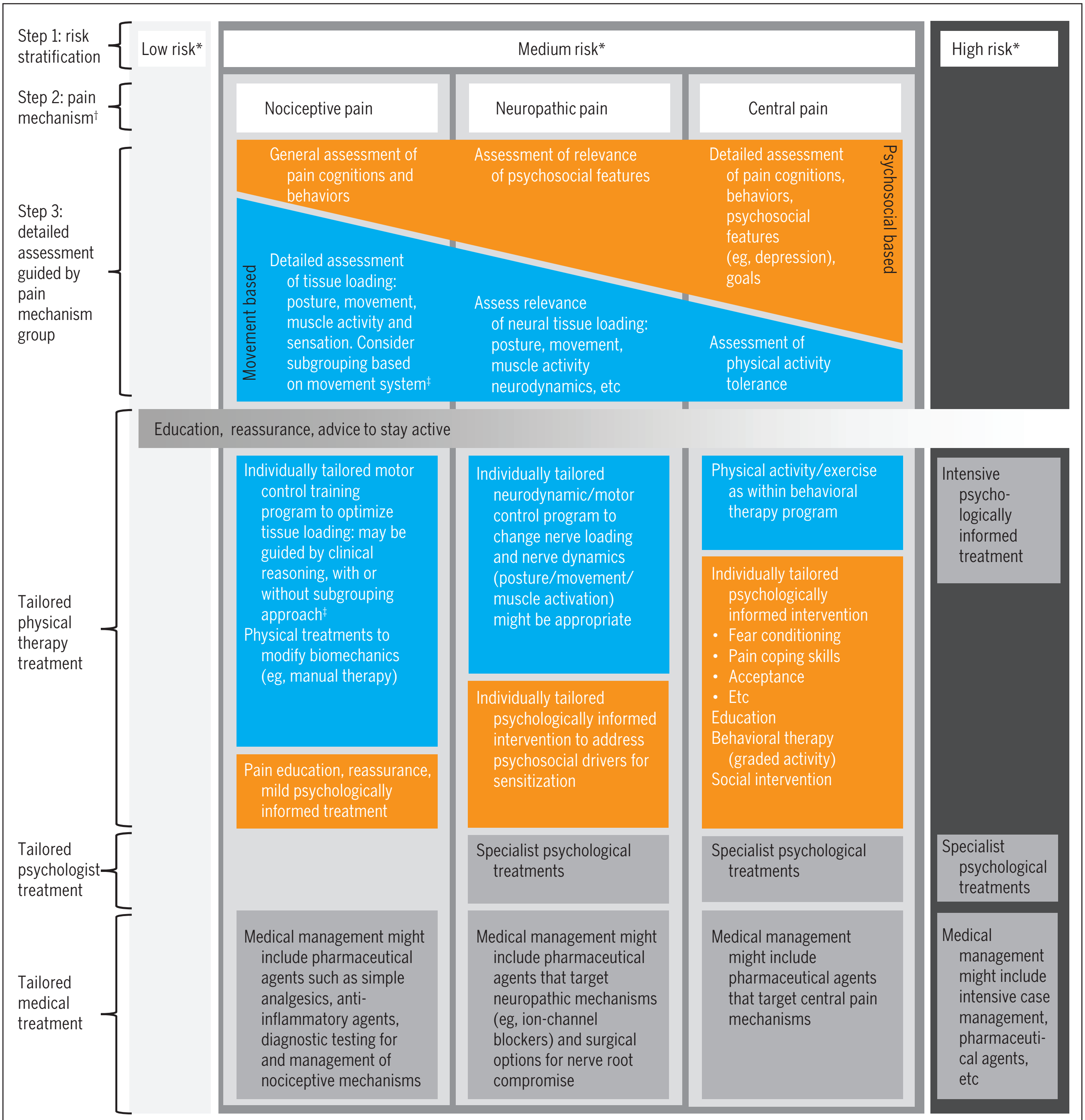

FIGURE. Proposed model of care for management of low back pain based on a hybrid of subgrouping methods. The initial step involves stratification/subgrouping using a risk prognosis method (eg, STarT Back). Treatments for low- and high-risk groups are implemented according to this allocation. For the medium-risk group, further assessment identifies the predominant pain mechanism to guide balance of movement versus psychosocial-based assessment and treatment selection. Treatments are tailored to the individual based on assessment. For each pain mechanism category within the medium-risk group, the suggested assessment and treatments are highlighted by their organization to columns under the pain mechanism title. The link between assessment and treatment is highlighted by use of similar colors. The relative bias toward assessments and treatments is indicated by the space allocated (eg, decreasing space allocated to assessment of movement when moving from nociceptive to central sensitization pain). Integration with medical and psychological management can also be guided by subgrouping. *Guidance for content of treatment allocated by risk group, with clear guidance for low- and high-risk groups, is presented in **TABLE 1**. †Suggested criteria for differentiation of pain mechanisms are presented in **TABLE 3**. ‡Possible methods for assessment and individualization of treatment based on motor control features are presented in **TABLE 2**. Abbreviation: STarT Back, Subgroups for Targeted Treatment Back Screening Tool.

How could recognition of pain mechanism guide treatment selection? In a multidisciplinary framework, this information can guide allocation of treatments as diverse as pharmacological management (many drugs have effects that are specific to pain mechanisms[10]), psychological interventions (eg, fear conditioning, pain coping skills), tailored pain education, and physical interventions across a spectrum from general physical activity to individualized motor control training. As a general guide, treatments and assessments would have different biases for each pain mechanism group; for example, nociceptive pain would imply greater bias toward assessment of physical features, and central pain would imply a bias toward assessment in the psychological domain (**FIGURE**). **TABLE 1** includes consideration of management by medical and psychology disciplines to provide context for where their expertise may be most critical, as well as some suggestions for tailoring. This table is not intended to provide comprehensive guidance for these roles.

Central Sensitization When pain is primarily maintained by central sensitization, existing theory (and some evidence) argues for an approach biased toward psychologically informed treatments,[50] similar to that advocated for the high-risk group, to desensitize and activate the patient. Psychological features may regulate/moderate the biological processes that underlie central sensitization, but the mechanisms by which psychological management reduces sensitization are diverse. Psychologically based treatments may require involvement of a psychologist or physical therapist with training in this area. Movement training as an element of a behavioral intervention may be important to changing behavior and cognitions about pain (graded activity[40]). The goal in such an intervention may be to provide healthy movement experience and aid return of function. Attention to other lifestyle interventions, such as sleep hygiene, may be required. For medical management, certain classes of drugs are advocated[50] but may not be the only or best solution.

Nociceptive Pain There is considerable debate regarding the relevance of ongoing nociceptive input. There is not a one-to-one relationship between nociceptive input and pain[74]; this is not how pain is experienced. Pain is a product of the nervous system, generated based on all information received as well as other cognitive, emotional, and biological processes. It is well known that nociception is neither required nor sufficient to explain pain.[74] Yet, it is reasonable to expect that for at least some individuals with ongoing pain, the pain experience may be maintained by ongoing nociceptive input. This is not to say that pain can simply be relieved by removal of the nociceptive input, but that this is likely to be an important element of recovery in those individuals. Studies that have successfully reduced pain by application of local anesthetic provide some support,[17] but this must be considered with respect to the potential beneficial effects of simply "taking action" to relieve pain, which may explain the relief of pain from peripheral analgesia. Recent work shows that people report reduced pain despite no reduction of nociceptive input when they "take action" to protect that painful part (Bergen et al 2018, unpublished data). If a nociceptive element is presumed (**TABLE 3**), then a detailed assessment of how the person uses his or her body and how this affects the pain experience is likely to be required to identify relevant movement/posture/muscle activity. Psychological features also require consideration, but generally with less emphasis (**FIGURE**).

The identification of suboptimal tissue loading strategies is likely to be most relevant to individuals in the medium-risk group with nociceptive pain. An underlying assumption of motor control approaches is that pain is maintained by ongoing nociceptive input from loading of tissues[27] (other than neural tissue; see below). This would be expected to be highly individual. As such, patients would require detailed assessment of how they use their body to identify features of motor control that might be related to suboptimal tissue loading. Comprehensive assessment would require consideration of movement/posture/muscle activation.[27] As discussed above, multiple schemes aid this assessment (**TABLE 2** presents several options; it is not the intention of this commentary to recommend one over another, but there may be value to drawing principles from several approaches because an individual may present in a manner that suits one approach more than another[30]). It is at this point that it makes sense to consider movement-based subgrouping and clinical-reasoning methods to aid the identification of relevant motor control features to target treatment.

Each movement-based subgrouping approach involves patient interviews and a series of specific postural assessments and movement tests to identify the features that provoke and relieve symptoms.[22,31] As described above, each approach has a different foundation and applies a different method to identify the features that are considered relevant for the clinical presentation and may become the targets for treatment (**TABLE 2**). There is convergence and divergence between approaches.[22] In brief, most include a component of cognitive modification of movement/posture/muscle activation, with differing emphasis, to modify the strategy of tissue loading (**TABLE 2**). Some suggest passive treatments[9] or repeated movements,[66] whereas others have a stronger bias to optimize posture/movement/muscle activation.[27] As yet, there is no clear basis to use one method over another. A recent review suggests that an approach that combines schemes is likely to be helpful, as some patients cannot be clearly categorized within one scheme or fail to respond as expected to the aligned treatment.[30]

Ultimately, the choice of a subgrouping or clinical-reasoning model depends on the skills, training, and preference of the clinician and the preference of the patient. Ideally, clinicians would have experience with multiple systems so that they would possess the flexibility to adapt the assessment and training to match the individual.

As mentioned above, many individuals allocated to the primary nociceptive pain group will also present with some signs of central sensitization or neuropathic mechanisms. In those cases, consideration of features such as psychological elements may be required, as described earlier.

Neuropathic Pain For individuals presumed to have pain maintained by ongoing neuropathic mechanisms, treatment selection can be multifactorial and requires a balanced consideration of physical and psychological features (**FIGURE**). As with individuals with central sensitization, psychological/education interventions aimed at desensitization would be helpful. This approach might be combined with pharmacological management.[50] Training of posture/movement/muscle activation may be relevant for individuals with peripheral neuropathic pain, where pain is provoked by nerve loading. Neurodynamic assessment may reveal specific features to address and guide treatment selection.[7]

Critical Appraisal of a Hybrid Model of Care

The proposed hybrid approach combines several subgrouping methods for treatment selection using a method that applies them in a stepwise manner (eg, identify risk; if allocated to the medium-risk group, then identify the underlying pain mechanism; if allocated to the nociceptive pain group, then identify the "movement" subgroup). Although the hybrid approach is logical and evidence has been presented for some of its components, it cannot be assumed to be more effective than standard care or the separate application of any of its combined approaches. High-quality RCTs are required to test the model. This could take several forms, such as a head-to-head comparison of the hybrid model of care versus 1 element of the approach or versus standard care. Alternatively, it could involve a complex design that compares approaches of differing levels of complexity.

Further development is required to refine and validate differential diagnosis of primary pain mechanisms. The final model might include a combination of clinical pain features, psychological features, quantitative sensory testing, and response to a simple physical examination (eg, movement or posture).

There may be value gained from further refinement and, perhaps, hybridization of the subgrouping and clinical-reasoning models for identification of loading features related to pain presentation. Likewise, refinement of optimal methods for modification of motor control is needed. A major issue in any exercise intervention is adherence to training. Use of behavior-change methodology is likely to be required, but this involves training of clinicians and development of tools for assessment of individual needs to adopt a behavior, as well as methods to address them.

The potential implications for health service utilization and workforce issues require consideration. A major intention of the model is to allocate more comprehensive services to those who need them, thus avoiding overtreatment of individuals who can be treated with a less intensive approach. Costs savings would be predicted based on previous data[25] but require evaluation. For the workforce, the major implication is adequate training to implement the steps in assessment and treatment and the opportunity for interdisciplinary involvement, as required and recommended by the model.

CONCLUSION

This commentary aimed to bring together several contemporary models that have been devised and applied to simplify the task of decision making in the management of LBP. Rather than advocate for a single approach, the purpose of this paper was to highlight the logic behind stepwise application of several methods to identify patients who would benefit from approaches targeted to different domains. Critically, the approach highlights the path of decision making that would lead to the decision to apply a movement-based approach.

Each method combined into the hybrid model has pros and cons, and this model of care has been developed in an attempt to take advantage of the most promising aspects of each and combine them into a model that guides allocation of more comprehensive management to patients who need it the most, followed by guidance related to priorities for assessment and management. The proposed model addresses key issues that challenge existing methods, such as the allocation of time-consuming comprehensive care that would not be feasible (and would probably be unnecessary) to apply to all, examines mechanisms to consider multidimensional aspects of presentation and non–mutually exclusive groups, and provides balanced consideration of the biological and psychosocial aspects of an individual's presentation. There appears to be sufficient foundation to consider testing such a model of care.

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Changes in Structure and Function of the Back Muscles in Low Back Pain: Different Time Points, Observations, and Mechanisms

SYNOPSIS: Spinal health depends on optimal back muscle performance, and this is determined by muscle structure and function. There has been substantial research evaluating the differences in structure and function of many back muscles, including the multifidus and erector spinae, but with considerable variation in results. Many studies have shown atrophy, fat infiltration, and connective tissue accumulation in back muscles, particularly deep fibers of the multifidus, but the results are not uniform. In terms of function, results are also somewhat inconsistent, often reporting lower multifidus activation and augmented recruitment of more superficial components of the multifidus and erector spinae, but, again, with variation between studies. A major recent observation has been the identification of time-dependent differences in features of back muscle adaptation, from acute to subacute/recurrent to chronic states of the condition. Further, these adaptations have been shown to be explained by different time-dependent mechanisms. This has substantial impact on the rationale for rehabilitation approaches. The aim of this commentary was to review and consolidate the breadth of research investigating adaptation in back muscle structure and function, to consider explanations for some of the variation between studies, and to propose how this model can be used to guide rehabilitation in a manner that is tailored to individual patients and to underlying mechanisms. *J Orthop Sports Phys Ther 2019;49(6):464-476. doi:10.2519/jospt.2019.8827*

KEY WORDS: *acute back pain, chronic back pain, electromyography, imaging, multifidus muscle, rehabilitation*

Back muscle function is a prerequisite for optimal control of spinal stiffness and movement. Muscle structure affects muscle function, function affects structure, and pain/injury impact both, as is common for back muscles in low back pain (LBP). In the short term, acute pain and nociceptive stimulation, in addition to injury-related afferent input, affect back muscle function. Conversely, altered back muscle function has been proposed to underpin LBP development and recurrence.[54] In the long term, ongoing effects of pain and inflammatory mechanisms exert additional effects on back muscle structure (eg, atrophy, muscle fiber change, fatty infiltration, reduced strength/endurance) and function, and vice versa. These complex bidirectional interrelationships could drive a circular process of persistent or recurring LBP.[32,38,54]

The interaction between pain, injury, and back muscle changes has many elements: different mechanisms, time dependencies, and relationships to structure, function, and outcomes. Results are variable and somewhat confusing. For context, it is necessary to consider the anatomy of back muscles, as most work identifies changes in some muscles but not in others, or focuses on specific components. Briefly, the multifidus lies medially and includes short/deep fibers that cross as few as 2 segments[79] (often referred to as the deep multifidus [DM][75,86]), with progressively longer/superficial fibers crossing up to 5 segments[79] (often referred to as the superficial multifidus [SM][75,86]) (**FIGURE 1**). Lateral to the multifidus are the longissimus and iliocostalis, which include lumbar[78] and thoracolumbar[78] portions (collectively referred to as the erector spinae [ES]). Functionally, the DM primarily provides compression, with a limited extension moment,[9] with relevance for segmental control,[75,86] whereas the SM and ES generate spine extension, and a lesser contribution to lateral flexion and rotation,[9] to move the spine or increased stiffness when cocontracting with antagonist muscles.[14,75,86] Further, recent work has shown that multifidus muscle fibers are shorter and arranged in tightly packed bundles,

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which creates a physiological cross-sectional area (CSA) that is greater than that of other paraspinal muscles. This muscle architecture enables large force generation over small excursion, which is consistent with a contribution to stabilization rather than spine movement.[114] All back muscles are important for coordination of movement and stiffness.[47]

This commentary reflects on contemporary theories regarding the relationship between pain/injury and change in back muscle structure and function in acute, subacute/recurring, and chronic/persistent LBP. A state-of-the-art understanding of back muscle changes is presented, along with interpretation of different underlying mechanisms at different time points and their differing implications for rehabilitation.

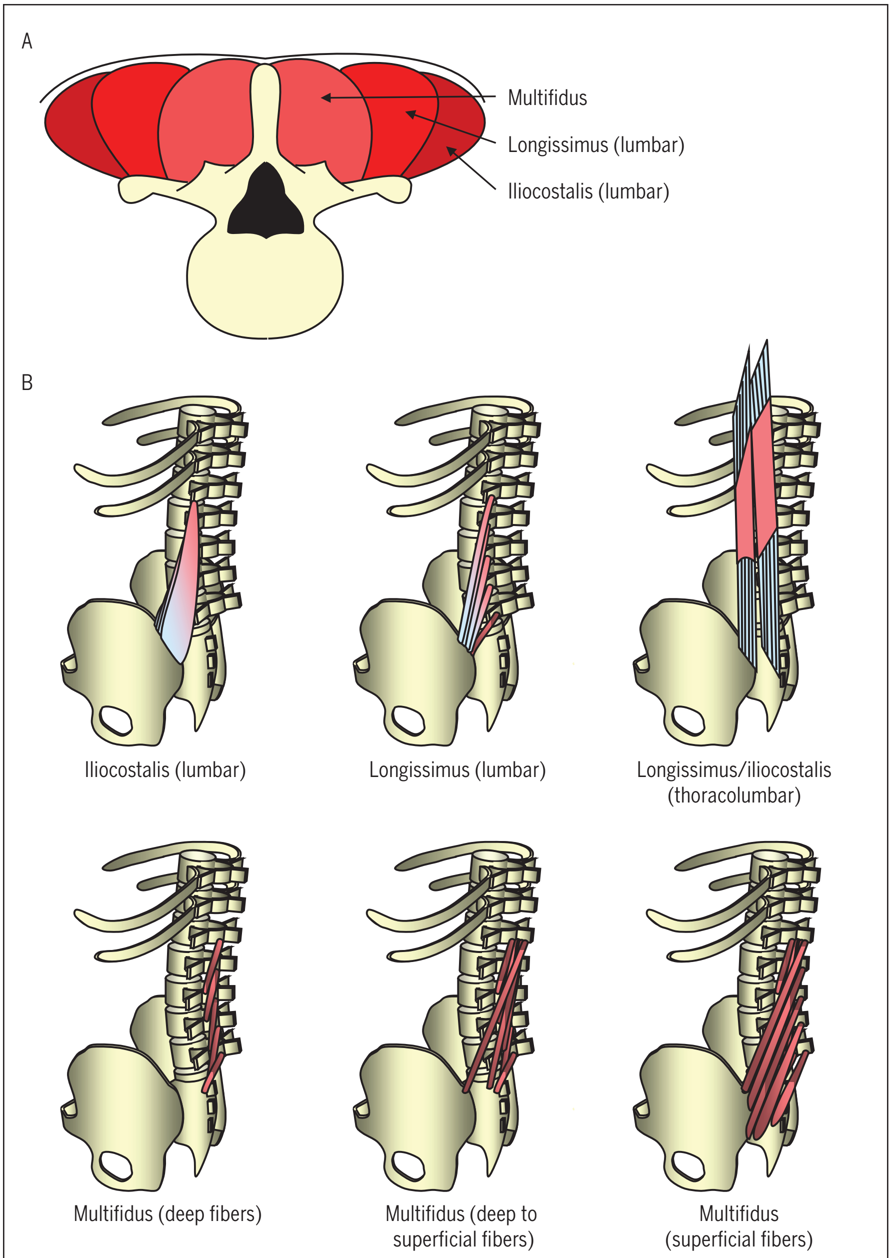


FIGURE 1. Anatomy of the back muscles. (A) Cross-section of back muscles at the L4 lumbar level. (B) Anatomy of multiple layers of back muscles.

Changes in Back Muscle Structure and Function in Acute LBP and/or Injury

Acute Clinical LBP In clinical LBP, muscle structure and function could be impacted by a range of biological and/or psychosocial influences. Biological influences relate to pain/nociceptive input, and afferent input related to tissue injury (eg, intervertebral disc [IVD] lesion), even in the absence of pain.[49,100] Although clinical studies provide important insight, interpretation is complicated by the heterogeneous nature of clinical LBP, and their cross-sectional design precludes interpretation of causal mechanisms.

Few studies have focused on acute LBP, but there is some evidence of localized reduction in CSA of the multifidus during an episode of acute unilateral LBP on the painful side and at the clinically determined level of symptoms.[44] This was replicated in an animal injury model[45] (see Animal Studies of Tissue Injury below). It has been necessary to study the effects of experimental pain and animal models of injury to understand the nature of changes to acute stimuli, the causal pathways, and the mechanisms.

Human Experimental Pain Studies Experimental procedures to induce nociceptor stimulation/pain and the threat of pain in humans enable investigation of causal relationships for muscle structure/function changes. Although not replicating all aspects of the pain experience, intramuscular hypertonic saline injection induces a deep muscular ache and shares features of clinical musculoskeletal pain,[103] lasting for 3 to 10 minutes.[41]

Altered back muscle function during experimental LBP has been examined during tasks involving automatic control of spinal posture, using paradigms adapted from clinical studies,[53,75] with variable results. During arm elevation in prone, multifidus activation, estimated from increased muscle thickness using ultrasound, was less with than without experimental LBP.[67] When noxious input was applied unilaterally at a single level, reduced muscle thickening occurred bilaterally at multiple levels.[31] Intramuscular multifidus electromyography (EMG) showed reduced activation during forward weight shift in standing.[66] In contrast, multifidus EMG was greater on both sides when an arm was lowered from 90° of shoulder flexion in standing,[66] and greater (and/or initiated earlier) during rapid arm flexion.[52,88] Different results are obtained when pain is anticipated; baseline SM EMG increases in advance of arm flexion, and DM EMG is delayed and decreased.[87] During walking, Arendt-Nielsen et al[3] showed increased ES EMG during swing (the phase of low ES activation) and decreased ES EMG during stance (the phase of high ES activation). Together, these data show task-specific changes in the multifidus and ES. One interpretation is that when challenged by acute noxious input, multifidus and ES activation is decreased when the muscles produce spine extension (prone arm elevation, forward weight shift, stance phase), but increased when the activation prevents spine motion (rapid arm flexion, swing phase).

Experimental pain during dynamic trunk movements reveals similar task-specific observations. Reduced trunk flexion velocity and range are accompanied by absence of expected ES relaxation at terminal flexion, but reduced ES EMG when it extends the spine to upright.[118] When contraction history is estimated from T2 shifts in muscle function magnetic resonance imaging (MRI), prone trunk extension (between 45° and 0° of flexion) induces lesser T2 shifts when the task is performed with than without experimental pain.[29] Although T2 rest values differed between the DM and SM (which might be explained by different muscle fiber composition[30]), pain similarly affected both muscle regions.[31]

Other trunk movements reveal less stereotypical outcomes. Although activation of the DM was not recorded, net muscle activation recorded with surface EMG electrodes over 12 abdominal and back muscles and spine stability estimated from an EMG-driven model were increased by experimental pain during slow forward/backward movements in upright sitting, but activation changes of the individual superficial trunk muscles (including the ES) varied between participants.[48] This interindividual variation was apparent despite an identical noxious stimulus (hypertonic saline injection into the right L4 longissimus) and similar evoked pain area and intensity, and appears consistent with interindividual variation in clinical LBP subgroups.[4,110] Interindividual variation is inconsistent with the stereotypical pain adaptation proposed by Lund et al.[73] Instead, changes appear to take advantage of the versatility of the complex trunk muscle system to enhance spine protection in response to acute noxious input using person-specific strategies, as predicted by contemporary models of pain adaptation.[54] High-load tasks have been found to have unchanged muscle activation (Danneels et al, unpublished data), which may indicate that no option for modification of the recruitment strategy is available if output is to be maintained, or that subtle individual variation was induced but not observed in group analysis.

The conclusion from studies of noxious back input is that back muscle activation is modified in acute LBP, but this varies between tasks and individuals. Although multifidus and ES activation has been reported to increase or decrease, when apparently inconsistent data are taken together, the data can be reconciled to imply a general goal to protect the spine. This phenomenon is characterized by increased activation of the ES (and some evidence of DM activation) when the spine is challenged into flexion or as part of a cocontraction, but by reduced multifidus (and some evidence of ES) activation when the task involves active extension of the spine (**FIGURE 2**). Further, an understanding of the mechanisms for task-specific differences has been provided by animal studies.

Animal Studies of Tissue Injury Animal studies enable investigation of the causal impact of tissue injury on muscle structure, although relative contributions from injury and pain cannot be differentiated. Effects from IVD or nerve injury have been studied (rabbit,[10] pig,[45] sheep[51]). Macroscopically, rapid multifidus atrophy was detected at 3 days after injury in pigs. Ipsilateral multifidus atrophy was localized to a single level after IVD injury but was multisegmental after nerve injury. Localized multifidus changes concur with clinical data in acute unilateral LBP.[44] Atrophy could not be explained by changes in water and fat that were observed at multiple levels[45] but was consistent with localized immediate reduction in excitability of spinal neural pathways, assessed using stimulation of the spinal cord.[49] This parallels reduced multifidus reflex responses to electrical IVD stimulation in pigs after infusion of physiologic saline into a facet joint.[59] Both observations could be explained by reflex inhibition, similar to that observed after knee injury[100] (**FIGURE 3**). In contrast, response of the multifidus to motor cortex stimulation increased after IVD lesion.[49] Whether multifidus activation is facilitated or inhibited depends on the balance between increased excitability of descending input from the brain and decreased spinal cord excitability. As this may differ between tasks and individuals, it is reasonable to speculate that this could explain diversity in response to experimental pain in humans; differences in the relative contribution of spinal and descending inputs to back muscle activation between tasks may shift the balance from inhibition to facilitation.

Summary In the acute phase, multifidus activation can be reduced or increased, depending on the task. The mechanism appears to be neural, through mechanisms including spinal reflex inhibition and increased descending drive. The observation of atrophy of the multifidus implies that, although multifidus activation may be inhibited or facilitated, the net effect of inputs to the multifidus in the acute phase is likely to be inhibition. Although many studies focus on the multifidus, some evidence points to variable changes in the ES.

Research supports a causal relationship between injury/nociceptive input and changes in muscle structure and function. These findings have several important implications for rehabilitation (**FIGURE 4**). First, treatments to reduce "drivers" of inhibition/facilitation may be warranted (eg, interventions to reduce pain and/or enhance tissue healing may be advocated—medication, manual therapy, etc).

Second, data from a randomized controlled clinical trial suggest that exercises aimed at gentle, precise activation of the multifidus are sufficient to restore multifidus size.[106] As this type of activation would not be sufficient to induce muscle hypertrophy, this implies that early muscle atrophy is not related to loss of muscle fiber mass, and simple activation to overcome inhibition is sufficient to restore muscle health. This is likely to require specific attention to the pattern of muscle activation used during the exercise, and simple extension of the spine is unlikely to be sufficient, as many different patterns of activation are available to extend the spine,[48] and these might not involve the multifidus. This would argue for an approach that specifically targets activation of the multifidus, such as a motor control approach for rehabilitation of back pain.[55]

Third, many current clinical practice guidelines recommend that patients not seek care for an acute episode of uncomplicated back pain and instead remain active. However, the potential for multifidus changes to recover with exercise but to fail to recover with general functional activity, and the potential for training to reduce recurrence of LBP (all shown in a small study of acute LBP and requiring reproduction),[106] provides a foundation to consider that benefit may be gained from early intervention to restore multifidus activation. This needs to be undertaken with an emphasis on optimizing spine health, with care not to instill a belief in the patient that the spine is "at risk," which may promote unhealthy attitudes

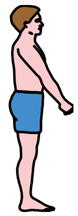
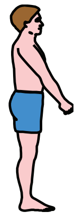
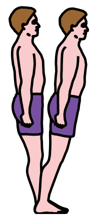
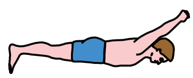
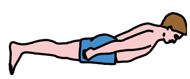
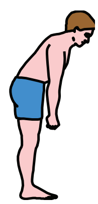
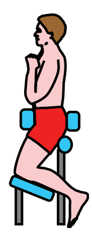
Task		Primary Role of Back Muscles	Change With Experimental Pain
Rapid externally triggered arm flexion in standing[52]		Anticipatory activity to counteract reactive spine flexion moment from arm acceleration	DM increased SM nonsignificant ES nonsignificant
Self-paced arm elevation and lowering in standing[66]		Elevation and lowering: counteract spine flexion from arm mass	Elevate to 90°: DM nonsignificant Lower from 90°: DM increased
Forward/backward weight shift[66]		Forward: spine extension to maintain upright trunk Backward: cocontraction with flexors	Forward: DM decreased Backward: DM nonsignificant during pain
Arm elevation in prone[67]		Activation to extend spine to aid arm elevation	DM decreased
Prone trunk extension[29]		Activation to extend spine	DM decreased SM decreased
Trunk flexion[118]		Lowering: relaxation at end flexion Elevation: activation to extend trunk	Lowering: no ES relaxation (ie, increased activation) Elevation: ES decreases during elevation
Walking[3]		Stance: activation during stance Swing: relaxation during swing	Stance ES decreased Swing ES increased
Slow trunk flexion and extension around neutral[47]		Cocontraction of flexor and extensor muscles to stabilize trunk around neutral	ES variable increased

FIGURE 2. Changes in back muscle function in acute experimental back pain. Summary of tasks tested, function attributed to the trunk muscles in these tasks, and changes that have been observed in muscle activation. Data support the proposal that adaptation in acute pain depends on the function performed by the muscle in a specific task. Abbreviations: DM, deep multifidus; ES, erector spinae; SM, superficial multifidus.

and catastrophizing (see Reeves et al[94] for review of the potential negative iatrogenic effects of messages patients interpret about impact of spine control). Although systematic reviews do not report superior efficacy of motor control training that includes rehabilitation of back muscles in acute LBP, this is based on the short-term impact of training on pain and function in a small number of studies.[95] It is plausible that the greatest impact of back muscle training on LBP is prevention of recurrence, as highlighted in 1 study[43] that requires replication in a larger group and comparison with other approaches.

Changes in Back Muscle Structure and Function in Subacute LBP and During Remission

Subacute and Recurrent Clinical LBP Of interest is the potential role for persistence of changes in back muscle structure and function after symptom resolution in LBP recurrence. Delayed DM (but not SM) activity has been reported during automatic control of spinal posture accompanying limb movements.[75] The DM was recruited as an extensor rather than undergoing non–direction-specific recruitment (a characteristic interpreted to imply a role in the fine-tuning of segmental control[47]), which suggests a change in function. During predictable trunk loading into flexion, DM EMG was less on both sides than in pain-free controls, with greater reduction on the previously painful side.[74] Superficial multifidus EMG increased earlier than on the previously painful side or in pain-free controls.[74]

This change in function and differentiation between regions of the multifidus is also apparent in dynamic trunk movements involving voluntary spine extension[26] and flexion.[28] During static/dynamic trunk extension, activity of the multifidus (without separate analysis of the DM and SM) estimated from the T2 shift with muscle function MRI was greater bilaterally at multiple levels during LBP remission, suggesting a change to extensor function.[26] During rapid-onset trunk flexion from upright sitting, EMG was analyzed from group and individual perspectives.[28] Flexor and extensor muscle cocontraction was higher throughout the task. Extensor muscle EMG was also higher when acting as an antagonist during flexion and when approaching the return to upright. Deep multifidus EMG was higher in the period before movement, but during flexion, participants with a history of unilateral LBP demonstrated

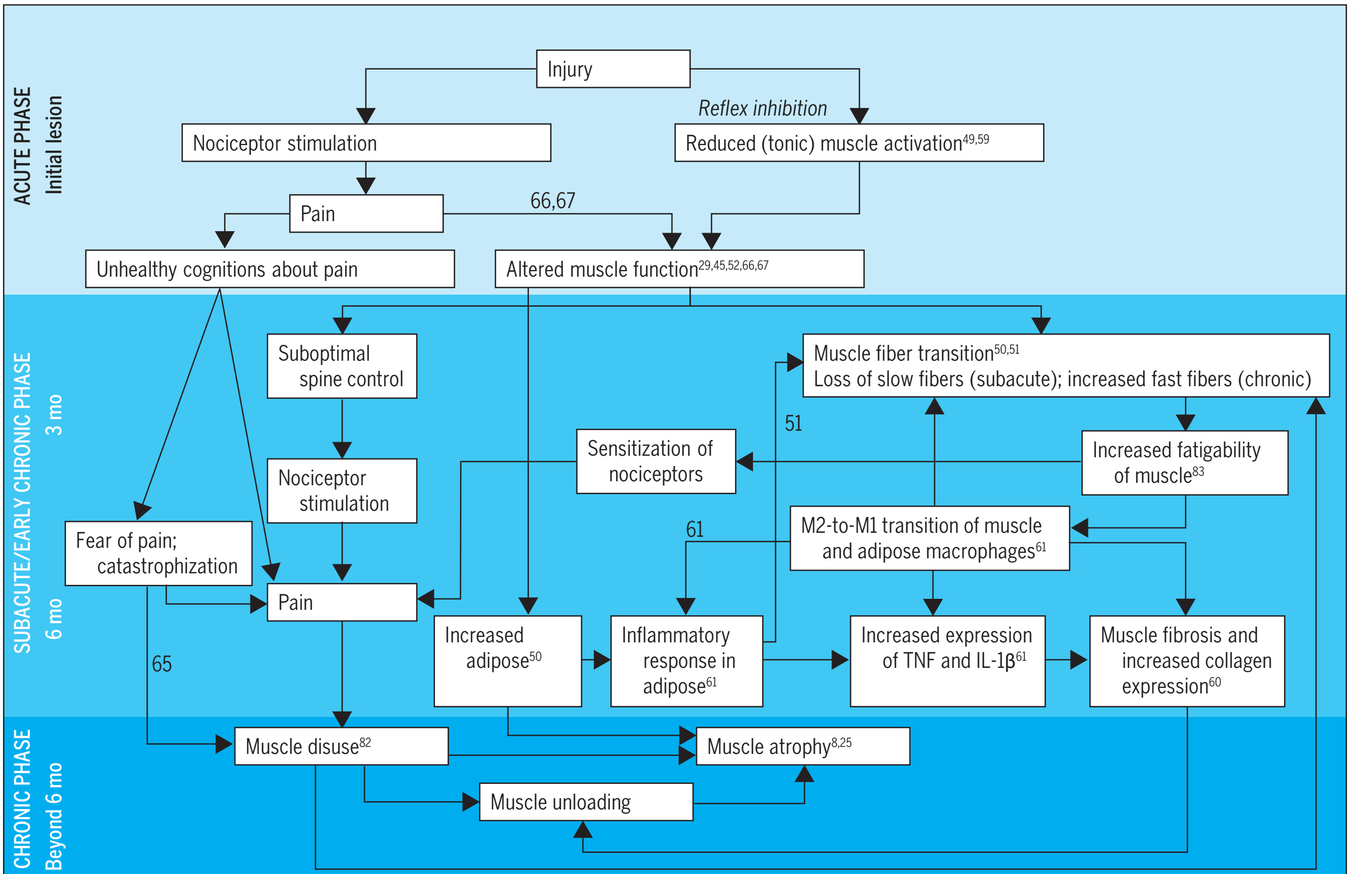


FIGURE 3. Proposed model of the timeline and mechanisms underlying the structural and inflammatory changes in the multifidus muscle after intervertebral disc lesion. Three phases are shown, with different mechanisms and different changes in structure and function: acute (top), subacute-early chronic (middle), and chronic (bottom). Citations from the text that provide evidence for the proposal's features and the causal links are provided. Abbreviations: IL, interleukin; TNF, tumor necrosis factor.

lower DM and higher SM EMG on the previously painful side, and the converse on the nonpainful side. No other muscles differed in a systematic manner. Taken together, studies of function suggest a different profile of change to that observed for an acute episode of LBP and imply a more systematic reduction of DM activation and shift in its recruitment to act as an extensor, rather than fine-tuning segmental control with non–direction-specific activation. In contrast, SM activation is commonly affected in an opposite manner with increased activation. Increased DM activation has also been observed, but that tends to occur on the side that was not the previously painful side.

Back muscle structure changes have been evaluated in cross-sectional studies using T1-weighted MRI during remission of unilateral LBP.[27] Although no differences were found in total (muscle and fat) and lean muscle and fat (macroscopically visible fat depositions) CSAs of the multifidus, ES, and psoas muscles, differences were identified in the measure of fat infiltration (relative muscle fat index) in lean muscle tissue. The muscle fat index was higher (indicating greater fatty infiltration) for those in LBP remission for all muscles on both sides and was correlated with the frequency of LBP episodes. These differences in lumbar muscle quality suggest lower contractile ability. Reduced contractile ability is also inferred from lower resting T2 MRI measures of the multifidus (but not the ES and psoas) during LBP remission on both sides at lower lumbar levels.[26] As resting T2 measures reflect the resting metabolic state of the multifidus, lower values in the LBP group would be consistent with higher proportions of type II muscle fibers (consistent with animal data[51] and cross-sectional studies of human muscle biopsies[81,82]). Although plausible, findings of changes in muscle fiber types in human studies are variable.

Lean and total CSAs were positively correlated with the time since the last episode, which suggests recovery of muscle size after resolution.[27] Further, studies of individuals with ongoing subacute LBP have observed hypertrophy of total muscle CSA because of greater fat CSA.[6] Taken together, these observations imply that multifidus muscle CSA recovers after the acute period, but that fat deposits increase in those with ongoing or recurrent symptoms.

Human Experimental Pain Studies During LBP Remission When pain is induced experimentally during LBP remission,

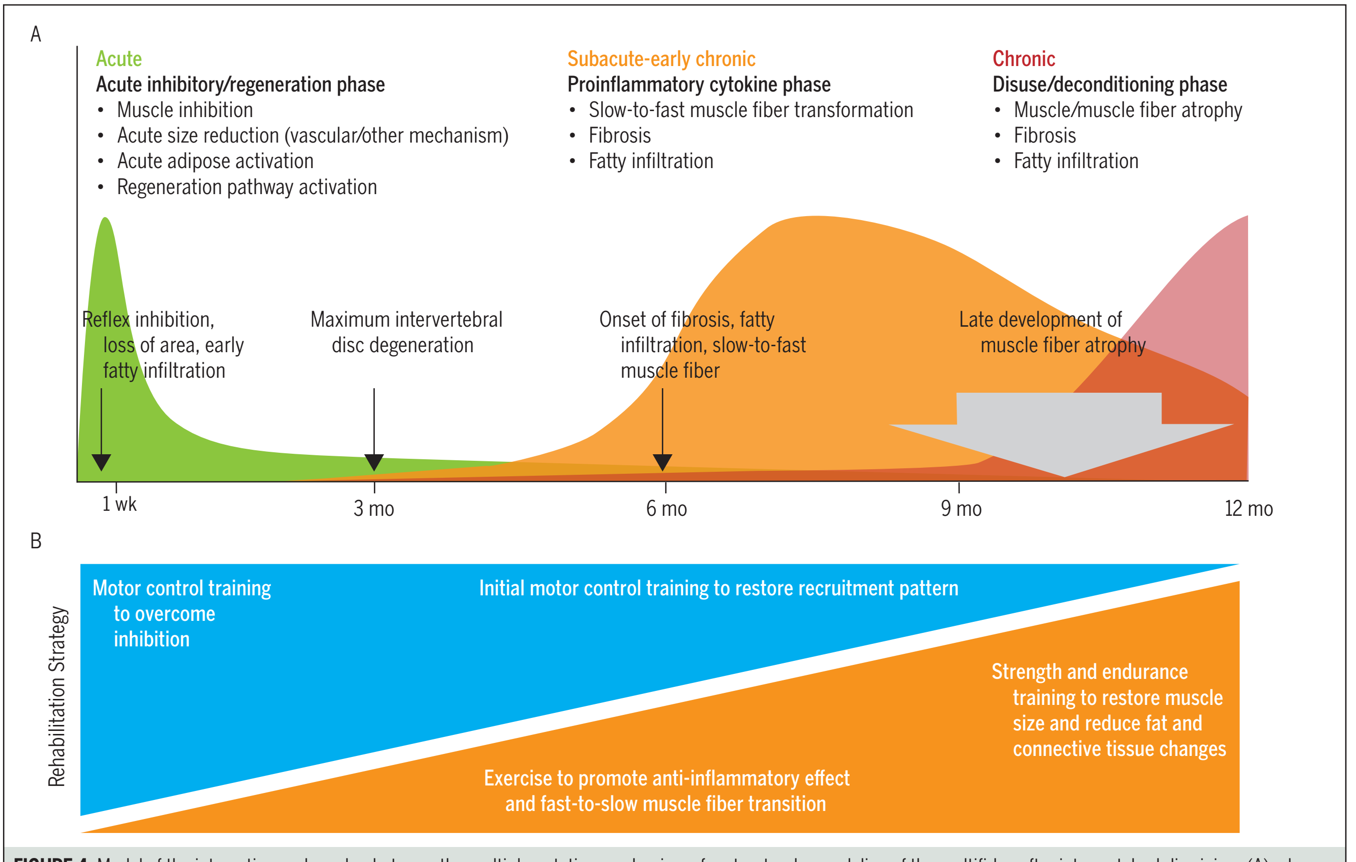


FIGURE 4. Model of the interaction and overlap between the multiple putative mechanisms for structural remodeling of the multifidus after intervertebral disc injury (A), along with (B) proposed interventions. Each mechanism has a different time course, physiological basis, and consequences for multifidus structure. Although correction of muscle recruitment patterns is likely to be relevant at each phase, the relative importance of muscle loading to train strength, endurance, and hypertrophy will increase over time. Adapted with permission from Hodges et al.[50]

changes in muscle function tend to be more generalized. In this case, hypertonic saline injection tends to induce pain intensity and location that replicate clinical LBP episodes. Multifidus, ES, and psoas activation is reduced on both painful and nonpainful sides, and at multiple segmental levels, during trunk extension.[21] This implies a more generalized response to noxious input after previous exposure to pain, consistent with animal observations of less localized adaptations over time.[51]

Subacute Animal Studies Animal studies have investigated time-dependent change in muscle structure in detail. At 3 and 6 months after IVD lesion, there is no atrophy of lean muscle or individual muscle fibers.[50,51] There is no upregulation of molecular pathways for atrophy or downregulation of hypertrophy pathways.[50] Adipose CSA increased along with connective tissue (fibrosis, which cannot be differentiated in human MRI),[50,51] and the proportion of slow type I muscle fibers reduced.[51] Although changes were localized to the injured level at 3 months, they were more generalized at 6 months.[51] In parallel studies of IVD lesion in rabbits, passive mechanical properties (stiffness) increased and multifidus fiber density decreased at 12 weeks, but not 4 weeks. Changes were not related to fiber type or protein (titin) changes, but were probably related to collagen reorganization.[10] These data imply an important role for adaptation of noncontractile tissue in the multifidus after injury.

Animal studies have enabled detailed analysis of mechanism. On the basis of evidence of involvement of proinflammatory cytokines in persistent LBP[113] and muscle remodeling,[92] involvement of inflammatory cytokines in multifidus structural changes has been examined in the subacute period.[50,51] These studies have identified elevated messenger ribonucleic acid expression of proinflammatory cytokines (tumor necrosis factor, interleukin-1β) within the multifidus by 6 months after IVD injury,[51] despite the absence of injury to the muscle. More recent work highlights that M1 (proinflammatory) macrophages are the likely source.[61] These cytokines are implicated in fibrosis,[104] adipose accumulation,[13] and muscle fiber changes,[71] providing putative mechanisms for multifidus structural remodeling[61] (**FIGURE 3**). Changes in muscle function can be both a cause (M1 macrophage polarization could follow reduced endurance from slow-to-fast muscle fiber transformation, leading to lactate accumulation) and a consequence (reduced contractile potential) of the inflammatory response[61] (**FIGURE 3**).

Bed Rest in Humans Another potential stimulus for muscle adaptation is reduced muscle loading. This was originally proposed on the basis of differential impact of muscle unloading on "slow" and "fast" muscles in rats[39]; atrophy was greater in the soleus than in the extensor digitorum. The hypothesis that reduction of muscle loading due to gravity would preferentially impact the multifidus was investigated by exposure to 3 months of bed rest. The CSA of the multifidus, but not those of the ES and quadratus lumborum, decreased,[7] and psoas CSA increased. Thus, inactivity affects the back muscles nonuniformly, with a similar distribution (biased toward the multifidus) to that found in LBP.

Summary Back muscle structure changes persist beyond acute LBP resolution. Although lean muscle atrophy tends to recover, there is evidence from carefully controlled animal studies suggesting that structural changes in the multifidus develop to include fibrosis, fat infiltration, and slow-to-fast muscle fiber transition. Thus, although neural mechanisms can explain changes in the acute context, subacute changes appear to be more likely to be explained by a muscle inflammatory response (**FIGURE 3**). In terms of function, DM activation is generally decreased, and most consistently on the painful side. Even if DM activation is increased, as is suggested in some studies, compromised muscle structure suggests that the muscle output would be less. Frequently observed augmented activation of more superficial back muscles (including the SM) implies a strategy of protection, which, when accompanied by compromised DM activation, could reduce the robustness of spine control.[47,75,108]

Compromised structure (and function) of back muscles could plausibly increase the risk for further LBP. For rehabilitation, there are numerous implications, but many questions remain regarding the potential to reverse structural changes. Although inflammatory mechanisms might be interpreted to suggest the need for pharmacological management, perhaps a safer and more direct approach is to address the problem with exercise. Exercise can promote M1-to-M2 (anti-inflammatory) macrophage polarization,[70] reduce inflammatory cytokine expression in the multifidus (in a rat model of IVD degeneration),[61] and promote transition from fast to slow muscle fibers.[93]

Which type of exercise can promote an anti-inflammatory effect and modify muscle structure? Animal studies show that regular general physical activity (mice using a running wheel) can promote polarization to the anti-inflammatory M2 macrophages and reduce connective tissue accumulation, but general exercise did not prevent changes in some components of the extracellular matrix in the multifidus.[60] These data have 2 important implications. First, the data highlight that general physical activity can prevent inflammatory and fibrotic changes in muscles, but whether the changes, once developed, can be reversed by general physical activity has not been established. This provides a basis to consider potential anti-inflammatory effects of early introduction of physical activity. Second, general activity was not sufficient to rectify all muscle changes, which implies that it may be necessary to introduce more specific exercise to address fibrotic changes in the DM muscle. There is preliminary evidence that loaded exercise reduces fat proportion.[90] In terms of muscle fiber transformation, strength training appears to be necessary to increase the proportion and size of slow-type muscle fibers.

Taken together, we propose that, in contrast to the gentle specific exercise advocated in the acute phase, subacute rehabilitation requires training with progressive resistance. However, it is important to consider that patients may preferentially use muscles other than the DM, and it is likely that initial exercise to enhance activation of the DM, and potentially reduce activation of the more superficial muscles (see Tsao et al[106]), would be required to optimize the pattern of muscle recruitment before adding load (**FIGURE 4**). Load might be added using specialized equipment[22] or training programs (eg, leg-loading tasks requiring control of the trunk).[97]

Changes in Lumbar Muscle Structure and Function in Chronic/Persistent LBP

Chronic/Persistent Clinical LBP When LBP persists, back muscle structure changes become more extensive. Most studies identify smaller multifidus CSA that is bilateral (unlike changes that are specific to a painful side in acute LBP).[8,25,37,112] Results differ between studies for other muscles. Parkkola et al[91] reported atrophy of a combined measure of the ES and multifidus, whereas several studies have reported atrophy of the multifidus but not of the ES in chronic LBP.[8,25] Kamaz et al[64] reported more generalized, smaller multifidus, psoas, and quadratus lumborum CSAs. Smaller multifidus (and psoas) CSA was observed with longer-duration LBP.[17] Comparisons between continuous versus intermittent LBP found no difference in the multifidus or ES.[40,57]

Analysis of individuals with asymmetrical pain provides some variable data. Some report smaller CSA of the multifidus,[58,62] psoas,[19] or both the multifidus and psoas[5] on the painful side, but extending over multiple segments.[5] In horses, facet degeneration of unknown duration involves localized reduction in multifidus CSA.[102]

Fat depositions that are either localized to the multifidus or distributed more generally have been reported using qualitative[62,91] and quantitative methods.[57] Goubert et al[40] showed greater fat CSA and lean muscle fat index (greater fatty infiltration) in the multifidus and ES in continuous than in noncontinuous/recurrent LBP. Although computed tomography measures have failed to find generalized fatty infiltration,[25] computed tomography muscle density measures of the multifidus and ES are lower at levels with facet joint osteoarthritis, spondylolisthesis, and IVD narrowing.[63] Animal data show a transition from localized to generalized changes over time.[50,51]

Findings for muscle fiber type proportions in chronic LBP are variable.[11,76] As an example, a matched case-control muscle biopsy study revealed lower proportions of type I fibers and higher proportions of type II and intermediate type IIc fibers (suggesting ongoing fiber transition) in LBP.[83] There was no difference in the CSA of individual fibers,[83] consistent with animal studies,[50] suggesting a smaller area of muscle occupied with type I fibers and lower fatigue resistance.[83]

Further evidence is provided by a negative correlation between proportion of type I fibers and duration of pain, but a positive correlation with type II fibers.[82] Comparison of T2 resting values shows a tendency, although nonsignificant, toward a lower mean value for the multifidus and ES in LBP, suggesting a higher type II fiber proportion.[40] In contrast, Crossman et al[18] found no differences in fiber size, type I fiber proportion, or area occupied by type I fibers in mild disabling LBP, despite earlier failure during a back extensor muscle fatigue test. Some variation may be explained by different biopsy locations, symptom severity, and control samples harvested from cadavers with unclear LBP history.[76] An important consideration is that human studies are all cross-sectional, and no longitudinal data are available. Animal studies that test separate animals at different time points but in very carefully controlled conditions provide sensitive evidence of muscle fiber differences.[50] Longitudinal human studies would help resolve this issue.

Mechanisms underlying structural muscle changes in chronic LBP have not been determined, but are plausibly explained by deconditioning. Reduced capacity as a result of earlier neural and inflammatory mechanisms may transition to reduced use in function (see below). Denervation could also explain atrophy and fat infiltration in some individuals[98,117] with conditions that compromise the intervertebral foramen (eg, spinal stenosis[42] and IVD disease[117]).

Back muscle function has been extensively studied in chronic LBP, with highly variable results (see van Dieën et al[109]). This is exemplified by studies of dynamic trunk movements that show no difference[2,68,72,89] or decreased[1,12,99,116] ES EMG during lumbar extension and no difference,[68,89] decreased,[1,2,116] or increased[99] ES EMG during lumbar flexion. When the multifidus has been studied specifically, Danneels et al[23] showed lower multifidus EMG during gentle lordosis coordination exercises, but lower activity of all back muscles during high-load exercises. This was corroborated by higher T2 shifts in the multifidus and ES after trunk extension, consistent with reduced back muscle endurance.[40]

We speculate that variation is likely to have several possible explanations. First, different adaptations have been observed in different patient groups (eg, LBP subgroups[20]). For instance, studies have shown opposite changes in multifidus activation (recorded with surface EMG electrodes) depending on whether the individual with LBP typically adopts a flexed or extended posture in sitting.[20] Second, individual differences in motor adaptation have been observed, but with a similar goal.[54] For superficial back muscles, including the ES, estimates from mathematical modeling of the net outcome of muscle adaptation (regardless of individual pattern) show increased "stability" or protection of the spine.[35,107] Although this could be reasonable to protect the spine from further pain/injury in the short term, it has the long-term costs[54] of increased load,[35] decreased movement/damping,[84] and decreased movement variation for load sharing,[54] which may increase risk for ongoing pain. Third, different studies have used different EMG recording sites, which reflect different muscles that could adapt in different ways.[75] Fourth, different EMG analysis methods, such as amplitude normalization, can lead to differences in response. Fifth, as mentioned earlier, changes in back muscles can be task specific; for instance, extension of the lumbar versus thoracolumbar regions involves different activation[15,16] and, if not controlled, can lead to variation.

Some work has considered automatic control of spinal posture in chronic LBP. Studies of trunk loading frequently reported delayed ES reaction in predictable and unpredictable perturbations,[80] or only during predictable perturbations.[69]

Back muscle function has also been investigated from the perspective of central nervous system function using transcranial magnetic stimulation. These studies show lower excitability of descending pathways to the ES[101] and modified organization of the motor cortex representation of the multifidus.[105] This latter feature was characterized by convergence of the discrete brain representations of the DM and ES. This correlates with LBP severity[96] but was specific to individuals who presented with poor ability to differentiate lumbar from thoracolumbar motion.[34] Changes in corticomotor function support the argument for compromised multifidus function in LBP; however, further work is required to understand the relationship between brain changes, motor function, and symptoms.

Consideration of changes in multifidus/ES muscle structure and muscle function together exposes an important complication for interpretation. If lean muscle is reduced in chronic LBP in association with fibrosis and fat infiltration, then force output would be lower despite identical or increased EMG. Notably, EMG may be greater relative to maximum voluntary contraction than in controls but generate less force. Similar controversy complicates interpretation of differences in quadriceps activation in knee pain.[56] This requires careful examination for the deep and superficial back muscles, which differ in their propensity for structural and functional change, to resolve the understanding of back muscle adaptation in chronic LBP.

Summary Substantial evidence in chronic LBP points to compromised muscle structure, particularly involving the multifidus, but also other muscles. Despite variable changes in function, cumulative evidence suggests enhanced activation of many muscles for spine protection, and some evidence of compromised function of the DM, with some differences explained by patient subgroups. Notably, back muscle function must be considered with respect to muscle structure. Following the subacute inflammatory-related mechanisms for muscle changes, the dramatic muscle structural changes in chronic LBP have been generally explained by disuse secondary to changes in movement patterns (shielding the DM from load[15]), pain/fear avoidance,[65] or deconditioning.[82] These possible mechanisms require further examination in longitudinal studies.

This could include excessive protection, often involving the more superficial ES, requiring strategies to reduce activation and a compromised DM and strategies to enhance function and structure. Identification of the features to address may be facilitated by assessment of movements,[33] posture,[20] and pain characteristics. Of note, it is likely that restoration of fatty and fibrotic changes in muscle structure would require resistance training once activation of the muscle is established. Danneels et al[22] showed that low-load motor control training was insufficient to restore muscle CSA in this case; controlled application of progressive overload after low-load training to improve motor patterns was required to produce hypertrophy in the multifidus and reduce pain/disability.[24] This is supported by results of a recent systematic review.[97] Such training might also reduce fat infiltration.[85] Reduced type I muscle fiber proportion implies the need for endurance training. Training in chronic LBP may require initial consideration of activation patterns, according to individual adaptation, followed by resistance training for strength and endurance.

There has been extensive debate regarding the relative efficacy of general versus specific exercise approaches for LBP. Although systematic reviews support the efficacy of motor control interventions for the management of chronic LBP, they suggest that specific motor control training is not more effective than general exercise.

There are several issues to consider. First, specific motor control training may not be more effective than general exercise, despite physiological evidence that implies that specific attention to the structure and function of muscles such as the DM may be required. Second, studies that compare motor control training to general exercise have generally applied motor control training to a group with nonspecific LBP, and in a manner that is not individualized to the patient. This contrasts with evidence summarized above that highlights individual variation in changes in structure and function of the back muscles and implies that exercise must be tailored to the individual. Further, motor control intervention is unlikely to be appropriate for all individuals. For instance, individuals with pain that is primarily maintained by central sensitization may not be appropriate for this intervention (see Hodges[46] for review). Although not a focus of this commentary, the biopsychosocial nature of LBP implies that psychosocial factors may be the priority targets for intervention for some individuals and may interact with biological features, including back muscle structure and/or function. This has been confirmed in chronic (and acute) LBP[65,87,115] and must be considered in comprehensive LBP management. The relative weighting of psychosocial and motor interventions will likely depend on the individual, which has been confirmed in several randomized controlled trials.[36,77,111]

CONCLUSION

This commentary aimed to summarize the state of knowledge with respect to changes in back muscle structure and function in LBP, from acute to chronic contexts. A major observation that explains much of the variation observed in the literature is the time-dependent nature of changes and underlying mechanisms, and the need to consider different approaches to managing LBP at different times. Successful management will depend on individual examination of adaptation of back muscle structure and function and the relative importance of psychosocial features to develop a treatment strategy with consideration of time-dependent mechanisms to tailor intervention to an individual. ◉

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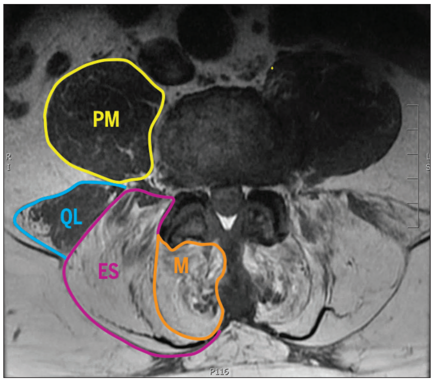

FIGURE 1. A T1-weighted, axial magnetic resonance image of the lumbar paraspinal musculature at the L4-L5 level indicating fatty replacement of the whole paraspinal muscle compartment, with preservation of the PM and some preservation of the right QL. Abbreviations: ES, erector spinae; M, multifidus; PM, psoas major; QL, quadratus lumborum.

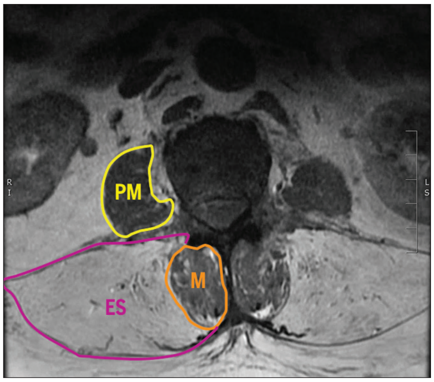

FIGURE 2. A T1-weighted, axial magnetic resonance image of the lumbar paraspinal musculature at the L2-L3 level indicating preservation of the M and PM muscles and complete fatty replacement of the ES muscles within the epimysium. Abbreviations: ES, erector spinae; M, multifidus; PM, psoas major.

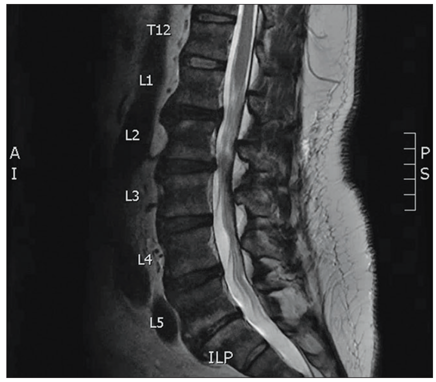

FIGURE 3. A T2-weighted, midline, sagittal magnetic resonance image of the lumbar spine demonstrating relatively normal spinal alignment and no evidence of major cord compression from soft tissue or bony structures.

Selective Fatty Replacement of Paraspinal Muscles in Facioscapulohumeral Muscular Dystrophy

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A 65-YEAR-OLD MAN PRESENTED TO his physician 3 months after a fall with a complaint of new-onset low back pain, bilateral foot numbness, and left lower extremity radicular symptoms with foot drop. The patient had a history of facioscapulohumeral muscular dystrophy. He subsequently underwent magnetic resonance imaging of the lumbar spine and was referred to physical therapy.

The patient's magnetic resonance images revealed complete fatty replacement of the erector spinae musculature throughout the lumbar spine (**FIGURE 1**). Interestingly, preservation of the lumbar multifidus muscles above the L4 level was observed, which has not previously been reported in patients with this condition (**FIGURE 2**). Psoas major and quadratus lumborum muscles were similarly preserved throughout. The patient's lower extremity symptoms were consistent with left L5-S1 radiculopathy, and the magnetic resonance images indicated mild to moderate central canal stenosis at L2-L3 (**FIGURE 3**) with severe bilateral L5-S1 foraminal narrowing.

The radiographic and clinical presentation of facioscapulohumeral muscular dystrophy typically includes upper extremity functional loss and axial muscle degeneration, which commonly manifests as bilateral and symmetrical atrophy and an increased proportion of fat within the muscle boundaries of the paraspinal musculature, ranging between 30% and 40%.[1,2] The magnitude of fatty infiltration in this patient's paraspinal muscle compartment was quantified to be over 80%, which has not been previously reported in the literature. Additionally, the selective fatty infiltration of the erector spinae muscle group with preservation of the multifidus, psoas major, and quadratus lumborum in the upper lumbar spine has not been previously described and may provide physical therapists with a viable target (the multifidus) for focused core-strengthening exercises in this patient population. ● *J Orthop Sports Phys Ther 2019;49(6):483. doi:10.2519/jospt.2019.8815*

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Déjà vu!

GUY G. SIMONEAU, *PT, PhD, ATC, FAPTA*
Interim Editor-in-Chief
J Orthop Sports Phys Ther 2019;49(6):366. doi:10.2519/jospt.2019.0103

"Research is to see what everybody else has seen, and to think what nobody else has thought."
— Albert Szent-Györgyi

It was an honor to serve as Interim Editor-in-Chief of the *Journal of Orthopaedic & Sports Physical Therapy* (*JOSPT*) for the last 18 months. Since December 2015, when my final editorial as Editor-in-Chief was published,[1] the *Journal* has rolled out several new features (Viewpoints, Perspectives for Practice, and most recently Evidence in Practice), extended its influence with now more than 155 000 monthly visits to the *JOSPT* website, and reached new heights for its impact factor (3.090).

The continued success of the *Journal* can be traced back directly to manuscript reviewers, who donate their time and expertise to provide meaningful feedback that elevates the quality of submitted articles; to editorial review board members, who provide leadership through the review process; and to the *JOSPT* production team, which ensures the high quality of the published *Journal* each month.

Most importantly, though, the *Journal* owes its success to clinicians and academics who are willing to challenge the status quo, investigate new ideas, share their expertise, and submit their work to the rest of the world for scrutiny and critique.

If you are looking for proof that physical therapy research is expanding in quality, complexity, and diversity of inquiry and methodology, you will find it in *JOSPT*.

Cheers to a bright future for the *Journal*, and for all of you!

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"Somewhere, something incredible is waiting to be known."
— Carl Sagan

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Analysis of Motor Control in Patients With Low Back Pain: A Key to Personalized Care?

In the treatment of low back pain (LBP), exercise that targets motor control is commonly used, with some success.[10,47,73] Motor control can be defined as the way in which the nervous system controls posture and movement to perform a given motor task, and includes consideration of all the associated motor, sensory, and integrative processes. Here, we use the term *motor control exercise* (MCE) to refer to exercise that aims to change the way a person controls his or her body (including posture/alignment, movement, muscle activation) to modify loading of the spine and adjacent structures.

The effectiveness of MCE has been the topic of several systematic reviews that have undertaken different comparisons.[10,47,73] A consistent outcome is that MCE is better than minimal intervention in reducing pain in the short, intermediate, and long term, and in reducing disability at long-term follow-up.[47] The pooled effect size was approximately 14% for pain and approximately 11% for disability when compared to minimal intervention.[47] Effects were better than those of many other interventions, although they were still modest and only better than other exercise interventions in the short term.[47]

SYNOPSIS: Motor control exercise has been shown to be effective in the management of low back pain (LBP). However, the effect sizes for motor control exercise are modest, possibly because studies have used a one-size-fits-all approach, while the literature suggests that patients may differ in presence or type of motor control issues. In this commentary, we address the question of whether consideration of such variation in motor control issues might contribute to more personalized motor control exercise for patients with LBP. Such an approach is plausible, because motor control changes may play a role in persistence of pain through effects on tissue loading that may cause nociceptive afference, particularly in the case of peripheral sensitization. Subgrouping systems used in clinical practice, which comprise motor control aspects, allow reliable classification that is, in part, aligned with findings in studies on motor control in patients with LBP. Motor control issues may have heuristic value for treatment allocation, as the different presentations observed suggest different targets for motor control exercise, but this remains to be proven. Finally, clinical assessment of patients with LBP should take into account more aspects than motor control alone, including pain mechanisms, musculoskeletal health, and psychosocial factors, and may need to be embedded in a stratification approach based on prognosis to avoid undue diagnostic procedures.
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KEY WORDS: *back pain, diagnostics, exercise, postural control, subgrouping*

Recent systematic reviews provide contrasting evidence for comparison of effects of MCE and general exercise on disability: one reported better outcomes for MCE,[10] and another concluded that there is low- to high-quality evidence that MCE is not clinically more effective than other exercises.[73] Of note, most large clinical trials with modest effects investigated the application of MCE in a standardized manner to a heterogeneous group of patients with nonspecific LBP. This contrasts with the prevailing clinical view that treatment effects are larger when treatments are targeted to the right patients, at the right time, and in a tailored, individualized manner. This has been a topic of considerable research and clinical attention.

It has been suggested that specific patient characteristics may predict who will

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or will not benefit from MCE,[46] or guide how exercise should be tailored to the individual patient. As reviewed by van Dieën et al,[93] laboratory studies of motor control in individuals with LBP and healthy individuals demonstrate high variability,[50,94] and also high variability between individuals with LBP within studies.[16,70] This concurs with the proposal that tailored rehabilitation programs may be required to address the specific changes in motor control that are unique to each individual.

This commentary aims to address the overall question of whether features of motor control can form an important element of a subgrouping scheme. Individualization of MCE could involve identifying subgroups of patients with similar motor control issues or a similar response to treatment, or individualizing treatment to match each patient's presenting characteristics. A further aim is to highlight the research and development that is needed to address the major issues of subgrouping, particularly related to motor control, for application in clinical practice.

Subgrouping of Patients With LBP

Based on diversity in presentation among individuals with LBP, it has been argued that no single treatment is likely to be effective for all patients, and various authors have emphasized the need to administer more personalized treatment.[6,7,27,96] Subgrouping of patients is generally considered to be a step toward personalization, and LBP is seen as a disorder for which subgrouping may be particularly useful in view of the large and heterogeneous patient population, the large variation in treatment outcomes, and the variety of available treatment options, with varying costs and risks. Clinicians generally believe that LBP includes many different conditions.[27] However, consensus on the best way to subgroup patients or to personalize treatment is lacking,[36,96] and there is no strong evidence yet for the effectiveness of subgroup-based treatment.[5,24,32,43,52]

To resolve the issues addressed above, Foster et al[26] proposed a set of requirements for subgrouping in LBP. First, the subgrouping system should be plausible; in other words, it should be compatible with current knowledge about pathology of and risk factors for LBP. Second, subgrouping should be reliable; for instance, repeated testing or testing by different clinicians should assign the same patients to the same subgroups. Third, methods need to be simple enough to allow application in clinical practice. The simplicity of a method must be balanced with acceptability to patients and clinicians as well as cost-effectiveness. Very sophisticated diagnostic instruments can be useful if the outcomes allow more effective treatment at a lower overall cost. Fourth, for clinical utility, a subgrouping system should yield mutually exclusive subgroups, meaning that all cases, at one point in time, should fit into only 1 subgroup and that this subgroup membership should guide a unique treatment choice. In the following sections, we review motor control subgrouping based on the criteria proposed by Foster et al.[26]

Is Subgrouping Based on Motor Control Plausible?

For subgrouping based on motor control to be plausible, issues with motor control would have to be relevant for the development or continuation of LBP, and relevant variation in motor control presentation would have to exist in the population of individuals with LBP.

With respect to the first question, the nature of loads on the spine and adjacent structures depends on the quality of motor control, in combination with anatomical factors (eg, muscle moment arms) and motor tasks that are performed. However, whether loading of these structures is relevant with respect to development of LBP has been heavily debated.[3,4,40,41,51,60,81,92] Recent systematic reviews and meta-analyses provide consistent evidence for a prospective association between LBP and some activities and tasks that induce high mechanical loads on the back.[11,14,30] In addition, variables that quantify (cumulative) mechanical load on lumbar tissues, such as lumbar moments and compression forces, are associated with LBP incidence or prevalence.[12,13,38,49,59]

Another line of evidence for the plausibility of a causal relation between mechanical loading and LBP stems from biomechanical studies in animal models and on human cadaveric material. Such studies indicate that loads on spinal tissues that occur in daily life can cause injury,[8,79] and, even without injury, ongoing mechanical stimulation of tissues can potentially activate nociceptors and initiate an inflammatory response.[45] Although it is difficult to confirm the presence of microtrauma, let alone noninjurious noxious stimulation of tissues in the back, in individuals with LBP, a range of literature supports the plausibility of a causal relation between mechanical loading and the development of LBP.[95]

Finally, several mechanisms can play a role in the transition to chronic LBP: nonhealing of injured tissues, ongoing nociceptive input, central sensitization, and neuropathic pain development. Mechanical loading of tissues would be relevant in relation to the first two of these mechanisms. It may both hamper and stimulate tissue healing, depending on intensity and frequency of loading and time after injury,[23,44,80] and, in the absence of frank injury, it can promote ongoing nociceptive input, especially in the presence of peripheral sensitization.[19,57,102]

With respect to the question of whether there is relevant variation in motor control presentation among individuals with LBP, a recent review of the literature concluded that the group with LBP may show overlap with or be at either extreme of the distribution in motor control found in healthy participants.[93] The groups deviating from normal motor control can be divided based on the mechanical consequences of the changes in motor control. One pattern of change involves increased activation of trunk muscles and may provide tight control over lumbar movements, but at the cost of higher loads on muscles and the spine.[90] The opposite pattern involves lower muscle activation and may avoid high muscle forces and compressive loading, but at the cost of reduced control over movement and potentially applying higher tensile strains to tissues. In the following discussion, we will refer to these 2 ends of a spectrum as "tight control" and "loose control." Clearly, tight control and loose control have different mechanical consequences that could be relevant for the development and continuation of LBP, but they also suggest different targets for MCE.

Is Subgrouping Based on Motor Control Practically Applicable and Reliable?

Studies on motor control in LBP, summarized in van Dieën et al,[93] have used a broad range of laboratory-based measurement techniques to characterize motor control. In principle, these techniques could provide a basis for the development of clinical tests to assess motor control to inform clinicians regarding subgrouping. However, application of these techniques involves substantial costs and requires specific expertise that is not readily available. Therefore, the following considers the extent to which subgrouping systems already applied in clinical practice take motor control aspects into account and the extent to which this results in reliable classification.

Several systems for subgrouping or profiling that are in common use clinically incorporate motor control aspects in the assessment of patients with LBP. Those studied most extensively are the treatment-based classification (TBC), the multidimensional clinical (MDC) framework (formerly named the O'Sullivan classification), and the movement system impairment (MSI) classification. If these assessments capture the differences in motor control that have been identified in laboratory-based motor control measures, then assessment of motor control issues based on clinically applicable tools may yield reliable outcomes.

Treatment-Based Classification The TBC system, originally proposed by Delitto et al,[18] and updated by Fritz et al[28] and Alrwaily et al,[1] proposes 4 LBP subgroups, each named for the treatment to which the patient is most likely to respond: (1) manipulation, (2) stabilization, (3) specific exercise, and (4) traction. The interrater reliability of examiners (physical therapists who are familiar with the classification system) to classify patients is clinically acceptable.[96]

With respect to the current understanding of motor control changes in LBP,[93] the criterion of hypomobility of the lumbar spine for allocation to the TBC manipulation subgroup could be considered to align with a group of patients with LBP who present with tight motor control. Importantly, other criteria for subgroup allocation (eg, time since symptom onset, pain location) cannot be considered specific to this motor control phenotype. Furthermore, it would seem plausible that the TBC stabilization subgroup could involve individuals who use loose motor control,[93] as this group is described as requiring restriction of excessive segmental motion. Consistent with this proposal, studies report that individuals classified in this subgroup more often have excessive segmental rotations or translation on flexion/extension radiography than others,[29] more aberrant segmental lumbar movement on flexion/extension radiography,[82] poorer ability to contract the transversus abdominis muscle in isolation from other abdominal muscles,[83] and lower multifidus activation,[31] which could all be considered to align with the loose motor control phenotype.

MDC Framework The MDC framework has evolved from a subgrouping approach[61] to an MDC profiling approach.[62] Within the MDC framework, motor responses are described in 3 broad contexts: adaptive/protective motor responses to an acute tissue injury and/or underlying pathological process (ie, "movement impairment"), motor responses secondary to dominant central pain mechanisms, or maladaptive/provocative motor responses that may contribute to the pain (ie, "motor control impairment"). These presentations may be associated with directional patterns of pain provocation (flexion, extension, rotation, sidebending) or multiple directions (multidirectional).[67] Reliability testing among trained physical therapists has shown good to excellent interrater reliability in the classification of patients.[17,98]

There is strong potential alignment between the MDC characterization of motor responses and the tight control and loose control phenotypes of LBP. The movement impairment presentation aligns well with motor control changes interpreted as tight motor control. The MDC movement impairment subgroup is characterized by abnormally high levels of muscle guarding and cocontraction of trunk muscles.[61] Whether the subdivision on the basis of the movement direction avoided by the individual aligns with detailed assessment of motor control has not been tested.[67] The motor control impairment presentation, which is described as demonstrating "an impairment or deficit in the control of the symptomatic spinal segment in the primary direction of pain," can be hypothesized to overlap with the loose control end of the spectrum of motor control changes. This applies in particular to patients with the flexion presentation, who tend to adopt flexed trunk postures that provoke pain. These individuals gradually increase trunk flexion over time when cycling[9] or when seated,[16,64] do not completely resume a "neutral" trunk posture (perhaps caused by proprioceptive impairment[58,63]), may have lumbar hypermobility in forward bending,[39] and demonstrate lower lumbar muscle activity in sitting.[15]

The "passive extension" subgroup of patients, who tend to hinge into extension with low trunk muscle activity,[61] may also align with a loose control group, while the "active extension" subgroup of patients, who tend to adopt extended trunk postures characterized by high muscle activity,[15,16] appear more aligned to a tight control phenotype.

MSI Classification The MSI classification system, developed and described by Sahrmann,[71] is based on the underlying assumption that people with LBP tend to move one or more lumbar joints more readily than adjacent joints/segments (eg, thoracic or hip joints). This is thought to result from habitual movement patterns during daily activity, eventually leading to excessive loading of tissues associated with the specific joint. Five LBP subgroups are proposed, named for the specific direction(s) of lumbar movement considered to contribute to the patient's symptoms: flexion, extension, rotation, rotation with flexion, and rotation with extension. Trained physical therapists can attain fair to excellent reliability in MSI classification.[96]

The MSI system describes motor impairments in LBP as a failure to constrain movement of some lumbar joints in a specific direction. This concurs with the notion of loose control, and the MSI system differentiates separate subgroups based on the movement direction in which the impairment is most apparent and linked to pain provocation. Whether the direction inferred from MSI classification parallels direction-specific differences in trunk mechanics or muscle activity requires clarification. Also, it is unclear how a tight control subgroup might relate to the MSI classification.

Do Clinical Tools Allow Reliable Classification of Motor Control? Current subgrouping methods were not specifically developed to classify patients based on motor control issues. Nevertheless, the fact that these methods reliably arrive at subgroups that likely show partial overlap with those that might be found using the laboratory-based biomechanical and electromyographic measurements used in motor control studies is promising. Objective measurement may add to the consistency, validity, and reliability of subgrouping and may, as an additional benefit, permit consideration as a measure of treatment effects, if found to be responsive. In several of the classification systems, motor control is assessed in a direction-specific manner. The relationship between directional specificity of the clinical presentation and underlying changes in motor control and their effects requires further study.

Is Subgrouping Based on Motor Control Clinically Useful?

Subgrouping based on motor control can be considered of clinical value if it has heuristic value, meaning that assignment of a patient to a specific subgroup implies a specific treatment and that such targeted care is more effective than a one-size-fits-all approach. Review of biomechanical, electromyographic, and modeling studies reveals a spectrum of changes in motor control in LBP, with extremes of tight control and loose control.[93] Motor control changes at both ends of this spectrum have the potential to lead to suboptimal mechanical loading of the spine, but in different ways. This implies that modification of motor control has potential benefit, with opposite treatment targets for the subgroups at either end. Loose control implies that enhancement of muscle activity is required, whereas tight control implies an emphasis on reduction of muscle activity.[35]

It should be kept in mind that these interpretations are based on the assumption that these motor control patterns are maladaptive, and that clinical benefit will be derived from "correction" of the strategy. For each of the motor control measures that have been used in research, there is a subgroup of individuals with LBP who show "normal" motor control,[93] which suggests that this subgroup would not benefit from MCE. There is some evidence to support this hypothesis. Two clinical trials have shown less clinical improvement for individuals without evidence of a motor control deficit (poor control of transversus abdominis) at baseline.[25,85] On the other hand, baseline findings on trunk muscle control were not correlated to clinical improvements in 2 other studies.[48,101]

The question of whether subgrouping based on motor control is useful can only be answered after appropriate clinical trials have been performed. To date, there is mixed evidence on whether interventions that target treatment based on motor control subgrouping achieve better outcomes than nontargeted treatments for LBP. Two randomized clinical trials (RCTs) with a focus on matching exercise to movement subgroups showed no benefit over general exercise in the long-term primary outcomes of pain and disability in chronic LBP.[2,72] In contrast, 2 recent RCTs demonstrated superior long-term outcomes with individualized MCE in people with chronic LBP, based on an integrated subgrouping approach: one included assistance of a wearable biofeedback device,[37] and another used an individualized approach to target relevant cognitive, motor control, and lifestyle factors.[97] A missing link is whether the clinical effects in these trials were related to a change in motor control. The possibility that other factors mediated the positive outcomes remains to be excluded. Given the preceding discussions, it can be concluded that an affirmative answer is plausible, hence subgrouping based on motor control would merit further research.

Are Subgroups Based on Motor Control Mutually Exclusive?

Mutual exclusivity of subgroups implies that an individual can only be allocated to a single subgroup and would only be expected to respond to the ascribed course of management. With the exception of the MDC framework, the existing clinical approaches described above force assessors to allocate patients to a single subgroup, making it difficult to evaluate whether subgroups are mutually exclusive. Some differences in subgroup allocation between testers (intertester variability) imply that overlap may exist.

The tight and loose control subgroups that are apparent in biomechanical and electromyographic studies would appear to be mutually exclusive, with some caveats. First, how the groups are separated is not yet clear. The literature indicates that a group with normal control sits between those with tight control and loose control. The measures that would be considered to differentiate between the groups and the cutoff scores have not been established.

Second, some patients may even present with elements of both subgroups: an overall tight presentation may be combined with elements of low stiffness in specific directions or of specific joints. For instance, increased activity of some muscles with pain, causing an overall increase in trunk stiffness, may coincide with reduced activity in other muscles.[34] While the overall change in muscle activity would allow tight control over thorax movements, it might coincide with reduced control over segmental movements in a specific direction in view of the inhibition of some muscles.

Third, motor control patterns are somewhat context dependent. An individual may show loose control in one situation and tight control in another situation. For example, a more threatening task may elicit a compensatory strategy with high levels of muscle activity, regardless of the strategy adopted in a less threatening situation.[91]

Subgrouping of patients with LBP purely based on motor control assumes that motor control and tissue loading are relevant for the underlying persistence of pain in all patients, yet not all pain is the same. As highlighted earlier, pain can be broadly considered to primarily involve nociceptive, neuropathic, or central sensitization mechanisms. In the presence of a primary nociceptive mechanism, loading of tissue is likely to be relevant. The motor control adaptation may be adaptive and potentially helpful or maladaptive and relevant for persistence. When the mechanism is neuropathic, loading may be relevant with respect to loading of neural tissue.

In the presence of primarily central sensitization pain, pain may persist despite the absence of ongoing nociceptive input from the tissue, and treatment targeted to optimization of tissue loading through motor control training is unlikely to address the underlying mechanism but could aid recovery through exposure to healthy movement. Consideration of pain mechanisms in a motor control subgrouping approach could take two main paths.

First, the approach may involve a hierarchical process, of which the first step is to identify the primary pain mechanism. If a nociceptive (and perhaps neuropathic) mechanism is identified, then the patient would be characterized according to motor control presentation. If central pain mechanisms are identified, then an alternative course of management would be planned to address the pain mechanism (pain-coping training, pain education, fear deconditioning, etc), without primary consideration of motor control.

Second, the approach could also involve a parallel process, in which all patients would be assessed based on pain mechanism and motor control, and a treatment package would be developed to include components of intervention targeted to both domains, based on the presenting features. This latter model assumes that pain mechanism and motor control phenotypes are not mutually exclusive, and that some central sensitization may be present in those with nociceptive/neuropathic pain (which is highly probable) and some nociceptive input may contribute to maintenance of the pain state. In each case, assessment of the dominant pain mechanism requires attention. Several instruments have been proposed.[65,66,74-78] These assessments require further validation and development toward a clinical tool.

To be comprehensive, in addition to pain mechanism, the diagnostic system requires evaluation of patients across multiple biological, psychological, and social dimensions. These would include features relevant to motor control, such as patterns of pain provocation and relief,[20-22,61,71] muscle atrophy and weakness,[53,54] and proprioceptive impairment,[63,84] as well as differentiation of psychological features, including pain beliefs and fear of pain or reinjury,[55,99] depression, catastrophizing, self-efficacy, and social issues.[68]

An important consideration is that domains are not independent. For instance, measures of motor control may reflect psychological factors such as fear of pain.[31,42,56,69,86,88,89] Overlap of domains, particularly some of the sensory and motor domains, may reflect redundancy and may allow simplification of diagnostic schemes. Further, in many cases, characterization of patients occurs along a continuous scale, not necessarily yielding exclusive subgroups.[65] In the parallel model, rather than fitting explicit subgroups, it may be more ideal to profile patients across these dimensions, allowing outcomes to be monitored with respect to each of the dimensions, in line with the MDC approach.[65]

Comprehensive profiling of patients or subgrouping may also benefit from being embedded in a system with stratification based on prognosis.[1] Prognostic stratification tools such as the Subgroups for Targeted Treatment Back Screening Tool (STarT Back)[33] are based on the belief that many LBP cases recover within several weeks, irrespective of treatment,[87,100] and that more comprehensive management should be reserved for those with a greater likelihood of poor outcome. These tools attempt to predict which patients belong to this group, to avoid unnecessary diagnostic procedures and overtreatment in the "low-risk" group. The STarT Back specifically identifies greater psychological prognostic barriers for recovery in the "high-risk" group and recommends psychologically informed treatment. In the "moderate-risk" group, comprehensive treatment is recommended, and our model of patient characterization across multiple domains, including motor control (with or without allocation to subgroups), is likely to be most relevant in this group.

Potential Role for Objective Tests of Motor Control in Patient Assessment

Although clinical assessments can be used to reliably allocate patients to subgroups, there may be additional benefit for interpretation of underlying mechanisms and objectively and sensitively tracking recovery by using objective measurements. Further research is needed to verify that individuals can consistently be classified into motor control–based categories via a minimal battery of objective tests.

Motor control of the trunk comprises modulation of intrinsic stiffness through tonic muscle activity, anticipatory control, and feedback control.[93] To characterize trunk control in LBP, it may be necessary to evaluate these different aspects with dedicated tests. Given the emphasis on directional preferences or directional impairments in current classification systems, objective testing should probably be multidirectional.

The potential existence of positive (adaptive) and negative (maladaptive) subcategories of both tight and loose control requires further consideration. An additional consideration is that adapted motor control may be context dependent; for example, individuals with LBP may show more pronounced changes when they perceive the assigned task as threatening in terms of pain provocation or reinjury. These considerations suggest that a comprehensive set of tests and test conditions are necessary to characterize motor control in LBP. This might cast some doubt on the practical applicability of subgrouping based on objective measures of motor control.

As an alternative approach, assessment of trunk control in daily life could be considered as an efficient way to obtain a large amount of ecologically valid information with limited effort, although substantial work would be required to develop and test such an analysis. Comprehensive testing may be shown to yield redundant information. If motor control impairments in LBP can be sufficiently characterized based on a limited number of tests, then this would greatly simplify clinical implementation.

CONCLUSION

Targeting of treatment for the management of LBP based on motor control presentation may be helpful. Although clinical trials provide evidence for some aspects of the approach and motor control literature provides support for its plausibility, there are major gaps remaining in the literature. Large RCTs are required to compare the benefit of interventions that are matched to motor control presentation against treatments that are not matched.

Further insight might be gained from the establishment of a minimal battery of objective tests that aid in the identification of the specific motor control phenotypes. Approaches to allocate patients to subgroups to guide treatment or, alternatively, to evaluate patients across a range of domains and measures should be compared for their effectiveness. Both imply personalization of care to the individual patient, and both methods have positive and negative features. ◉

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[CLINICAL COMMENTARY]

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Motor Control Changes in Low Back Pain: Divergence in Presentations and Mechanisms

There is no question that many people with low back pain (LBP) move differently than do those without pain, but the mechanism of and reason for these motor control changes are poorly understood. There are several major challenges with interpreting current literature, particularly regarding how to reconcile the enormous interindividual variation in presentation. Motor control is defined here as the way in which the nervous system controls posture and movement to perform a specific motor task, and includes consideration of all the associated motor, sensory, and integrative processes. Given the redundancy in the musculoskeletal system, the nervous system has flexibility in how different muscles and joints are recruited to achieve a motor task.

The quality of the control process is reflected in how well a posture is maintained or a movement is achieved in response to specific demands. Trunk posture and movement are continuously perturbed by neuromuscular noise (ie, the imprecision in our control system), concurrent motor tasks such as breathing,[34] and external mechanical perturbations such as the impact forces at ground contact in walking.[46] These perturbations are dealt with by modulating trunk stiffness through tonic muscle activity,[8,33,121] anticipatory/feedforward control,[39,118] and feedback based on proprioceptive, visual, tactile, and vestibular information.[1,18,62,79] Since the early observations of differences in muscle activation in individuals with LBP, it has been generally considered that many, if not all, of those with LBP present with some change in motor control.

In section 1 of this article, Is Motor Control Different Between Individuals With and Without LBP?, we consider the current state of the evidence regarding changes in motor control in individuals with LBP and conclude that findings on motor control in LBP are largely inconsistent. This illustrates the danger of basing interpretations on a limited number of studies. Published data support a specific

SYNOPSIS: Compared to healthy individuals, patients with low back pain demonstrate differences in all aspects of trunk motor control that are most often studied as differences in muscle activity and kinematics. However, differences in these aspects of motor control are largely inconsistent. We propose that this may reflect the existence of 2 phenotypes or possibly the ends of a spectrum, with "tight control" over trunk movement at one end and "loose control" at the other. Both may have beneficial effects, with tight control protecting against large tissue strains from uncontrolled movement and loose control protecting against high muscle forces and resulting spinal compression. Both may also have long-term negative consequences. For example, whereas tight control may cause high compressive loading on the spine and sustained muscle activity, loose control may cause excessive tensile strains of tissues. Moreover, both phenotypes could be the result of either an adaptation process aimed at protecting the low back or direct interference of low back pain and related changes with trunk motor control. The existence of such phenotypes would suggest different motor control exercise interventions. Although some promising data supporting these phenotypes have been reported, it remains to be shown whether these phenotypes are valid, how treatment can be targeted to these phenotypes, and whether this targeting yields superior clinical outcomes. *J Orthop Sports Phys Ther 2019;49(6):370-379. Epub 12 Jun 2018. doi:10.2519/jospt.2019.7917*

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interpretation of motor control changes in those with LBP, but a similar number of studies contradict this interpretation.

In section 2, Divergence of Motor Control Features in LBP, we propose an interpretation of the large individual variation in motor control changes in those with LBP. We suggest that it may reflect the existence of 2 different phenotypes resulting from adaptations in motor control to LBP and interference of LBP with motor control. Furthermore, we discuss the relevance that the existence of such phenotypes would have for LBP.

Finally, in section 3, Implications for Clinical Approaches to Address Motor Control Adaptation, we present clinical implications and considerations for future development in this field. The interpretation of the literature on motor control in individuals with LBP proposed here requires further validation and, hence, cannot be translated directly into guidelines for clinical practice; however, if correct, this interpretation provides a framework for further research and clinical reasoning.

Is Motor Control Different Between Individuals With and Without LBP?

In relation to LBP, motor control has been studied at the level of the neural structures and processes involved,[47,107,108,132] but more commonly at the level of patterns of trunk muscle activity and trunk movements, which represent the outcomes of these processes. Evaluation of the sensory elements of motor control has largely been limited to conscious repositioning tasks and responses to muscle vibration. The following sections present a brief overview of the evidence for motor control changes in individuals with LBP.

Is Trunk Muscle Activity Different Between Individuals With and Without LBP? In general, investigations of motor control in people with LBP have separately considered 3 main classes of motor tasks: control of the trunk in steady-state posture and movement, control of trunk posture and movement when challenged by predictable perturbations (anticipatory/feedforward control), and control of trunk posture and movement when challenged by unpredictable perturbations (reactive/feedback control).

Theoretical models and empirical observations indicate that both excitatory and inhibitory effects on muscle activity may result from injury and nociception,[43] as well as from anticipation or fear of pain.[80,116] In line with this divergence of effects, a review on differences in lumbar extensor muscle activity during steady-state tasks between individuals with nonspecific LBP and pain-free participants showed that findings are highly variable when patients are considered as a single homogeneous group. Some studies reported higher lumbar extensor muscle activity in patients, other studies reported no differences, and still other studies reported lower activity in patients.[123]

Anticipatory activation of trunk muscles has commonly been investigated in association with perturbations of trunk posture caused by rapid movements of the upper and lower limbs, which are inherently predictable with respect to direction, timing, and amplitude of related forces.[5] Some studies have reported late activation of the transversus abdominis and multifidus muscles in participants with recurrent LBP[38,40,41,63,72] and in response to an experimental noxious stimulus to the low back.[37] In contrast, another study showed no difference in onset of activation of the abdominal muscles between patients with LBP and controls,[71] and 2 other studies showed earlier activation of the oblique abdominal muscles in people with LBP.[20,78]

A systematic review on reactive trunk motor control in response to mechanical perturbations concluded that delayed onset or offset of muscle activity in patients with LBP compared to healthy participants was found in all but 1 of the included studies, while amplitudes of these responses were highly variable between patients and studies.[87] Delayed offset of activity of the abdominal muscles following release of a load into trunk extension has been associated with greater risk for a subsequent episode of LBP in varsity athletes.[9] This highlights the possible role of motor control changes in the development or recurrence of pain.

Although not directly indicative of motor control deficits, the ability of the muscle to enact the commands from the motor system will determine the ultimate efficacy of motor control, and there is evidence of structural/morphological changes in the trunk muscles with LBP. Specifically, there are substantial data from human imaging[13,26,27,131] and biopsy[49,67] studies that show changes in muscle fiber types (transition from fatigue-resistant type I to fatigable type II muscle fibers,[49,67] muscle atrophy,[26,27,131] and fatty infiltration[13]) of the multifidus muscle in acute, recurrent, and persistent LBP. Animal models, which allow a more detailed analysis of structural changes, indicate that the muscle not only shows changes in adipose tissue content, but also undergoes a process of fibrosis.[35,36]

In summary, there is considerable evidence for changes in muscle activation and muscle morphology in individuals with a history of LBP, but the observations vary. Several features may account for this variation in findings. First, the trunk system is highly redundant, with many options available to achieve a similar objective, and different individuals may adopt different solutions for the same outcome.[31] Second, changes may depend on the specific muscles investigated; deeper muscles, such as the transversus abdominis and multifidus, appear more consistently inhibited,[38,40,41,63,72] whereas changes in the larger, more superficial muscles are more variable, though activity is often increased.[123] Third, differences in motor control may depend on the tasks and contexts investigated. For instance, in anticipation of a perturbation, an individual in pain may be more likely to adopt a strategy of trunk stiffening[7,121]; consequently, studies that include threatening perturbations may yield different results from those of studies with a less threatening paradigm. Finally, differences in measurement techniques, such as the use of surface versus intramuscular electromyography, may account for some differences in results between studies.[66] How to reconcile the individual differences is a major issue, and new hypotheses are presented in section 2.

Are Trunk Alignment, Trunk Posture, and Trunk Movement Different Between Patients and Healthy Individuals? Spine and pelvis alignment have often been considered in relation to LBP. Although many studies failed to find differences between individuals with and without LBP,[53,127] differences such as greater lumbar flexion/posterior pelvic tilt, lumbar extension, or flattening of the lumbar spine have been identified when specific subgroups within the heterogeneous LBP population were studied.[10,45,81]

Low back pain is commonly expected to be associated with compromised quality of control of trunk posture and the contribution of the trunk to overall whole-body postural control. Quality of postural control has been studied in several ways, but most frequently as postural sway in standing. These studies have largely identified that individuals with LBP tend to display larger postural sway, but this finding is not universal,[73] and interpretation is complicated by the potential capacity to compensate for changes in spine function with increased reliance on postural adjustments from the lower limbs.[100] A limited number of studies focused more specifically on postural control of the trunk in tasks that reduce the contribution of the lower limbs to balance control, such as seated balancing and standing on a narrow beam. Some studies showed worse balance performance in patients with LBP,[76,89,114] but others did not find a difference between participants with and without LBP.[59,122,134]

In dynamic movement tasks, trunk movements are usually performed more slowly by participants with LBP than by those without LBP.[53] In addition, some studies reported a stronger coupling of pelvis and thorax movements and reduced variability of trunk movements in gait[56,57,115] and in repetitive trunk bending.[14] The opposite observation has also been made, with higher variability of trunk movements in individuals with LBP than in pain-free individuals during gait,[55,130] reaching movements,[97] and repetitive trunk bending.[2]

Inconsistency between studies regarding variability of trunk movement requires further reflection, and it is important to distinguish intraindividual variation (variation between repetitions) from interindividual variation (different strategies adopted by different individuals), as well as between variability that negatively affects movement outcomes and variability that does not, as its effect on movement outcomes is compensated at other degrees of freedom in the motor system.[96] High intraindividual variability may reflect poor control, but may also reflect the ability of individuals to be variable because they can adequately limit variability if needed.[126] It may also be beneficial to share load between structures[103] or to provide exposure to new options of movement to aid learning and adaptation.[102] Ambiguity can be avoided by using tasks that require participants to position or move their trunk as precisely as possible. Although only investigated in a limited number of studies, there are indications that patients with LBP are less able to precisely control trunk posture,[135] trunk movement,[12,133] and force production by trunk muscles.[3,11,19,21,86]

When using mechanical perturbations of posture and movement to probe trunk motor control, inconsistent results were found, with smaller initial displacements after perturbations in patients, no significant differences between patients and healthy participants, and even larger initial displacements in patients.[87]

Another paradigm to study movement control has focused on the interaction between adjacent body segments. This work has identified greater and earlier motion of the pelvis and spine during movement of the hip in patients with LBP,[17,95] but, again, this was specific to some individuals.

As concluded for muscle activation, discussed above, the literature on changes in trunk alignment, posture, and movement in LBP clearly indicates that differences in trunk motor control are present between participants with and without a history of LBP. However, the literature is also characterized by inconsistency in findings. Methodological differences between studies may account for some of the inconsistency, but the disparities may also be related to variance between patients with LBP, which will be discussed below.

Divergence of Motor Control Features in Individuals With LBP

Overall, the literature regarding motor control in patients with LBP shows inconsistent results. Some methodological explanations for this were addressed above. In addition, many studies included only a small number of participants. As variance in parameters used to characterize motor control has generally been large, the differences between studies may simply be due to chance, which could be addressed by larger studies. Although the literature confirms that motor control may differ between individuals with and without LBP, it also shows that motor control changes are not observed in all patients and not in the same manner. This is no surprise, as heterogeneity in the presentation of individuals with LBP, across all domains from symptoms to response to treatment, is well known. In general, where group means have indicated different control in patients, the variance within groups (between-participant variation) has been substantial, and the range of observations in patient groups has partially overlapped the range of observations in the group of healthy participants.[56,58,117,133] Furthermore, between various studies, patients have sometimes differed from healthy participants in opposite directions.

Beyond methodological differences between studies, there are possible explanations for the variation between studies and the apparent variation between participants with LBP that have clinical relevance. The clinical literature has popularized the hypothesis that variation in motor control changes is a consequence of patient subgroups.[10,94] The foundation for this argument lies in a body of work that has proposed and tested divergence in mechanisms, presentation, and outcomes in patients with LBP.[125] According to this suggestion, variation between study participants with LBP would directly reflect the presence of subgroups within the heterogeneous LBP population, who present with different characteristic muscle activation, alignment, and movement changes.

Furthermore, differences between studies might be explained by intentional or unintentional biases in patient inclusion (ie, populations may have differed with respect to severity of LBP, psychological factors, or presumed pathology). Finally, such differences may be the consequence of an interaction between the differences between patient subgroups and study context. For instance, individuals with high fear of pain are more likely to stiffen their trunk in anticipation of a perturbation.[51] Consequently, differences between patients with LBP and controls may be more pronounced in patients who are more afraid of pain, especially in somewhat threatening paradigms.

As a starting point to understanding the variation between individual patients, it is important to consider that divergence in motor control presentation may not be explained by a single factor. Differences in presentation might be explained by divergence of the underlying mechanisms for the response to injury/nociceptive input/pain; for instance, the changed motor control may represent a purposeful strategy for protection, or, alternatively, it may be a consequence of interference by pain/nociception and injury.[30] From another perspective, the divergence of changes in motor control may be considered with respect to different mechanical consequences of adaptations; for example, in some individuals/contexts, the net outcome of the adaptation may be increased stiffness of the trunk, whereas in others it may be decreased stiffness. Both proposed models of understanding the divergence in motor control changes (ie, based on underlying mechanisms versus mechanical consequences) can help reconcile some observations and are worthy of further discussion.

Divergence of Mechanisms Underlying Motor Control Changes in Individuals With LBP The literature summarized in the preceding sections is largely based on cross-sectional studies, which do not allow inferences on the direction of causality, if existent, between motor control changes and LBP. Studies that introduced experimental nociceptive input and lesions suggest that many of the differences between patients and healthy individuals can be the direct or indirect effects of pain and/or injury. On the other hand, while, for example, delayed trunk muscle responses after mechanical perturbations can be elicited by experimentally induced pain,[4] similar changes have been observed to precede LBP and increase LBP risk.[9] Thus, motor control changes can likely be both a cause and an effect of pain and injury, but we will consider them as effects here.

Injury/nociceptive input and pain are potent stimuli to change motor control, and several mechanism-based theories have been developed to reconcile the diversity of observed changes. These can be distilled into 2 main categories: those that consider the change as a consequence of motivation of the system to adapt as a purposeful strategy to protect the body region from further pain/nociception and injury, and those that consider changes to result from interference by pain/nociception and injury with motor control.

In theories considering motor control changes as purposeful adaptations to avoid pain, it was initially assumed that reflex-like changes induced by nociception cause higher activation of antagonistic muscles and lower activation of agonistic muscles, leading to higher stiffness and slower movement.[61] This view has been criticized based on the variability of empirical findings[123] that we have also highlighted. More contemporary views imply that learning processes play a role in adaptation of motor control to LBP.[119] Such learning processes could result in different responses to a seemingly identical stimulus and in association with anticipation or fear of pain and/or (re)injury in the absence of injury or nociceptive input, or in response to pain-related distress.[25,54,78,80,90,113,116]

Injury or nociception can directly interfere with motor control, as it can change excitability of motor pathways at different levels of the nervous system. Importantly, it can cause either an increase or decrease of excitability,[32] which may account for some of the changes and variability in changes in muscle activation observed in patients with LBP. In addition, nociception may affect proprioceptive afference[105] and, consequently, interfere with motor control. This would be in line with findings of impaired proprioception in patients with LBP,[106] which appears to cause reduced precision in the control of trunk movement.[133] Changes observed in LBP in the sensory cortex[16] and in the motor cortex[108] and reduced corticomotor excitability[104] may also interfere with motor control. Finally, structural changes, such as loss of segmental stiffness,[74,88,137] muscle atrophy,[26-28] and connective tissue changes,[35] will change the relation between motor commands and motor output and may interfere with motor control as a result.

Divergence of Mechanical Consequences of Motor Control Changes in Individuals With LBP The literature on motor control changes with LBP suggests that patterns of change observed can be divided based on their mechanical consequences. One pattern of change, which involves increased excitability of trunk muscles, may provide tight control over lumbar movements at the cost of higher tissue loading.[117] This could be the result of increased cocontraction, reflex gains, and/or attention to movement control. The opposite pattern, which involves reduced muscle excitability, might avoid high tissue loading, at the cost of a loose control over movement. These 2 patterns, which are referred to as "tight control" and "loose control" in the following discussion, may be adaptations to LBP, as suggested by their positive consequences (enhanced control, reduced tissue loading), but may also be caused by interference.

Although plausible, the existence of different phenotypes of patients based on these mechanical consequences of divergent presentations has largely been inferred by data from separate studies. A single study by Reeves et al[91] provides evidence for 2 identifiable subgroups in line with this distinction. In this study, participants with LBP fell into 1 of 2 groups: those who showed preferential activation of lumbar extensors over thoracic extensors, and those who showed the opposite activation pattern. Biomechanical modeling predicts that preferential activation of the lumbar extensor muscles enhances control over lumbar movement, while causing higher tissue loads, and vice versa for preferential recruitment of thoracic extensors.[117] This study[117] thus provides an indication of the existence of tight control and loose control subgroups with high and low tissue loading, respectively. These subgroups are likely part of a continuum, as a middle group with normal trunk extensor activation was also present. The long-term consequences of, and clinical strategies to address, these responses are likely to be different for such subgroups.

In summary, individuals with LBP may show a spectrum of deviations in motor control, and this will affect mechanical loading on lumbar tissues. In some cases, these changes may be beneficial to the health of the tissue (at least in the short term); in others, the resultant loading may be or become the source of nociceptive input. Tissue loading may not be relevant in all individuals with LBP and is likely to be most important for those who continue to have a contribution of nociceptive input to their ongoing pain. Tissue loading may have enhanced relevance in the presence of peripheral and central sensitization, where lower load magnitudes may be sufficient to excite sensitized afferents. The potential consequences of tissue loading resulting from motor control changes at the divergent ends of the spectrum require more detailed consideration.

Consequences of Tight Control Tight control implies augmented constraint of movement, presumably with the objective to avoid nociceptive excitation, pain, or injury, or in anticipation of such threats. In the short term, tight control would tend to increase the "safety margin" for control of movement and resulting tissue strains. For example, increased cocontraction and reflex gains would increase trunk stiffness such that greater force would be required to perturb the spine from its position or trajectory. An advantage would be a reduction in the need to intricately control the sequences of muscle activation matched to the task demands, thus reducing the potential for error that may arise when sensory feedback is inaccurate or the force-generating capacity of the muscle has been modified. This strategy would also be expected to reduce variation in movement and the need for finely controlled anticipatory actions and feedback responses to counteract perturbations.

Tight control could be subtle, with slight modifications of activation within a region of a muscle,[109] or more extreme, such as bracing of the body region.[29,117] Complete avoidance of a task/function that is characteristic of some people with LBP might also be considered as an extreme example of a tightly controlled protection solution.[42] Although tight control appears logical and beneficial, at least in the short term, it could also have negative consequences. Data showing an association between pain relief after spinal manipulation and a reduction in lumbar stiffness suggest that stiffening of the trunk may even be directly linked to pain.[136]

Increased trunk muscle activation to tighten control comes at the cost of increased spinal loading. Patients with LBP have been shown to expose their spine to higher forces than healthy participants after perturbations[65] and during lifting.[68-70] Because the most pronounced differences in loading were found during the least heavy tasks,[69] the risk of acute overloading of the spine is probably limited, but increased cumulative loading may elevate the risk. Further, low-level cocontraction of trunk muscles has been found in patients with LBP even at rest,[123] implying that compression of the spine is sustained during recovery periods. Animal models implicate sustained low-level compression as a cause of intervertebral disc degeneration, allegedly due to disrupted fluid flow into and out of the disc.[60,85] Recovery of body height during rest after exercise, an indication of reuptake of water in intervertebral discs, was reduced in patients with LBP, and the lack of recovery was correlated to trunk muscle activity during rest.[23,24] This suggests that fluid inflow into the disc may be impaired by sustained muscle contractions in patients with LBP, with possible adverse effects on disc health.

Sustained low-level muscle activity, as was found in some patients,[123] may also have noxious effects in the muscles.[129] Trunk extensor contractions at intensities as low as 2% of maximum activation do cause fatigue manifestations within half an hour.[124] Patients who show sustained trunk muscle activity may thus incur muscle fatigue and related discomfort,[50,98,99] or even LBP of muscular origin,[129] especially if peripheral sensitization is present.

There may also be consequences of the decreased motor variability that is associated with tight control.[115] It is increasingly recognized that some degree of variation is essential for tissue health.[103] Although too much variation may reflect uncontrolled motion, some variation is beneficial, as it allows sharing the load between different structures across repetitions.[22,103] In addition, motor variability appears essential to provide an opportunity to learn through exposure to alternative ways of performing the same movement task.[6,48,102,111] Participants who showed a change in trunk muscle recruitment in fast arm movements during which pain was experimentally induced also showed a strong decrease in variability of muscle recruitment, and maintained these changes over the course of 70 arm movements when pain stimuli were no longer presented.[78] This clearly suggests that decreased variability hampers relearning of "normal" motor behavior, even after pain has subsided.

Finally, high trunk stiffness in patients with LBP appears to be related to a reduced use of trunk movement to counteract anticipated perturbations, which coincides with larger involuntary trunk displacement due to the perturbation.[75] Further, although enhanced trunk stiffness may be an effective strategy to counteract small disturbances, it may compromise an individual's capacity to maintain balance on unstable or restricted surfaces,[76,92] or when encountering larger disturbances.[77]

Consequences of Loose Control At the loose end of the spectrum, patients have less control over trunk posture and movement. This might be the result of a protective adaptation to prevent pain provocation and reduce tissue loading related to large muscle forces or resulting compressive spine loading.

It is well accepted that the lumbar spine is an unstable structure whose configuration requires control by the surrounding musculature. Given the large number of degrees of freedom in the spine and given the fact that loads imposed on this system can be high and unpredictable, this poses a substantial control problem.[83,120] Muscular control over spine movement would be reduced by inhibition of muscle activity and associated increases in delays in response to perturbations. This would be associated with faster and larger amplitude movements, with more variability between repeated performances of the same task. If muscular control over the spine fails, midrange alignment of the lumbar vertebral segments may be compromised, resulting in large tissue strains.[83,84,120] Also, sustained end-range alignment may, through creep loading of spinal tissues, cause tissue responses and, potentially, pain.[82,101] Whether modified or uncontrolled motion constitutes instability or is simply less robust control of motion with greater potential for abnormal tissue loading has been debated.[93] Cholewicki et al[9] showed that large displacements after trunk perturbations were predictively associated with LBP, providing support for the notion that loose control can cause LBP.

Implications for Clinical Approaches to Address Motor Control Adaptation

Given the mechanical consequences and loading outcomes of the divergent presentations of motor control changes in people with LBP, it follows that different interventions are likely to be required to address different patient phenotypes. From the perspective of tight control linked to protection, in the early acute phase, the response may seem reasonable; however, if persistent, the negative consequences (increased loading, reduced movement) would likely become problematic for spine health. Thus, clinical strategies in later stages could be reasonably targeted to reduce excitability and cocontraction and to increase movement and potentially movement variation.[44]

For loose control, strategies to augment control may be required.[44] The notion that loose control has a negative impact on clinical outcome in LBP forms the foundation for many exercise approaches. This has been targeted in some trials of interventions tailored to specific phenotypes of patients with LBP.[15,52,64] In support of this approach, 2 clinical trials have shown greater clinical efficacy in patients identified to have deficient control of deep trunk muscles at baseline, and better clinical outcomes in those with improved function of these muscles after motor control intervention.[15,112] Complicating treatment choices, there is a potential for overlap between effects of adaptation to and interference by LBP (eg, lower activation of the multifidus muscle might occur due to reflex inhibition with a concurrent protective strategy of increased activation of the erector spinae muscle).

Despite promising data, there are significant challenges before validity of the existence of the proposed subgroups or phenotypes can be supported. It is critical to have valid assessments that can identify the pattern to change, therapeutic methods (eg, exercise approaches) to enact the change, and evidence that treatment targeted to the individual presentation leads to better outcomes than treatment that is not targeted. Some data are available,[128] but are far from complete.

CONCLUSION

Although motor control adaptations to pain present across a spectrum, 2 broad phenotypes of patients with LBP have been tentatively defined at the extremes of a spectrum, based on changes in trunk motor control observed from many studies. One phenotype shows tight control over trunk posture and movement due to increased excitability, at the cost of increased tissue loading secondary to increased muscle contraction. The other group shows loose control due to reduced excitability, with the potential cost of increased tissue loading from excessive spinal movements. Both groups involve abnormal loading of tissues in the low back, but with different mechanisms.

For both groups, there may also be an adaptive value of changes in motor control, at least in the short term: the first group may avoid excessive movement, and the second group may avoid high muscle forces. For both, it remains unclear whether the adaptive value outweighs the negative consequences, and this may differ between individuals, depending on the motor tasks to be performed and the integrity of the tissues in the low back. It is, in this context, important to note that nonspecific triggers, such as fear, can cause changes in motor control similar to those identified with pain.[110] In case of unwarranted fear, there would be no benefit of the adaptation, as no additional protection is required. Differences between these different phenotypes of motor control changes in individuals with LBP and the different consequences for mechanical loading support the notion that targets in motor control intervention should be different, and possibly even opposite, for these groups. This supports the plausibility of phenotyping and treatment targeting based on motor control presentation for the management of LBP. ◉

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