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# Biopsychosocial risk factors associated with chronic low back pain after lower limb amputation

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ABSTRACT

Low back pain is a common secondary health condition after lower limb amputation with important implications related to functional capabilities and overall quality of life. Despite the high prevalence of low back pain after lower limb amputation, the underlying etiologies of the disorder remain unknown. This hypothesis-driven communication provides evidence in support of using the multifactorial, biopsychosocial model of low back pain experience in the general population for identification of potential risk factors and rehabilitation targets for low back pain after lower limb amputation. Key findings that link biological, psychological, and social factors and the experience of low back pain in the general patient population with LBP are discussed while highlighting gaps in our current state of knowledge related to the association of these factor and presence of low back pain after lower limb amputation. Importantly, the aim of this communication was not to propose a new model, but rather to organize data originating from prior work into a coherent hypothesis-driven conceptual framework to better understand the need for multifaceted and multidisciplinary intervention approaches for effective treatment of low back pain after lower limb amputation.

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## Introduction

Low back pain (LBP) is a common health condition worldwide, with 11–38% of the general population reporting symptoms over a one year period [1,2]. LBP is currently considered the leading cause of disability globally, ahead of 290 other conditions, and is responsible for 83 million years lived with disability [3]. Additionally, LBP is a major source of activity limitation, work absenteeism, and increased cost of medical care throughout much of the world [2,4–6]. LBP is also a common and perhaps more impactful, secondary health condition after lower limb amputation (LLA), with high estimated annual prevalence rates between 50–90% [7–13]. Individuals with LLA often report more LBP after amputation than before [8,9] and in most cases directly attribute their LBP to their amputation [10]. Additionally, presence of LBP daily or several times per week has been associated with moderate to severe physical disability and limitations in performing daily activities in patients with LLA [8,9,13–15]. To this end, LBP is often rated by patients with LLA as more bothersome than phantom or residual limb pain [11], suggesting LBP is an important secondary musculoskeletal condition associated with functional limitation and disability after LLA.

Despite the high prevalence of LBP after LLA, the exact etiologies of the disorder in this population remain unknown, thereby making its treatment exceptionally challenging. Importantly, there are currently no published randomized clinical trials or clinical practice guidelines specifically tailored toward the management of LBP for individuals with LLA. Therefore, there exists a clear need for comprehensive identification of contributing factors to the LBP

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experience after LLA that can serve as a basis for the development of targeted rehabilitation strategies and future research investigations. Here a new application of the multifactorial, biopsychosocial model for LBP, previously developed for the general population [16–18], is proposed as a way of identifying risk factors and potential intervention targets for treatment of LBP after LLA. The objective of this hypothesis-driven communication was to organize data originating from prior studies of the biopsychosocial correlates of LBP after LLA into a coherent conceptual framework. We hypothesized that alterations in biological, psychological, and social factors are related to the development of LBP symptoms and disability after LLA that merit specific attention during the clinical decision making process and for future research efforts to improve patient-related outcomes.

### *The biopsychosocial model of low back pain*

Treatment of LBP has historically centered around the traditional biomedical model of illness, which assumes a direct relationship between regional pathoanatomy and the perception of pain [18]. As such, it was expected that once the anatomical source of LBP is identified, biochemical and/or mechanical treatments of underlying pathoanatomy would result in cessation of pain. Despite leading to successful treatment of many other disease processes, the outcomes of interventions based on the biomedical model have proven to be less than ideal for treatment of LBP [18–20]. One potential reason for the failure of the biomedical model to provide an effective treatment option for LBP is that no single underlying pathoanatomical lesion has been consistently identified [18], with up to 85% of LBP patients left without a precise pathoanatomical diagnosis [21]. Additionally, determining the pathoanatomical sources of LBP frequently lacks interexaminer reliability and adequate evidence for generalizability [22]. The often equivocal outcomes from many "lesion-specific" treatment options such as intra-articular corticosteroid injections [19] and spinal fusion surgeries [20], along with the generally poor predictive value of diagnostic imaging to identify pathoanatomical sources of pain [23], have led to a recent paradigm shift toward a "non-structural" approach for the management of LBP [24].

A growing body of evidence now suggests that successful treatment of LBP should include biological, psychological, and social assessments to comprehensively address the patient's unique pain experience [18]. The so called "biopsychosocial model" of LBP suggests that the patient's perceptions and reactions to pain should also be considered as these factors often lead to unnecessary avoidance of physical activity and social interactions, work absenteeism, and high health care utilization [16,17]. Whereas the pathoanatomy may initiate the pain process, the psychological and social factors appear to play an important role in exacerbating the biological component of LBP by influencing the perception of pain [25]. For example, it has been hypothesized that the presence of mechanical LBP can lead to a pain-generated stress response that could have a negative impact on the endocrine and immune systems, which in turn may negatively affect the cognitive assessment, emotional response, coping strategies and health practices of the individual [26].

## The hypothesis

Proponents of the biopsychosocial model argue that the complex, multidimensional nature of LBP does not lend itself to the reductionist view of the biomedical model; instead, the patient's unique biologic, psychological, and social factors must equally be considered [18]. Therefore, the term biopsychosocial implies that the biological, psychological and social factors are interwoven within the context of the patient's overall LBP experience and should be directly and concurrently considered as a part of a comprehensive treatment program [26]. In support of this theory, multidisciplinary treatment approaches that include biopsychosocial components for treatment of LBP in adults have demonstrated positive effects on pain, disability, and health-related quality of life [27,28]. It stands to reason that LLA likely amplifies and/or alters specific components within the multifactorial biopsychosocial model of LBP, previously suggested for the general population. Given that LLA may differentially affect the various components of this model (Fig. 1), we hypothesize that discriminating clinically meaningful sub-groups of patients with LBP after LLA will most likely require assessments of biological, psychological and social domains [22].

### *Biological factors*

#### *Biomechanics*

Altered mechanics of gait and movement have been historically proposed to play a causative role in the development and/or recurrence of LBP after LLA [29]. In fact, persons with LLA perceive "uneven postures and compensatory movements" affected by "fatigue" and "prosthesis-related factors" during functional activities as the primary contributors to LBP [30]. Though at the expense of higher metabolic cost of transport [31], compensatory movement strategies adopted after LLA typically involve adaptations to maintain the body's center of mass within the base of support (i.e., improve stability and balance), primarily with a preference for the intact limb, if applicable [32]. During gait, for example, the intact limb (relative to prosthetic limb) is characterized by a longer stance time, shorter step length, wider stride width, and larger vertical ground reaction forces [33]. As the trunk accounts for approximately two-thirds of total body mass [34], altered motions of this segment play a substantial role in post-amputation movement strategies, thereby warranting more trunk-focused biomechanical investigations for assessing potential links with the development and persistence of LBP.

Altered trunk and pelvic movements in persons with LLA have been previously identified in all three cardinal planes, including larger forward trunk lean and flexion-extension range of motion, greater lateral trunk flexion (towards the prosthetic limb) and pelvic obliquity motion, as well as more axial rotations between the shoulders/pelvis or regional/intervertebral motion segments [35,36]. The presence (and likely severity) of LBP further influences such trunk and pelvic movements [37]. For instance, it has been reported that patients with transfemoral amputation and LBP elevate their pelvis on the intact side, minimize their lumbar lateral flexion, and keep their lumbar spine rotated toward the prosthetic limb throughout the gait cycle as compared to patients with transfemoral amputation without LBP [38].

LBP has also been associated with more in-phase mediolateral coordination between the trunk and pelvis [39], which is indicative of inter-segmental rigidity (i.e., "guarding behavior") previously reported in able-bodied individuals who are experiencing LBP [40,41]. Additional evidence suggests that individuals with LLA employ an active mediolateral trunk movement strategy, inferred from increases in generation and absorption of energy between the trunk and pelvis [42,43]. Although actively increasing mediolateral trunk sway is likely an attempt to improve joint stability within the lower extremity by altering lever arms of ab/adductor musculature [44], most notably within the hip among patients with transfemoral amputation [7], such strategies have been associated with LBP/discomfort among able-bodied individuals performing gait training aimed at reducing knee joint loads via trunk lateral flexion [45].