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REVIEW ARTICLE

Discussion paper: what happened to the 'bio' in the bio-psycho-social model of low back pain?

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Abstract
Purpose Over 20 years ago the term non-specific low back pain became popular to convey the limitations of our knowledge of the pathological source of most people's low back pain. Knowledge of underlying pathology has advanced little since then, despite limited improvements in outcomes for patients with low back pain.
Methods This paper discusses potential misunderstandings related to diagnostic studies in the field of low back pain and argues that future diagnostic studies should include and investigate pathological sources of low back pain.
Results Six potential misunderstandings are discussed. (1) Until diagnosis is shown to improve outcomes it is not worth investigating; (2) without a gold standard it is not possible to investigate diagnosis of low back pain; (3) the presence of pathology in some people without low back pain means it is not important; (4) dismissal of the ability to diagnose low back pain in clinical guidelines is supported by the same level of evidence as recommendations for therapy; (5) suggesting use of a diagnostic test in research is misinterpreted as endorsing its use in current clinical practice; (6) we seem to have forgotten the 'bio' in biopsychosocial low back pain.
Conclusions We believe the misunderstandings presented in this paper partly explain the lack of investigation into pathology as an important component of the low back pain experience. A better understanding of the biological component of low back pain in relation, and in addition, to psychosocial factors is important for a more rational approach to management of low back pain.

Keywords Low back pain · Diagnosis · Back pain

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Introduction

Over the last three decades there has been a major shift in the clinical and research approach to low back pain. Prior to this, clinical practice and research activities were mainly based upon a biomedical model with patients receiving specific pathoanatomical diagnoses and treatments directed to these diagnoses. A notable example of this type of thinking is Mixter and Barr's 1934 classic paper describing surgical treatment of disc prolapse [1]. This traditional approach was challenged in 1987 by two landmark publications that correctly pointed out that most diagnoses were nominal and of doubtful validity. Gordon Waddell's seminal paper titled "A new clinical model for the treatment of

low-back pain" [2] introduced the biopsychosocial model of low back pain, emphasizing the distinction between pain and disability and the need to address the biological, psychological and social aspects of the condition. He promoted the term "simple back pain" to describe the majority of patients with this common symptom in whom a specific source of pain could not legitimately be identified. The report of the Quebec task force [3] used the term "non-specific spinal disorder" to describe the same patients. Importantly while both documents highlighted the problems with existing diagnostic tests they also identified the need for future research to identify methods and tests that would allow clinicians to determine the source of a patient's pain [2, 3].

Clinical practice guidelines for low back pain have existed since the late 1980s and now uniformly endorse an approach where most patients do not receive a patho-anatomical diagnosis [4]. Once patients with radiculopathy and serious causes of back pain (such as cancer) are excluded, the remaining patients, approximately 90%, are provided with the label 'non-specific low back pain' or some equivalent term. In these patients a patho-anatomic diagnosis is not pursued but instead clinicians apply generic symptomatic treatments such as advice to stay active and avoid bed-rest, analgesic medicines, exercise and manipulation. While this approach is simple, it does not work particularly well, especially not in those patients with a tendency towards persistence or recurrence of their pain and disability over time. Yet more effective alternatives for patient care are not apparent. The limitations of current approaches are further illustrated by the many systematic reviews of treatments for low back pain that reveal existing treatments for non-specific low back pain have, at best, only small effects [5, 6].

One potential explanation for the lack of effectiveness of treatments for non-specific low back pain is that clinicians are unable to direct treatment to the specific pathology underlying back pain and instead rely upon generic treatments in heterogeneous patients. In most areas of medicine, diagnosis is considered the cornerstone of effective management, but this is not a common view in the back pain field. In fact, related diagnostic research is quite uncommon. A focus of today's clinical research in the field of low back pain is on identifying subgroups of patients with a (un)favorable prognosis or likely to respond favorably to specific treatments. The classification of these subgroups, however, is seldom based on pathoanatomical findings or diagnosis. We contend that important research into possible sources of low back pain has been stifled by a misunderstanding of the biopsychosocial model of low back pain, including the term non-specific low back pain. The danger is that potentially important breakthroughs in the understanding and management of low back pain are not made. This paper discusses some potential misunderstandings related to diagnosis studies in the field of low back pain and argues that future diagnostic studies should also include and investigate biomedical sources of low back pain.

Key concept: low back pain is a symptom not a disease

A fundamental issue is that low back pain is a symptom and not a disease. Like many symptoms (e.g. shortness of breath, abdominal pain), low back pain could arise from several different pathologies. Typically in other areas of medicine when a patient presents with a symptom (e.g. shortness of breath) investigations focus on identifying pathologies or diseases known to cause the symptom (e.g. does a patient with shortness of breath have emphysema, coronary artery disease, asthma, lung cancer, etc.?). In low back pain this would involve trying to identify patients who have a pathology which is capable of causing the symptom of low back pain, either through referred pain or directly involving the lumbar spine. For example, can we identify patients who have pain resulting from an annular tear, muscle injury, facet joint osteoarthritis, endplate defects, disc degeneration etc. [7, 8]? We acknowledge that based upon our current limited knowledge of the patho-anatomical basis for low back pain, a symptom-based approach to diagnosis and management is most likely the best current approach to clinical management. However, it is important to consider whether the current symptom-based focus in low back pain research might hamper the generation of new diagnostic knowledge on which to base more effective strategies in the future.

Misunderstanding 1: until diagnosis is shown to improve outcomes it is not worth investigating

A common argument against research into the pathology responsible for low back pain is that currently there is no evidence that diagnosis improves patients' outcomes [9]. We agree a diagnostic test should be shown to improve patient outcomes before it is recommended for clinical use. However, lack of evidence that a diagnostic test improves outcomes does not mean the diagnosis is incorrect. Also lack of evidence is not the same as evidence that a diagnosis is not improving outcome. A recent Volvo award winning paper [10] concluded discography was not a valid test for identifying discogenic low back pain because patients with positive discography did not respond well to spinal fusion. In this study response to treatment was considered the "gold standard" for the validity of the

diagnostic test. Yet it is not possible for a diagnosis to influence patients' outcomes if no effective treatment exists for the specific disease or pathology identified. Although, the study may have demonstrated that discography results are not useful as an indication for fusion, an important clinical finding, it does not necessarily demonstrate that discography does not identify a painful disc.

Diagnosis may be of value even without the availability of effective treatment as it may provide a logical avenue for the development and testing of future interventions. Although we await verification of results through replication, such an example comes from a recent paper by Peng et al. [11] who found improvements in chronic low back pain when a new treatment (intradiscal methylene blue injection) was developed and tested to target a specific pathology (innervation of annular fissures). The paper illustrates the potential for pathoanatomic research to result in the discovery of new effective methods of treatment. There are many examples in medicine where the pathology/disease was identified prior to any effective treatments being available, but resulted in the later development of highly effective interventions. An example is the recent discovery that infection is the cause in many stomach ulcers [12] and antibiotic treatment not previously considered a treatment option is now widely used. A better understanding of the pathological source of low back pain will likely precede, and be a pre-requisite for, the identification of new effective treatments for low back pain.

Misunderstanding 2: without a gold standard it is not possible to investigate diagnosis of low back pain

The requirement of a gold standard for low back pain diagnostic research is not consistent with contemporary understanding in the diagnostic field. There are very few diseases for which a gold standard is available [13]. A reference standard is the best available standard and is used for most diagnostic research. Current reference tests for the tissue source of low back pain including discography and anaesthetic injections may not be perfect, but have reasonable face validity when performed according to recommended criteria. Imperfect reference standards will most likely reduce rather than inflate the diagnostic accuracy of clinical diagnostic tests. It is therefore not logical to dismiss significant diagnostic test accuracy of clinical index tests (e.g. MRI findings) when compared to reference tests (e.g. discography) because of controversy over the reference test. Despite this, controversy about the reference standards for tissue sources of low back pain is likely to persist. An alternate approach suggested by Rutjes et al. [13] may be more productive for future back pain research. This approach involves abandoning the traditional test accuracy paradigm (index test compared to reference test) and directly validating the index tests compared to important clinical outcomes [13]. An example in back pain would be investigating specific pathology on MRI as predictors of the development or course of back pain, in longitudinal cohort studies. Currently almost no quality literature of this type exists.

Misunderstanding 3: the presence of pathology in some people without low back pain means it is not important

Probably the most common argument against the importance of some pathologies believed to be capable of producing low back pain is that cross-sectional studies have reported these same pathologies in people without low back pain. Notable examples include MRI findings such as disc bulges or degeneration in people without pain [14–16]. While this is an important finding it does not automatically exclude the possibility that these pathologies can cause low back pain. For example, it may be that the pathology needs to be quite advanced before it produces low back pain. It is noteworthy that in knee osteoarthritis or cardiovascular disease the pathology (thinning of the articular cartilage of the knee or blockage of a coronary artery) can be very advanced in some patients before they experience symptoms or signs of the disease [17]. A recent cross-sectional population study of over 1,000 people found some degree of lumbar disc degeneration was present in the majority of people [18]. However, there was a strong association between the severity of disc degeneration and low back pain. While 20% of those with no degeneration still experienced low back pain the rate increased to over 60% in those with the highest levels of degeneration [18]. Another recent study showed that disc space narrowing, especially at two or more spinal levels was strongly associated with the presence of low back pain in the elderly [19]. Pathologies including endplate (Modic) changes have been shown to be rare in those without low back pain and far more common in those with back pain [20]. A recent systematic review found the prevalence of modic changes was only 6% in non-clinical populations while the prevalence was 43% in patients with non-specific low back pain [20]. Therefore dismissing the importance of pathology because it exists in some people without low back pain seems premature. Importantly because most research in this area is cross-sectional there is almost no quality evidence on whether lumbar pathology observed on imaging predicts the development of low back pain, course of low back pain (including recurrences) or response to specific interventions.

Misunderstanding 4: dismissal of the ability to diagnose low back pain in clinical guidelines is supported by the same level of evidence as recommendations for therapy

International guidelines uniformly recommend a diagnostic triage where 90% of patients with low back pain receive the diagnosis non-specific low back pain [4]. The European guidelines, for example, state "It is, however, well-accepted that in most cases of acute low back pain it is not possible to arrive at a diagnosis based on detectable pathological changes" [21]. Intriguingly no evidence is provided or discussed to support this statement about the inability to diagnose, while similar statements about treatment efficacy are always substantiated by citation of primary studies or systematic reviews.

Misunderstanding 5: suggesting use of a diagnostic test in research is misinterpreted as endorsing its use in current clinical practice

It is essential to differentiate between investigating diagnostic tests to improve understanding of low back pain, and investigating if use of tests in clinical practice improves patient outcomes. Currently there is no evidence that imaging improves outcomes for primary care patients with low back pain but this does not mean that studies should not investigate a better understanding of the pathology identified on imaging by recruiting patients from primary care with back pain. The European guidelines point out that there is "no evidence on the association between degenerative signs at the acute stage and the transition to chronic symptoms" [21]. This highlights a limitation in our knowledge base, namely a lack of studies investigating if pathology identified on imaging predicts the future course of low back pain, including recovery from the current episode and likelihood of recurrences. A few previous studies have investigated clinical (e.g. psychosocial) predictors of future recurrences. In one such study only the number of previous episodes was associated with recurrences within the next year [22]. Predicting and limiting future recurrences is a growing area of interest in low back pain research. There is a reasonable rationale for pathology identified on imaging to be associated with future recurrences in the same way as carotid artery occlusion in people having a TIA is highly associated with the chance of a future stroke or death. To dismiss investigations of imaging in clinical populations, which aim to better understand the source and causes of low back pain, because imaging is currently not recommended for low back pain is missing the point of this line of research.

Misunderstanding 6: forgetting the bio in biopsychosocial

It is widely accepted that low back pain is a biopsychosocial condition. Our concern is that current research has forgotten the biological component. As an example there are systematic reviews of psychosocial predictors of developing chronicity after an episode of low back pain [23]. However, to our knowledge, there are no quality studies investigating the biological component as a predictor of chronicity. This situation appears clearly unbalanced especially as the evidence is strong that while psychosocial factors play a role they explain only a small portion of the prognosis or course of low back pain [23, 24]. If we believe low back pain is a biopsychosocial condition then we need to investigate all components and interactions between them, expecting each component to have varying degrees of importance in different patients.

Suggestions for future research

Future research investigating the full biopsychosocial spectrum of low back pain is important, namely research which investigates biological and psychosocial factors concurrently. Such research would enable the independent value of different components to be identified as well as important interactions between them. Low back pain in any individual may be caused by a single or several concurrent pathologic entities and there are many factors that may influence the pain and disability experienced. Therefore, it is likely we will gain a better understanding of prognosis, mediators and effect modifiers by investigating all domains of the biopsychosocial model.

It seems reasonable that biological and psychosocial factors could independently predict outcome from back pain. Importantly biological and psychosocial factors may interact, with psychosocial factors being more or less important in people with differing degrees of pathology. We are unaware of any large longitudinal studies simultaneously investigating a range of biological and psychosocial factors in the prognosis of low back pain.

The search for subgroups of patients who respond best to specific interventions has been identified as a research priority [25]. For some low back pain interventions such as lumbar disc replacement or spinal fusion there is a theoretical rationale why pathology may identify people who respond best to the intervention. However, psychosocial factors are likely to also modify treatment response and as such investigation of effect modifiers from the full biopsychosocial spectrum seems most likely to identify clinically important subgroups. Other recommended treatments for low back pain such as manipulation and exercise lack a

clear rationale for how they work. However, it remains reasonable that patients with different pathologies or different degrees of pathology may respond quite differently to these interventions. A further benefit of this line of research is that it might shed further light on the mechanisms by which some of the treatments for low back pain work, especially if (changes in) biological markers are studied over time. As an example a recent study found preliminary evidence of immediate increase in diffusion of water within the L5-S1 disc in people who had immediate improvement after mobilisation and exercise [26].

Biological factors requiring further investigation are broader than potential nociceptive sources and include central modulation of pain and physical impairments. While these factors are unlikely to represent the primary source of pain they may be important contributors to the development, persistence or recurrence of low back pain. Major advances have been made in the understanding of central modulation of pain but little is known about these factors as predictors of outcome or effect modifiers. Altered motor control of trunk muscles is one example of a physical impairment potentially related to low back pain [27]. There is a theoretical rationale why altered motor control may predict response to exercise aimed at normalising motor control but currently no quality studies directly investigating this have been published. Currently little is known about the importance of most physical impairments as predictors of prognosis or response to intervention.

Summary

Over 20 years ago the term non-specific low back pain became popular to convey the limitations of our knowledge of the pathological source of most people's low back pain. Unfortunately, knowledge of underlying pathology has advanced little since then and there seems to be an acceptance that this will always be the case, despite limited improvements in outcomes for patients with low back pain. Research over recent decades has focussed heavily on the psychosocial domain, possibly at the expense of biological factors. Misunderstandings presented in this paper may partly explain the lack of investigation into pathology as an important component of the low back pain experience. While not advocating a return to widespread imaging or inappropriate diagnostic testing, this paper highlights the need for research aiming to better understand the biological component of low back pain in relation, and in addition, to the psychosocial factors. In his 1987 paper Gordon Waddell argued we must "develop a rational basis for choosing the most effective treatment for individual patients" [2]. This is not possible without better understanding of the biological component of the biopsychosocial model.

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