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Can Biomechanics Research Lead to More Effective Treatment of Low Back Pain? A Point-Counterpoint Debate

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There is little doubt that biomechanics, considered here as the mechanics of body movement, including neuromuscular control, plays a role in the development of low back pain (LBP) and perhaps in the persistent and/or recurrent nature of this condition.[66,100] There is consensus that LBP is a multifactorial problem, and many biopsychosocial factors affect the clinical presentation of LBP and treatment outcomes.[19] Although biomechanics is acknowledged to be one aspect of the “bio” component, much of the biomechanics research in LBP has not considered other biopsychosocial factors (that have been discussed since the 1980s[121]) and their interactions. Notwithstanding all past accomplishments of biomechanics,

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it is timely to evaluate the role of biomechanics as a stand-alone discipline to address the LBP problem.

This commentary focuses on 2 questions. First, does biomechanics in isolation have the potential to advance treatment of LBP? Second, how likely is it that such an approach would improve treatment strategies for LBP? This commentary is presented as a point-counterpoint discussion. First, we present the view that biomechanics alone cannot lead to improved outcomes for LBP. Second, from among the various models that consider biomechanics, we select 3 that offer very different perspectives on how biomechanics may improve outcomes for LBP by guiding selection of clinical interventions. Third, we present the counterpoint reaction of the authors of the biomechanical models to the presented debate. The overall objective is to foster discussion to encourage fruitful development in this field.

POINT

Consideration of biomechanics alone is unlikely to lead to more effective treatment strategies for LBP. Many elegant biomechanical models of LBP have been developed based on clinical observation, basic research, and discovery of common biomechanical features in samples of individuals with LBP. Various treatment strategies were then designed to address these proposed mechanisms, many of them evaluated in clinical trials. For example, a deficit in the recruitment of the transversus abdominis was proposed in 1996 as an important mechanism associated with LBP.[44,123] Additional observations of delayed trunk muscle reflex responses and mathematical formulation of static and dynamic models assessing spinal stability[15,18,59] informed the development of various forms of motor control rehabilitation and trunk stabilization exercises.[92,97] Other examples of models involving biomechanical factors in LBP include the movement system impairment[107,113] and directional preference (eg, McKenzie method for treating LBP) approaches.[70] These models are based on sound anatomical, biological, and mechanical principles, which provide a foundation for internal validity. Furthermore, the relationship between pain and the proposed biomechanical measures is supported by a body of research demonstrating, for example, differences between patients with LBP and healthy controls. The development and refinement of models of LBP such as these have increased our knowledge regarding biomechanics and LBP. The critical question is whether these biomechanical representations of LBP can lead to intervention strategies that are superior to other nonsurgical therapies.

Recent systematic reviews of randomized clinical trials (RCTs) have revealed that consideration of biomechanics alone does not produce a uniquely distinct and effective treatment[20] (with the exception of exercise therapy, which can affect multiple systems beyond biomechanics[114]). Although treatment strategies based on these models are generally better than no treatment, clinical trials have generally not shown them to be superior to other forms of exercise or other types of treatment when applied to patients with chronic nonspecific LBP. On this basis, it has been broadly argued that the efficacy of interventions (including those based on biomechanics) would be greater if applied to specific patient subgroups that would be expected to respond best to specific interventions.[32,57,91,104] Although identification of subgroups has been a goal of research for some time,[31] a

successful subgrouping method that guides treatment based on biomechanical factors remains elusive.[26,32,57,91]

There are several possible reasons why consideration of biomechanics has not led to improved outcomes. First, it seems likely that consideration of factors other than biomechanics will be required for effective patient selection and treatment allocation.[42,104] Second, biomechanics-based interventions may not have reached adequate refinement to achieve their highest possible impact. Third, they may only be effective in a very narrow subset of patient presentations, and methods to select those patients may not be realized. Fourth, the identified biomechanical factors may not be the cause of nociceptive input contributing to the pain response, or pain may be continuing for reasons other than ongoing nociceptive input. For example, even when a "provocation" test reproduces a patient's pain, or local injection of an anesthetic agent reduces pain, it cannot be concluded that the identified motions or structures are responsible for the maintenance of pain[34] or, more uncertainly, whether targeting them with some intervention will lead to clinical improvement.[13,43]

Whether any of these alternatives explains the lack of strong evidence for efficacy of an approach based on biomechanics is not yet clear, but we suggest that the most likely explanation is that LBP is a multifactorial problem, in which any individual factor or mechanism plays a small role in the overall condition, and the outcomes of interventions based on any such mechanisms might easily be obscured. If we consider LBP a truly multifactorial problem[17] (FIGURE 1), what potential is there for identification of individual biomechanical factors to play a role in the treatment of LBP? Numerical and analytical simulations of multifactorial presentation of LBP demonstrated that if a large number of factors contribute to an individual's LBP, then any treatment strategy that seeks and treats the most dominant factor is less effective than treating any 2 or more factors chosen arbitrarily.[16] Furthermore, the probability of identifying subgroups that might respond favorably to a specific intervention in such a population approaches zero as the number of factors contributing to the LBP presentation increases (FIGURE 2). If biomechanical factors are intertwined with many other factors across the biopsychosocial domains in individuals with LBP, then the most likely results from RCTs will be the inability to subgroup and effectively treat LBP based purely on biomechanical factors.

An additional concern about the evaluation of any intervention, including one based on biomechanics, is that the interpretation and synthesis of clinical trials designed to isolate an individual factor or a mechanism of LBP are complicated by nonspecific effects associated with various therapeutic modalities (eg, clinician-patient alliance,[30] placebo effects,[6] etc). Unfortunately, nonspecific effects of treatment present some unique challenges, because RCTs are ill equipped to estimate or control them.[6] For most nonpharmacological interventions, it is not possible to design a double-blinded sham treatment where both the therapist and the patient are blinded as to the treatment administered and received. This problem makes it difficult to estimate the specific effect size attributable to any intervention, including one based on biomechanics.

For all the reasons described above, we argue that it is unlikely that biomechanical factors alone will be able to guide treatment with an effect size greater than that achieved by other therapies. Nevertheless, we believe that biomechanics has its place among the constellation of factors contributing to LBP. The challenge is to identify how best to systematically integrate knowledge from various fields of science and stakeholders, which appears necessary to achieve the goal of more effective management of LBP treatment and reduction in disability.

COUNTERPOINT

Biomechanics research can lead to more effective treatment strategies for LBP.

The following 3 biomechanical models present examples of how biomechanics has been used as a primary feature to guide management of LBP. These examples have been studied extensively and were selected to show the diversity of biomechanical concepts that have been considered.

Biomechanical Model 1: Intervertebral Mechanical Dysfunction in Nonspecific LBP

There is consensus that chronic nonspecific LBP is a biopsychosocial problem. Consideration of psychosocial factors alone explains little of the variance in outcome, and effects are small when they are used to guide treatment.[41,86] In the biological domain, there is diversity across chemical, mechanical, and neuroplastic mechanisms, and it is questionable whether a mechanical phenomenon, such as lumbar movements, could alone provide a marker of sufficient influence to identify phenotypes and pain generators in LBP populations.[33]

As most LBP presentations are affected by movement or position, it is likely that mechanical factors play a role. Attempts have been made to measure mechanical factors to assess whether they are more prevalent in people with LBP than in those without LBP, and whether they cause pain episodes and/or moderate or mediate outcome. This counterpoint presents a view of LBP that includes lumbar segmental mechanics and how mechanical assessments may be used to identify factors important in back pain generation and perpetuation—and therefore in physical treatment.

Measurement of Intervertebral Motion—Lumbar segmental motion can be measured by digital registration of the positions of individual vertebral segments on fluoroscopic sequences, and the movement between them. This technique has been validated.[7,10–12,28,78–80,102,125] Kinematic variables include intervertebral range of motion, anterior/posterior translation, disc height, finite center of rotation, intervertebral laxity (as initial rotational attainment rate), phase shift, and proportional motion pattern variability. There is little evidence that intervertebral range of motion, translation, disc height change, and laxity are different between people with and without LBP, but there is some evidence that translation and laxity are relevant for those with LBP related to injury.[54,55,58,85]

Proportional motion, representing the sharing of bending across segments, is more variable in people with LBP. Using flexion/extension radiographs, Abbott et al[1] demonstrated that

patients with LBP had proportional intervertebral range of motion and translation ranges outside of reference intervals of pain-free controls. Continuous fluoroscopic sequences that combine left and right and flexion and extension motion into a single summary measure of the variability of motion sharing show differences between patients with LBP and matched controls.[80] Results have been replicated and expanded in recent studies (FIGURE 3).[8,9]

Implications—The observation that motion sharing variability is significantly greater in people with LBP than in controls suggests that it is worthy of exploration as a biomechanical marker, and perhaps a moderator, of LBP outcomes. Good intrasubject reliability over 6 weeks makes it a suitable measure to identify subgroups. Poor agreement parameters suggest that it may be unsuitable as a mediator or outcome measure.[24] Because data can be recorded during passive recumbent lumbar motion and during standardized weight-bearing active motion, results can be extrapolated to make inferences about both intervertebral restraint (passive mode) and performance (upright mode).

Uneven motion sharing at segmental levels is a consequence of heterogeneity in passive system restraint. This may be the result of structural factors, such as disc degeneration or ligament tighten ing.[63] If these are related to abnormal motion patterns, then treatment options that alter the patterns could be tested as interventions. Likewise, relationships between aberrant motion in the upright position and the presence of LBP could be interpreted to suggest that pain is generated by the effects of untoward muscular effort and/or inconsistency of loading, where metabolic deficit in the former, or rapid displacements in the latter, could be responsible. Either way, they relate to the extent of control that is achieved at segmental levels.

The case for consideration of the importance of this biomechanical factor in guiding LBP management is supported by evidence of differences between patients and controls without any stratification based on factors in other biopsychosocial domains. The biomechanical model may benefit from expansion to include more diverse variables such as muscle metabolic factors,[27] inflammatory markers,[65] mathematical modeling to infer loads with input from combined quantitative fluoroscopy and 3-D magnetic resonance imaging,[126] muscle activation patterns, lumbar configuration (eg, lordotic curve), and response to perturbation and changes in balance. Consideration of relationships between segmental motion patterns and patient-reported data (directional preference, pain impact, kinesiophobia, distress, and somatization) may also be important to outcomes.

Summary—Mechanical dysfunction has been rightly called into doubt as a single explanatory variable for LBP, and the view that a biological approach alone is inadequate is indisputable.[124] However, through the mist of complexity, biomechanical features cannot be discounted, especially if they are measurable, frequently present, and able to discriminate individuals with LBP from pain-free individuals.

Biomechanical Model 2: The Kinesiopathologic Model

The kinesiopathologic model was designed specifically to describe the mechanically related processes proposed to contribute to the development and course of LBP (FIGURE 4). The basic premise is that LBP results from the repeated use of direction-specific (flexion,

extension, rotation, lateral bending, or a combination of these) stereotypic movement and alignment patterns in the lumbar spine. The model proposes that the patterns begin as the result of adaptations of the musculoskeletal and neural systems due to repeated use of specific movements and alignments during daily activities. The nature and rate of the adaptations can be modified by intrinsic and extrinsic characteristics of the individual, for example, sex, anthropometrics, or typical activities of the person. The typical pattern is one in which, during performance of a movement (eg, forward bending) or assumption of a posture (eg, sitting), the lumbar spine moves into its available range in a specific direction more readily than other joints, such as the knees, hips, or thoracic spine, do.

In this context, movement occurs sooner or proceeds farther than is ideal. With the repetitive use of the same pattern across multiple activities, some/all of the lumbar joints become relatively more flexible than other joints. Repetitive use of the same relative flexibility pattern(s) decreases variability in the types of lumbar movements and alignments used throughout the day, contributing to repeated subfailure magnitude loading and accumulation of localized areas of lumbar spinal tissue stress. Over time, the rate of accumulation of tissue stress is proposed to be greater than the adaptive tissue remodeling needed to prevent tissue failure. The result is tissue irritation, LBP symptoms, microinjury, and potentially macroinjury. Finally, the model maintains that until the relative flexibility patterns are modified, LBP may persist or recur. The following sections present evidence that supports the model.

Existence of Direction-Specific Relative Flexibility Patterns—A major assumption of the kinesiopathologic model is that people with LBP display direction-specific relative flexibility patterns across clinical tests (eg, forward bend test) and daily activities, and that there are subgroups of people with LBP who differ based on their specific relative flexibility pattern.[90]

In a study of people with LBP, findings from standardized clinical tests designed to identify relative flexibility patterns[111] revealed 3 intercorrelated groupings of tests related to 3 LBP subgroups: lumbar (1) extension, (2) rotation, and (3) rotation with extension.[113] Subsequent evidence of examiner reliability to identify relative flexibility patterns and LBP subgroups with clinical tests supports the existence of the patterns and proposed subgroups.[39,40,61,69,83,88,103] Additional studies using clinical tests reported that people with LBP display a greater number of relative flexibility findings than people without LBP,[68,122] and in people with LBP the prevalence of the patterns differs between sexes.[95] Laboratory-based studies quantifying patterns during individual clinical tests have documented more relative flexibility patterns in people with LBP than in people without LBP,[29,60,61,76,83,93] subgroup-specific differences in patterns,[37,46,60,75,83,105] and a different prevalence of patterns between sexes in those with LBP.[36,47,48,83]

Laboratory-based studies of patterns during daily activity tests have documented that people with LBP, compared to those without LBP, display a relative flexibility pattern during the activities of picking up an object and stand-to-sit.[37,72,96] Further, the relative flexibility pattern with the picking-up-an-object test is used across other daily activity tests[71] and is related to the pattern used during the clinical test of forward bending.[72] Finally, subgroup-

specific differences in relative flexibility patterns have been identified among LBP subgroups with the picking-up-an-object test, aspects of gait, and lumbar alignment in sitting.[35,37,46,49]

Relevance of Relative Flexibility Patterns to the LBP Condition—A second kinesiopathologic model assumption is that relative flexibility patterns are relevant to the person's LBP. Two studies reported that systematically modifying relative flexibility patterns displayed during symptom-provoking clinical tests improved reported LBP during the tests.[106,112] Within-session treatment of the relative flexibility pattern during the picking-up-an-object test resulted in both an improvement in the pattern and an improvement in reported LBP during the test.[73] Moderate to large[21] relationships also have been documented between the severity of the relative flexibility pattern across daily activity tests and LBP-related functional limitations.[71,72] Finally, relative flexibility patterns during clinical tests are related to the LBP condition severity,[25,68,110] a person's ability to correct the pattern,[94] and the risk for LBP development.[88,98,99]

Repetitive Activities and Relative Flexibility Patterns—A third assumption is that relative flexibility patterns are, in part, a consequence of repetition of movements and sustained alignments in the same direction with activities people perform regularly. Two studies of people with LBP showed that the type[89,109] and number[109] of relative flexibility patterns were related to an individual's regular leisure activity. Another study compared findings during clinical tests among people without LBP who did not participate in a rotational sport and 2 groups who participated in a rotational sport—people with and people without LBP. People who participated in the sport (LBP and no LBP) displayed a greater number and asymmetry of relative flexibility patterns associated with lumbar rotation than people without LBP who did not participate in the sport.[122] Of those who played the sport, those with LBP displayed more relative flexibility patterns with tests of limb movements[122] and spent a greater proportion of their leisure time playing the sport than those without LBP.[14]

Summary—The kinesiopathologic model describes the mechanically related processes proposed to contribute to the development and course of LBP. A primary process proposed to contribute to LBP is the tendency for 1 or more of the lumbar joints to move more readily than other joints in a specific direction (ie, a relative flexibility pattern) when performing daily activities. Numerous studies support that relative flexibility patterns are prevalent in LBP, relevant to the person's LBP condition, associated with increased risk of LBP, and associated with the activities people participate in regularly. Collectively, these findings suggest that targeting symptom-provoking, relative flexibility patterns used during repetitive daily activities may be an effective, efficient, and feasible method to improve outcomes and, potentially, maintain the improvement over time.

Biomechanical Model 3: Anatomy, Biomechanics, and Pathology of the Sacroiliac Joints

One specific subgroup of patients with lumbopelvic pain with a clearly defined anatomical/biomechanical model involves those with sacroiliac joint (SIJ) involvement. The SIJs are highly specialized joints that permit stable (yet flexible) upper-body support. In bipeds, the

pelvis serves as a platform with 3 large levers acting on it (spine and legs). The tightness of the well-developed fibrous apparatus and the specific SIJ architecture limit mobility. Sacral movement involves the SIJ and directly influences the discs and higher lumbar joints; for example, forward and backward tilting of the sacrum between the iliac bones affects the joints between L5 and S1, as well as higher spinal levels.[119,120]

The SIJ is unique, with elements of a combined synarthrosis and diarthrosis, that is, amphiarthrosis (FIGURE 5). The joint's main portion is surrounded by a complex capsule and lined with cartilage (diarthrosis). Its shape is auricular, and "opens" posteriorly. The sacrum and ilia have an extracapsular, dorsally located articulation (synarthrosis), which is augmented by the vast interosseous ligament that provides considerable internal stability. The SIJ is encased in a capsule, with a smooth anterior wall and irregular bands/ligaments comprising the posterior wall.[118]

Besides small internal pelvic motions of the SIJ and symphysis, substantial motion of the external pelvic platform takes place. Movements of the pelvic platform upon the hip joints, such as flexion and extension (pelvic anteversion and retroversion), rotation, and abduction/adduction, strongly influence lumbar and SIJ movement.[118] Coupled hip flexion and extension play a key role in establishing lower spine lordosis and kyphosis.[64] The SIJs are postulated to act as important stress relievers in the "force-motion" relationships between the trunk and legs. These joints ensure that the pelvic girdle is not a solid ring of bone that could easily fracture under the great forces to which it might be subjected, either from trauma or its many bipedal functions.[67] Analysis of gait mechanics demonstrates that the SIJs provide sufficient flexibility for the intrapelvic forces to be transferred effectively to and from the lumbar spine and legs.[64]

The ventrally directed angle between L5 and the sacrum tends to become more acute when loaded as the sacrum nutates. Accordingly, the thick anterior longitudinal ligament spans the ventral aspect of L5 and S1, buttressing against excessive extension.[118]

Biomechanical calculations show the influence that a higher friction coefficient and greater wedge angle of the sacrum have on SIJ stability.[119,120] It was suggested that during juvenile growth, lever arms like the spine and legs generate an increasing force until full body weight is reached. Consequently, the SIJ is dynamically modified by changing form closure in the direction and strength of imposed forces.[120] Disturbed or excessive force transfer through the SIJ can exaggerate compressional or torsional stresses on these joints. Such altered transmission to the spine and legs can cause tissue effects with deleterious consequences.[23,74]

In contrast to excessive SIJ force closure, a counter-opposing condition of diminished stability occurs in pregnancy-related pelvic girdle pain (PGP).[117] Insufficient and/or asymmetric compression of the SIJs can result in PGP.[22,81,119,120] Nonoptimal load transfers and clinical effects would be expected to occur from either the suspected excessive pelvic and SIJ stiffness[64,74] or the documented insufficient pelvic girdle stability with PGP.[81,117] Sufficient SIJ force closure can be defined as the amount needed to provide the necessary stiffness for the particular demands of static or dynamic load transfer, at optimal utilization

of energy.[115,117,120] Thus, stability is an instantaneous phenomenon and is antagonistic to instability.[77] Neither too little nor too much SIJ stability, from either mechanical stiffness properties or force closure/compression, is optimal.[118]

Biomechanics and PGP—European guidelines[117] for PGP define sacroiliac and symphyseal pain: “PGP generally arises in relation to pregnancy, trauma, arthritis and osteoarthritis. Pain is experienced between the posterior iliac crest and the gluteal fold, particularly in the vicinity of the SIJ. The pain may radiate in the posterior thigh and can also occur in conjunction with/or separately in the symphysis. The endurance capacity for patients standing, walking and sitting is diminished. The diagnosis of PGP can be reached after exclusion of lumbar causes. The pain of functional disturbances in relation to PGP must be reproducible by evidence based specific clinical tests.”

The following clinical tests are recommended for PGP (European guideline[117]): the posterior pelvic pain provocation test (P4/thigh thrust test), the Patrick (flexion, abduction, and external rotation) test, the Gaenslen test, pain with palpation of the long dorsal SIJ ligament, the symphysis palpation test, and the modified Trendelenburg test. The recommended functional test is the active straight leg raise.

The biomechanical model of PGP is based on the concept that altered motor function of the deep abdominal muscles in PGP leads to insufficient bracing of the SIJs/pelvis. This is combined with weakened erector muscles and fascia over the lower lumbar spine and SIJ.[118] Studies demonstrate that, among other muscles, contraction of the internal oblique and transversus abdominis results in force closing of the pelvic ring.[4,5,52,53] Conversely, in patients with PGP, a maladaptive compensatory pattern occurs, characterized by diminished activity of these muscles and a subsequent failure to brace the pelvis.[52]

As evidence in support of the biomechanical model, Sturesson et al[101] showed that painful movement in patients with the most severe PGP can be instantly reduced by an external Hoffmann-Slätis surgical frame. This finding agrees with studies using pelvic belts to normalize SIJ movement.[81,82,119] Application of the external fixator in patients with chronic severe PGP generates an anterior compression on the ilia, leading to effective force closure.[101] Using Roentgen stereophotogram-metric analysis, precise measurement shows how the surgical frame changes the position of the SIJ from counternutation to nutation, which reduces pain in the long dorsal ligament and, possibly, deeper dorsal SIJ ligaments.[101,118] When successful external frame application is verified with diminished pain after several weeks, the frame is removed, followed by surgical SIJ arthrodesis. This methodology is only indicated in patients with severe PGP who failed intense rehabilitation, and with confirmation of the appropriateness of SIJ arthrodesis by a successful fixator frame trial. It is important to note that activation of transversely oriented abdominal muscles also reduces SIJ laxity,[87] providing the foundation for an active approach to control these joints as an alternative to surgery for some.

In conclusion, as in other joints, the SIJ is highly innervated from L3 to S2, and when PGP occurs,[118] the dorsal SIJ ligaments especially are targeted. This represents a clearly defined biomechanical subgroup of pain.

COUNTERPOINT RESPONSE

Authors of the point and counterpoint agree that chronic nonspecific LBP has biological, psychological, and social components to various extents in different people, and that biomechanics plays a role in the development of LBP. None of the biomechanical models presented above suggests that biomechanics research alone should be the desired approach. Whichever treatment approach is decided upon, it seems obvious that it should be based on a biopsychosocial assessment that includes biomechanics. The question, therefore, is not whether biomechanics alone should be the desired approach, but whether biomechanics is a sufficiently dominant factor in a high-enough proportion of cases to make it worth including as an explanation for pain generation, moderation, or mediation in LBP.

There are 2 primary sources of evidence to suggest that biomechanics is a sufficiently dominant factor in LBP. The first is that most LBP is aggravated or relieved by movements and postures. For example, there are reports that systematically correcting biomechanical impairments during symptom-provoking movements and postures results in an immediate improvement in LBP symptoms.[73,106,112] Treatment directed at correcting the impairments, particularly training a person to make corrections during performance of daily activities, results in short- and long-term improvements in both functional and biomechanical outcomes.[45,50,51,62,107,108] Importantly, people with LBP also are more likely to adhere to correcting performance of movements and postures during daily activities than traditional therapeutic exercise.[107] Thus, if a biomechanical impairment contributes to a patient's LBP presentation, then training the patient to correct the impairment during daily activities should facilitate both short- and long-term improvement because of the repeated opportunity to practice across the day.

The second source is the discovery of measurable biomechanical markers for LBP (eg, differences in intervertebral motion[8,9,80]). As for neuropathological and vascular disorders, the pursuit of quantitative biomarkers for LBP should continue and should include biomechanics. These should join research into inflammatory, neuropathic, and muscle metabolic markers, to name a few, to give a more complete picture of LBP by correlating them with each other and with symptomatology, clinical examination findings, and outcomes.

Subgroups of patients with LBP for use in RCTs based on mechanical biomarkers will require more and better research into their identification, validation, and interactions, as well as their roles as prognostic factors, moderators, and mediators. Subgrouping will also require greater sophistication in methods and a move beyond surface markers placed on the skin overlying the spine in the laboratory and cadaveric studies. There have been calls for biomechanics research to address dynamic, multiseg-mental issues in vivo as well as in cadaveric models.[3,56,84] Although it seems to be agreed that biomechanics research has not yet reached adequate refinement to achieve its highest impact, we argue that it is on its way and will likely be relevant to more than a narrow subset of patient presentations.

We offer the following to address the 2 questions posed initially. In response to the question of whether current biomechanics research has the potential to advance treatment of LBP, it

appears that the answer is yes, but mainly recently and not to the exclusion of other factors. In response to whether this will lead to better treatment strategies for LBP, the answer is quite likely, so long as individualized and intrinsic biomechanics is investigated with sufficient depth and rigor.

CONCLUSION

THREE MODELS PRESENTED IN THE counterpoint to illustrate how biomechanics is being used to understand the problem of LBP and guide treatment are based on sound anatomical, biological, and mechanical principles, which ensures internal validity. Furthermore, the described relationship between pain and biomechanical measures is supported by a body of research demonstrating, for example, differences between patients with LBP and controls. Each author has contributed to new knowledge by developing and refining these models of LBP. However, the question still remains whether these predominantly biomechanical representations of LBP can lead to intervention strategies that are superior to other interventions known to have only small to moderate effects on pain for a broad spectrum of patients.[5]

Whether any of these approaches, or others, based on biomechanics can advance outcomes is not yet clear. All do include some consideration of factors beyond biomechanics. This concurs with the view that a reductionist approach focusing only on biomechanics will not provide the solution for the LBP problem, and further underscores the need for a multidisciplinary approach. If LBP is indeed a very complex, multifactorial problem, then a much broader view, such as a systems approach,[2,38] that accommodates other biopsychosocial factors and their interactions, even when biomechanical issues may be dominant presenting factors, is necessary. Work is under way to truly integrate understanding from across the diverse biopsychosocial domains,[17] and approaches are being proposed to achieve this integration in guiding understanding and management of LBP.[13,42,43,116] This new approach must consider the massively multifactorial character of LBP, including nonspecific treatment effects of various therapies for LBP.

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SYNOPSIS:

Although biomechanics plays a role in the development and perhaps the persistent or recurrent nature of low back pain (LBP), whether biomechanics alone can provide the basis for intervention is debated. Biomechanics, which refers to the mechanics of the body, including its neuromuscular control, has been studied extensively in LBP. But, can gains be made in understanding LBP by research focused on this component of biology in the multifactorial biopsychosocial problem of LBP? This commentary considers whether biomechanics research has the potential to advance treatment of LBP, and how likely it is that this research will lead to better treatment strategies. A point-counterpoint format is taken to present both sides of the argument. First, the challenges faced by an approach that considers biomechanics in isolation are presented. Next, we describe 3 models that place substantial emphasis on biomechanical factors. Finally, reactions to each point are presented as a foundation for further research and clinical practice to progress understanding of the place for biomechanics in guiding treatment of LBP.

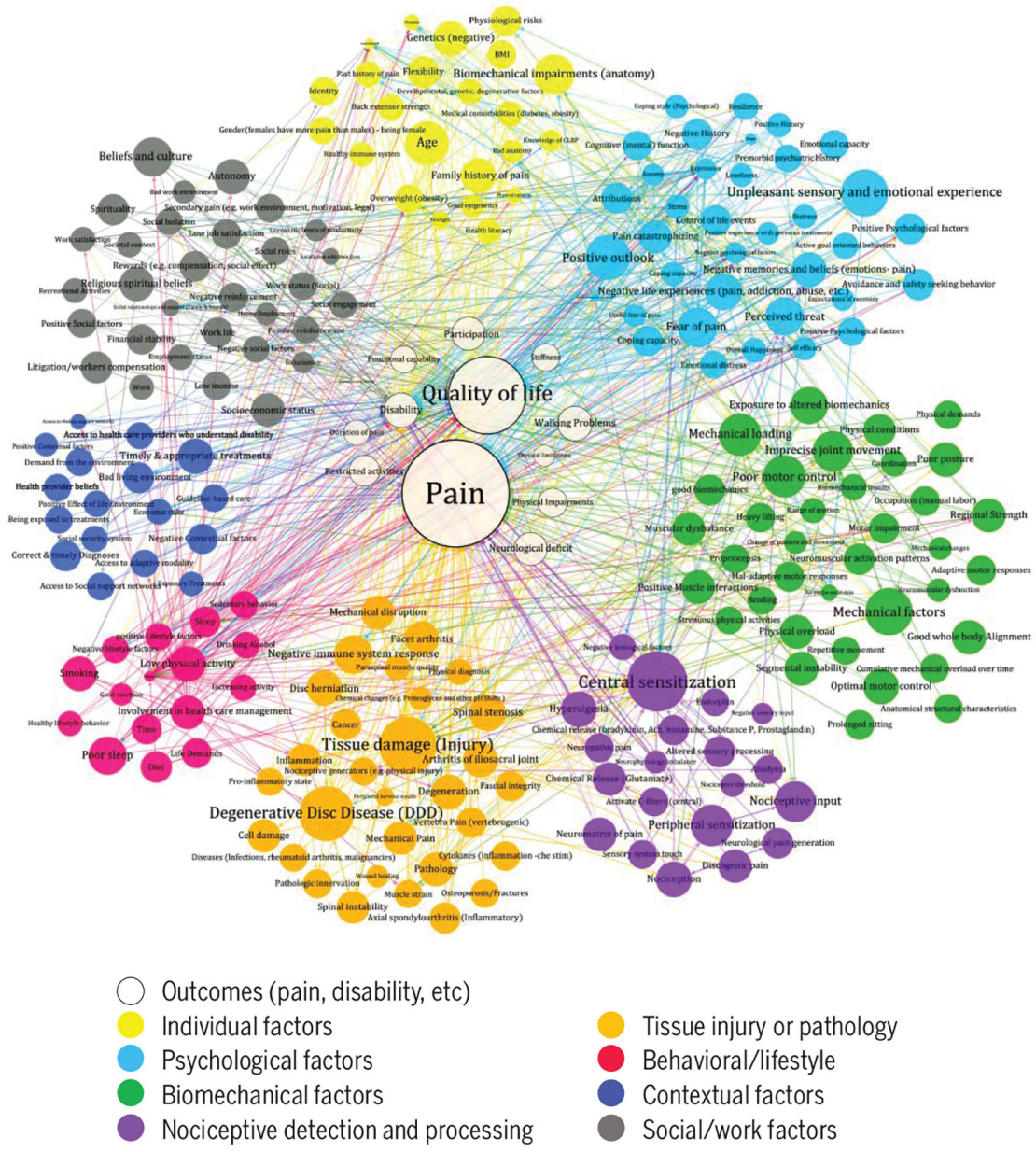

FIGURE 1.

A metamodel illustrating factors (colored circles) contributing to low back pain, disability, quality of life, and other outcomes (white circles) and their interactions (colored lines). This metamodel was constructed with input from the multidisciplinary panel of 27 experts in preparation for the symposium at the 26th Annual Meeting of the North American Spine Society (2017). Diameters of the circles are proportional to the number of experts identifying these factors and the number and strength of connections with other factors.

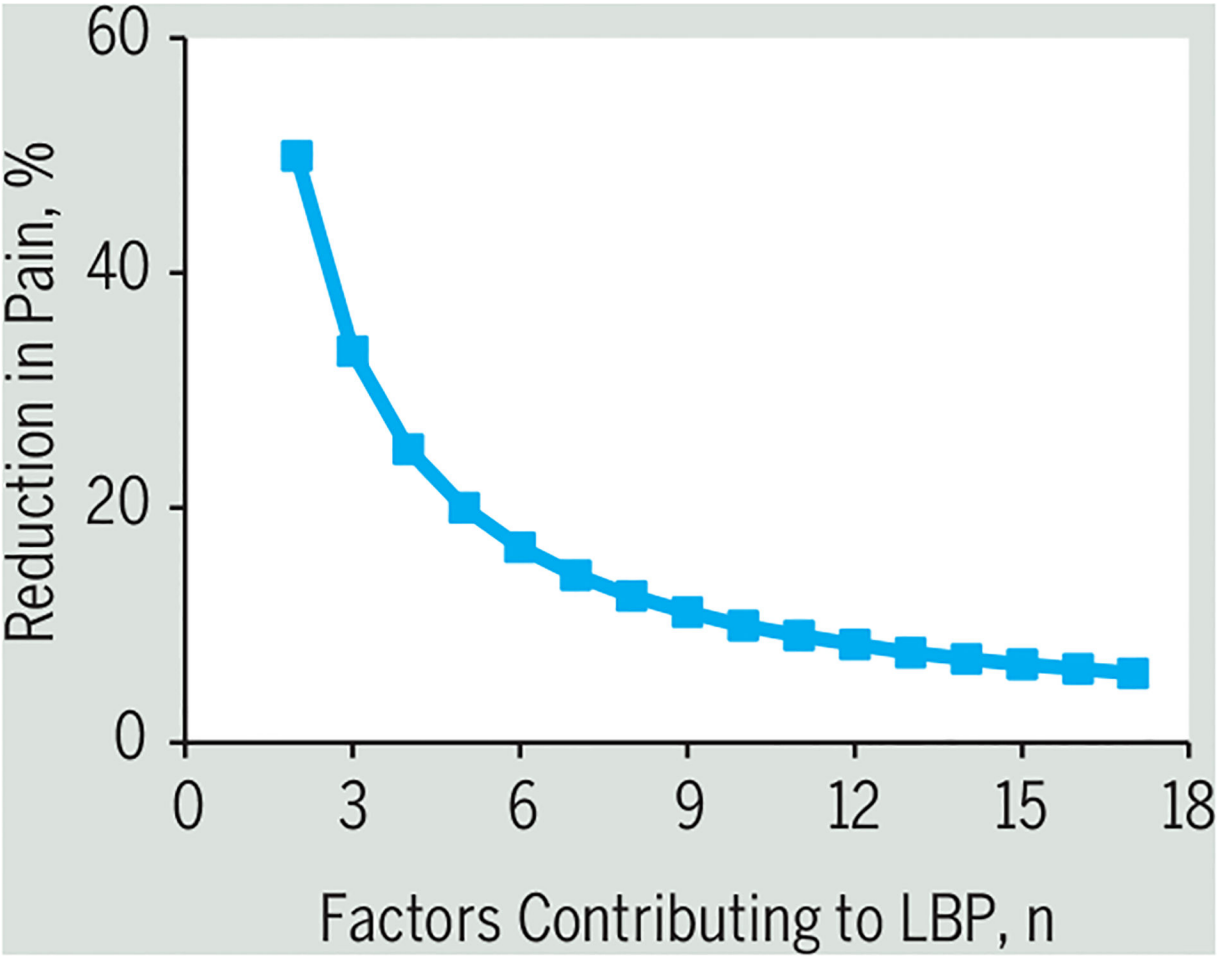

FIGURE 2.

Mathematical simulation of the predicted reduction in pain when the number of factors contributing to LBP that must be addressed with treatment is considered. On the vertical axis is the predicted success when a single factor that contributes the most to LBP is addressed with treatment. As the number of factors contributing to LBP increases, the effectiveness of such an intervention decreases. Abbreviation: LBP, low back pain.

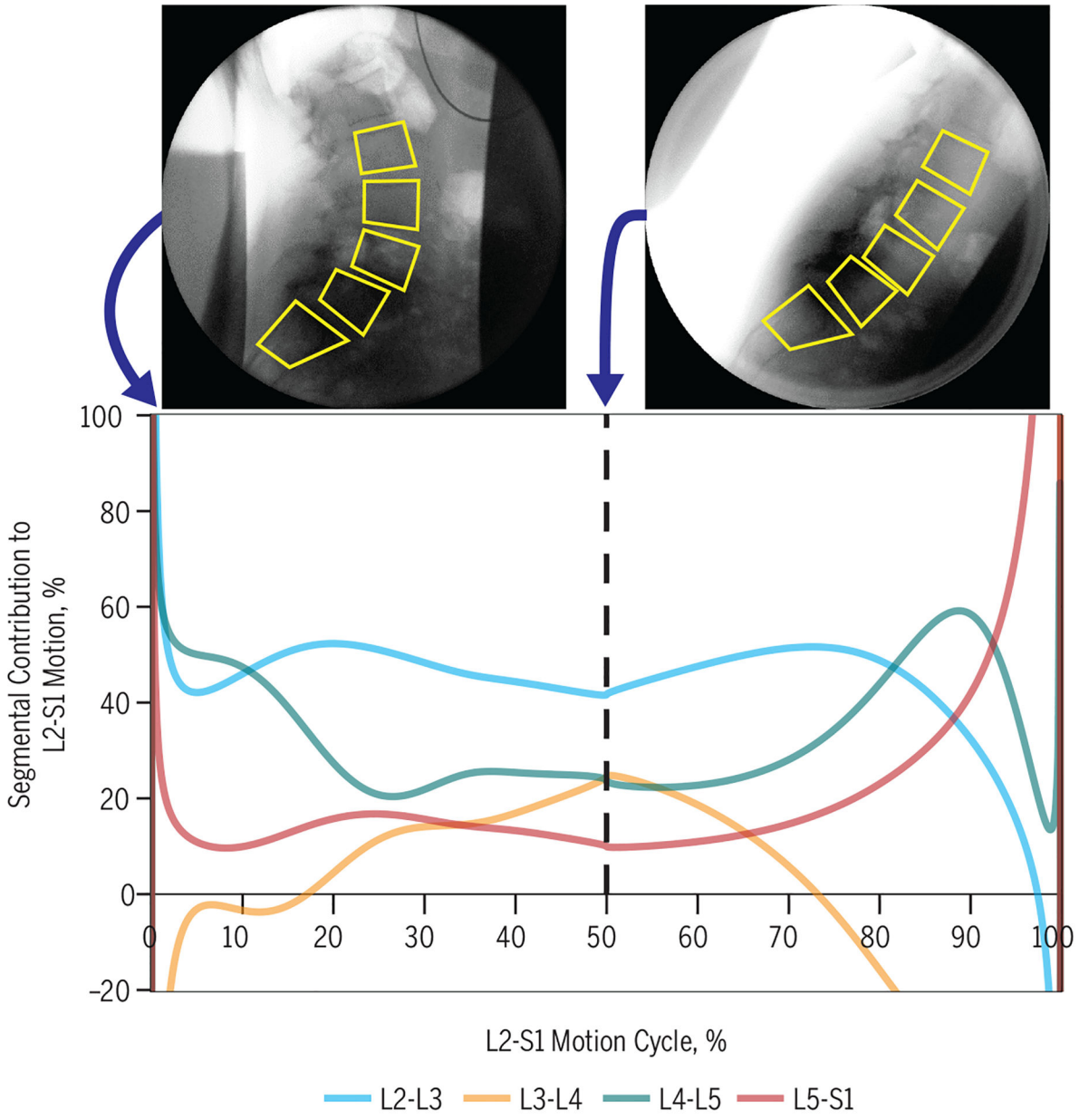

FIGURE 3.

Continuous proportional weight-bearing flexion intervertebral motion in a 63-year-old female patient with spondylolisthesis. Note that the segmental contributions to the total L2-S1 motion change continuously. On average, L2-L3 makes a higher contribution than the upper reference range of a control population, and the L4-L5 average share is in the normal range.

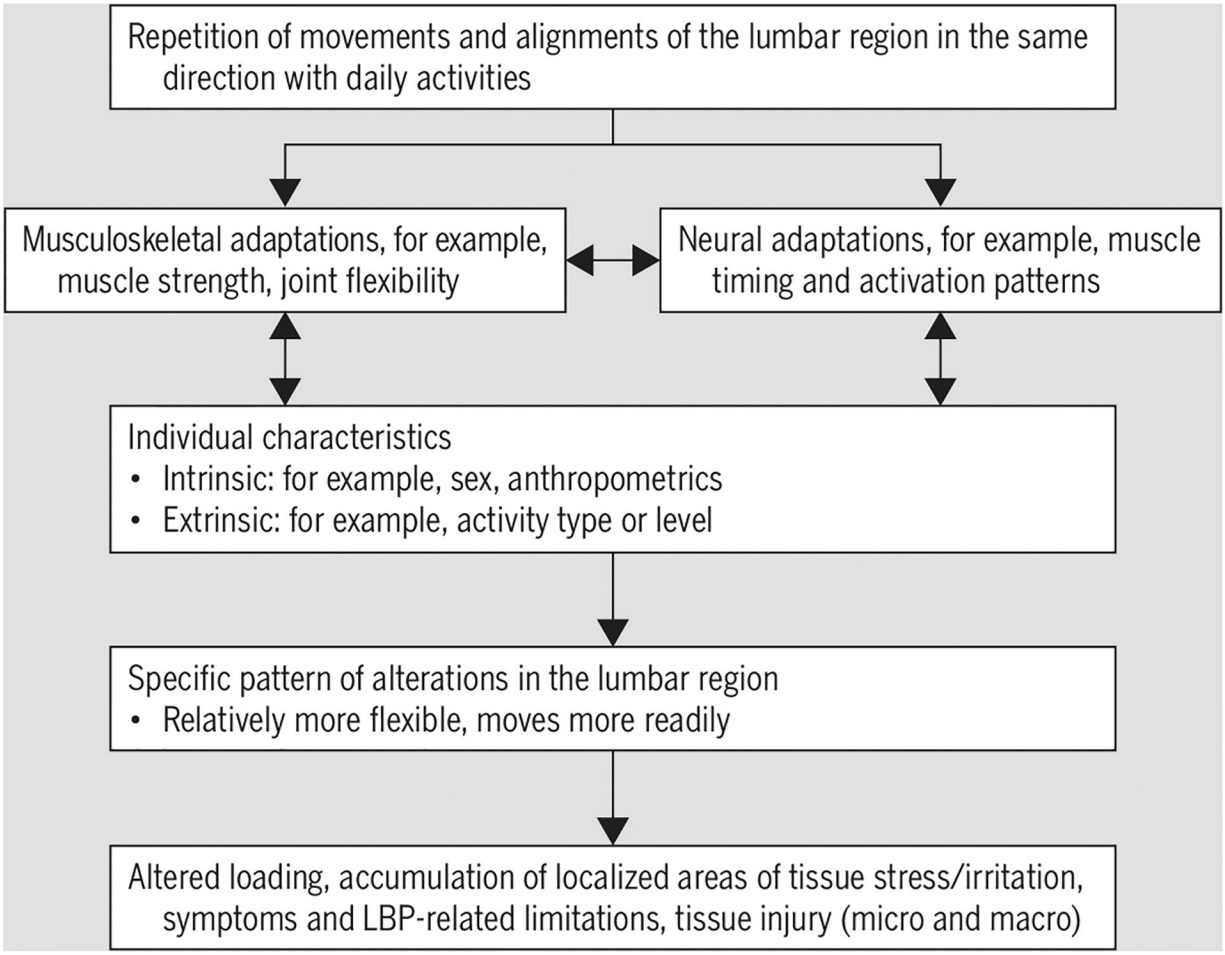

FIGURE 4.
Illustration of the mechanically related processes proposed to contribute to the development and course of LBP based on the kinesiopathologic model. Abbreviation: LBP, low back pain.

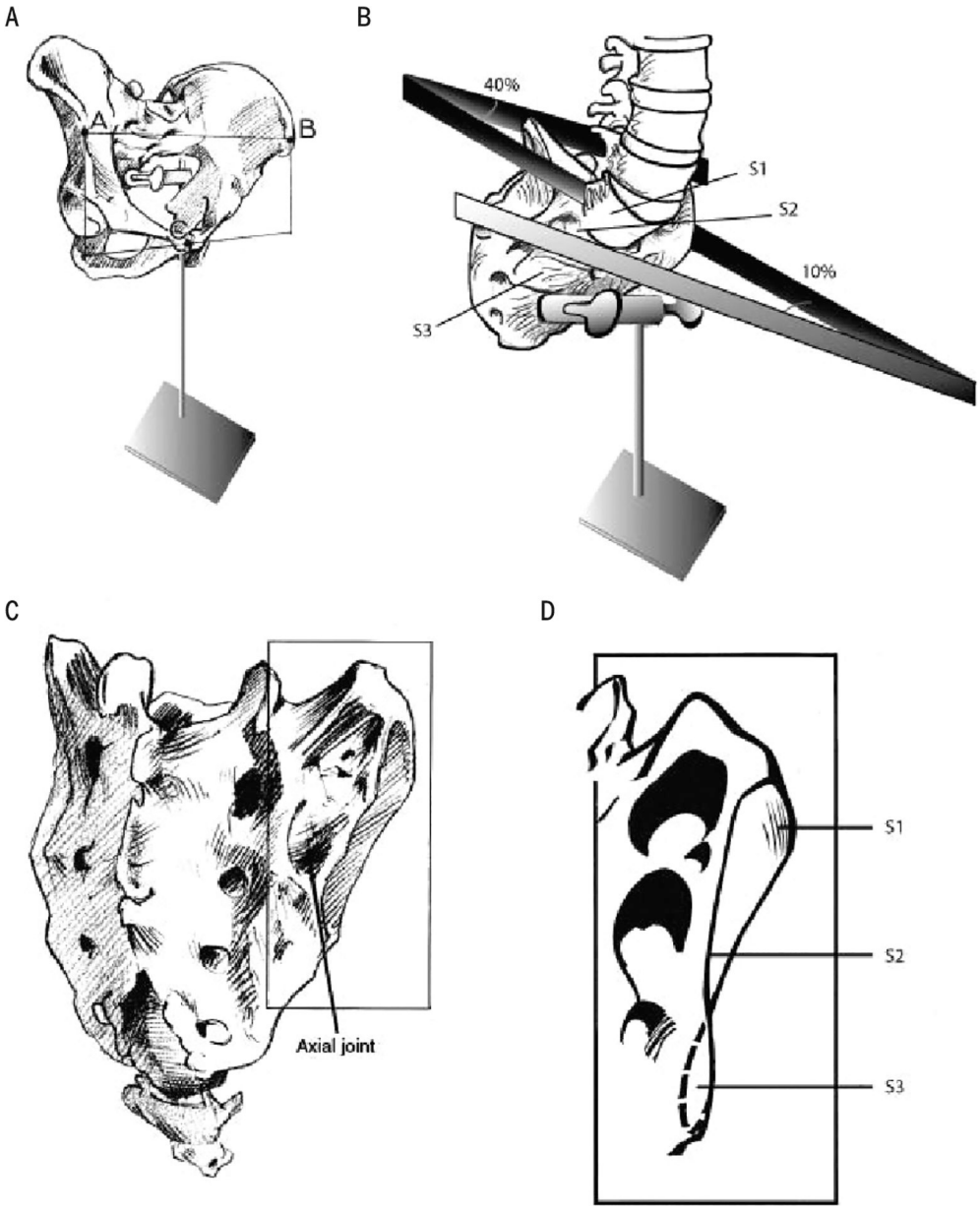

FIGURE 5.

(A) The pelvis in erect posture. (B) View of the sacrum from the ventrolateral side, showing the different angles between left and right sacral articular surfaces. (C) Dorsolateral view of the sacrum. The pointer indicates a cavity in the sacrum, in which an iliac tubercle fits, called the “axial” sacroiliac joint. (D) Sacral articular surface at the right side. The different angles reflect the propeller-like shape of an adult sacroiliac joint.