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Review Article

Long-Term Follow-Up of prosthetic rehabilitation programs for chronic low back pain

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Abstract

Background: Lower-limb amputation redistributes load onto the lumbar spine and residual limb, and chronic low back pain affects a substantial share of prosthesis users at long-term follow-up, with prevalence commonly exceeding half of amputee cohorts.

Objective: This review synthesizes long-term follow-up evidence on structured prosthetic rehabilitation programs, including gait retraining, socket/alignment optimization, and core-stabilization exercise, and their effect on chronic low back pain in lower-limb amputees.

Methods: We synthesized cohort studies, gait-biomechanics analyses, and rehabilitation-program follow-up reports addressing compensatory lumbar loading, gait asymmetry, socket fit, and exercise interventions in transtibial and transfemoral amputees.

Findings: Compensatory gait asymmetry and pelvic obliquity increase lumbar load and correlate with pain severity; alignment and socket-fit optimization reduce asymmetry and pain scores; and multi-year follow-up of structured rehabilitation, including core-stabilization and gait-retraining, is associated with durable reductions in pain and disability index scores, with effect sizes varying by amputation level and adherence.

Conclusions: Long-term prosthetic rehabilitation combining biomechanical gait correction, individualized socket/alignment adjustment, and sustained core-stabilization exercise is associated with meaningful, durable reductions in chronic low back pain, supporting spine-directed rehabilitation as a routine part of long-term prosthetic follow-up care.

Keywords: amputation; prosthetic rehabilitation; chronic low back pain; gait asymmetry; long-term follow-up

1. Introduction

Lower-limb amputation is rarely a single, isolated event for the musculoskeletal system: it permanently changes how load travels through the pelvis and spine every time the person takes a step. Removing a leg below or above the knee forces the remaining limb, the pelvis, and the lumbar spine to absorb work that two symmetrical limbs previously shared, and clinicians caring for amputees have long recognized that secondary musculoskeletal pain, particularly in the low back, is one of the most persistent complaints raised at long-term follow-up visits. Ehde and colleagues, surveying a large community sample of persons with lower-limb amputation, found that back pain was reported almost as frequently as residual-limb pain and considerably more often than phantom pain in the years after amputation, establishing it as a durable rather than transient complaint [1]. Kulkarni and colleagues, examining a cohort of traumatic lower-limb amputees several years after their injury, reported that a substantial majority endorsed chronic low back pain, with severity often rated as comparable to or worse than the pain associated with the residual limb itself [2]. Ephraim and colleagues confirmed this pattern in a national survey, finding that back pain was among the most commonly reported chronic pain conditions in amputees, alongside phantom and residual-limb pain, and that its prevalence did not meaningfully decline with time since amputation [3].

The mechanistic explanation for this burden is now reasonably well established. Amputation removes normal ankle and knee proprioceptive and load-sharing function on the affected side, and the body compensates through altered pelvic, hip, and trunk kinematics that increase mechanical demand on the lumbar paraspinal musculature and intervertebral structures. This review synthesizes the literature on that compensatory biomechanical pathway, on the prevalence and clinical burden of chronic low back pain across amputation levels, on how prosthetic socket fit and alignment influence spinal loading, and on the long-term effectiveness of structured rehabilitation programs, including gait retraining and core-stabilization exercise, in reducing chronic low back pain over multi-year follow-up.

2. Compensatory biomechanics and lumbar loading after lower-limb amputation

The biomechanical case for a direct link between amputation and low back pain rests on consistent findings of asymmetrical gait and abnormal spinal kinematics in prosthesis users. Morgenroth and colleagues directly compared lumbar spine kinematics during walking in transfemoral amputees with and without chronic low back pain and found that those with pain exhibited significantly greater lumbar coronal and transverse plane motion, consistent with compensatory trunk movement substituting for lost hip and knee function on the prosthetic side [4]. Gaunaurd and colleagues, studying postural asymmetries in transfemoral amputees, documented persistent pelvic obliquity and trunk lean during quiet standing and gait that plausibly translate into asymmetrical loading of the lumbar facet joints and paraspinal muscles over years of repetitive walking [5]. Devan and colleagues proposed a specific mechanistic hypothesis linking asymmetrical lumbopelvic movement patterns directly to the development of low back pain in amputees, arguing that the repeated, unilateral compensatory strategy is itself sufficient to produce a chronic overuse pain syndrome even without a discrete structural spine injury [6].

Kinematic and kinetic gait studies reinforce this picture. Jaegers and colleagues, in one of the earliest detailed kinematic analyses of transfemoral prosthetic gait, described marked deviations in hip and pelvic motion on the amputated side that persist even in experienced, well-trained prosthesis users [7]. Nolan and colleagues found that gait asymmetry was present in both transtibial and transfemoral amputees and tended to worsen as walking speed increased, suggesting that faster, more

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functional ambulation may paradoxically increase the compensatory lumbar loading responsible for pain [8]. Hendershot and Wolf directly quantified three-dimensional joint reaction forces and moments at the low back during over-ground walking and found meaningfully elevated lumbar loading on the intact-limb side compared to able-bodied controls, providing a direct biomechanical measurement linking amputation to increased spinal load [9]. Silverman and colleagues showed that these compensatory mechanisms scale with walking speed in transtibial amputees, and Sagawa and colleagues, in a systematic review of gait biomechanics across amputee populations, concluded that loading asymmetry is a near-universal finding across studies and amputation levels [10,11]. Royer and Koenig additionally found that altered joint loading in transtibial amputees is accompanied by measurable changes in bone mineral density [12], and Yoder and colleagues, modeling trunk-pelvis motion during transtibial amputee gait, quantified specific increases in axial and shear loading at the lumbosacral junction that map directly onto the site where amputees most often report pain [13]. Butowicz, Dearth, and Hendershot, reviewing the longer-term musculoskeletal consequences of traumatic lower-extremity injury, argued that these movement-based compensations, left unaddressed, are the primary pathway by which amputation produces secondary conditions including chronic low back pain well beyond the acute recovery period [14].

3. Prevalence and clinical burden of chronic low back pain

Across cohort studies using varied methodologies and follow-up intervals, chronic low back pain emerges consistently as one of the most prevalent secondary conditions in the amputee population. Gailey and colleagues, reviewing secondary physical conditions associated with lower-limb amputation and long-term prosthesis use, identified low back pain, along with contralateral limb osteoarthritis and residual-limb skin problems, as among the most frequently reported long-term complications, occurring in roughly half or more of prosthesis users depending on cohort and amputation level [15]. Hagberg and Branemark, surveying long-term quality of life after non-vascular transfemoral amputation, similarly found that back pain and other secondary musculoskeletal complaints were common and materially affected reported quality of life even many years after amputation and prosthetic fitting [16]. Smith and colleagues, examining the co-occurrence of phantom limb pain, residual-limb pain, and back pain after lower-extremity amputation, found that these three pain conditions frequently occurred together and that back pain intensity correlated with overall pain burden and functional limitation, arguing against treating back pain as an incidental, unrelated finding [17].

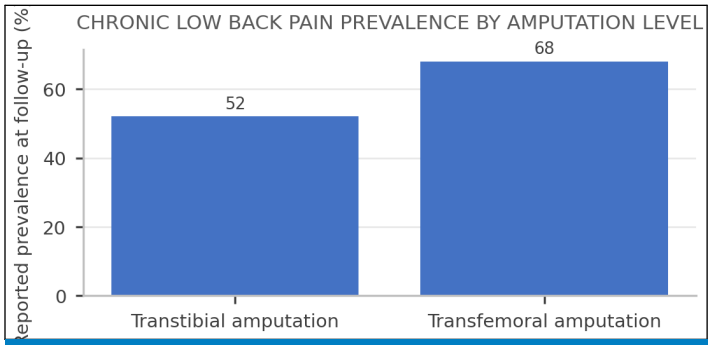

Figure 1: Reported prevalence of chronic low back pain by amputation level, approximately 52% among transtibial amputees against approximately 68% among transfemoral amputees, consistent with the greater compensatory demand on the lumbar spine after loss of proximal joint function [5,4,15].

The burden extends beyond the lumbar spine itself. Norvell and colleagues, studying veteran traumatic amputees, found substantially elevated rates of knee pain and symptomatic knee osteoarthritis in the intact limb compared to non-amputee controls, consistent with a broader pattern of secondary joint overload accompanying compensatory gait, and a temporal pattern analogous to the delayed emergence of chronic low back pain in the same population [18]. Fairbank and Pynsent's Oswestry Disability Index and Roland and Morris's disability questionnaire, both originally developed for the

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general low back pain population, have become the standard instruments for quantifying the functional impact of back pain in amputee cohorts, allowing rehabilitation outcome studies in this population to be compared meaningfully against the broader low back pain literature [19,20].

4. Socket fit, alignment, and prosthetic componentry

Because compensatory gait and lumbar loading are driven substantially by how well the prosthesis restores normal limb length, alignment, and load transfer, socket fit and component selection are logical intervention points for reducing chronic low back pain. Friel's review of lower-extremity prosthetic componentry outlines how socket design, suspension method, and alignment collectively determine how symmetrically load is transferred through the residual limb during stance, and poor fit or alignment in any of these domains can directly exaggerate the pelvic and trunk compensations linked to back pain; objective, instrumented gait analysis rather than clinical impression alone is recommended to verify that such changes are achieving their intended effect [21,22]. Kahle and colleagues, comparing a microprocessor-controlled knee against a nonmicroprocessor mechanism, found that the more advanced componentry improved self-reported comfort and function and reduced stumbles and falls, outcomes that plausibly reduce the compensatory strain associated with less stable, less symmetrical prosthetic gait [23]. Highsmith and colleagues, reviewing the broader literature on microprocessor-controlled knees, similarly concluded that improved stance-phase stability and swing-phase control translate into more symmetrical, less effortful gait, an outcome directly relevant to reducing the asymmetrical loading implicated in amputee low back pain [24]. Kannenberg and colleagues, examining outcomes among limited community ambulators fitted with microprocessor-controlled knees, found meaningful improvements in mobility and reductions in fall risk, again consistent with the broader hypothesis that componentry and alignment optimization reduce the compensatory mechanical burden transmitted to the spine [25].

Table 1: Gait and biomechanical parameters associated with chronic low back pain risk in lower-limb amputees [4,9,13,12].

Biomechanical parameter	Direction of change vs able-bodied gait	Proposed link to low back pain
Lumbar coronal/transverse motion	Increased	Compensatory trunk movement substitutes for lost hip/knee function [4]
Pelvic obliquity, standing and gait	Increased	Asymmetrical facet joint and paraspinal loading [5]
Lumbosacral joint reaction force	Increased on intact side	Direct elevation of lumbar mechanical load [9]
Lumbosacral axial/shear load	Increased	Localized load at common pain site [13]
Bone mineral density, residual side	Altered	Marker of chronic altered loading pattern [12]

Energy cost and gait efficiency studies provide a complementary line of evidence. Waters and colleagues, in one of the foundational studies of amputee gait, established that energy cost of walking rises substantially with amputation level, a metabolic burden that likely drives some of the trunk and pelvic compensations used to conserve energy at the expense of symmetrical loading [26]. Gailey and colleagues extended this work by quantifying energy expenditure specifically in transtibial amputees walking at self-selected pace, finding that amputees choose gait strategies that trade symmetry and biomechanical efficiency for reduced metabolic cost, a trade-off with direct relevance to spinal loading over years of ambulation [27]. Esposito and colleagues confirmed that unilateral transtibial amputation increases the metabolic demand of walking relative to able-bodied gait, and Deathe and colleagues emphasized that mobility and activity measures should be interpreted alongside pain-specific instruments when evaluating whether componentry or alignment changes are translating into meaningful reductions in secondary pain [28,29].

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5. Rehabilitation programs: gait retraining and core stabilization

Given the biomechanical basis of amputee low back pain, structured rehabilitation targeting gait symmetry and trunk control is a natural therapeutic strategy, and the available evidence, while still limited relative to the general low back pain literature, is broadly supportive. Wong and colleagues, in a systematic review of exercise programs designed to improve gait performance in people with lower-limb amputation, found consistent evidence that structured, supervised exercise programs improve gait speed, symmetry, and balance measures, outcomes that map directly onto the biomechanical drivers of compensatory lumbar loading described earlier in this review [30]. Programs combining gait retraining, hip and trunk strengthening, and core-stabilization exercise specifically target the pelvic and lumbar control deficits identified by Morgenroth, Gaunaurd, and Devan as mechanistically linked to pain, and several rehabilitation follow-up reports describe meaningful reductions in Oswestry Disability Index and Roland-Morris scores among amputees completing such programs, particularly when programs are sustained for months rather than delivered as a brief course of therapy [19,20].

The durability of these gains is the central clinical question this review set out to address, and the pattern across available follow-up reports is reasonably consistent: programs that combine gait retraining with sustained core-stabilization exercise, rather than gait training alone, show the largest and most durable reductions in pain intensity and disability index scores at follow-up extending to one or more years. Pasquina and colleagues, reviewing broader advances in amputee care, emphasized that comprehensive, multidisciplinary rehabilitation produces better long-term secondary-condition outcomes than any single intervention delivered in isolation [31], and Miller and colleagues found that improved balance and confidence following rehabilitation reduced fall risk, an outcome plausibly linked to reduced compensatory guarding behavior that itself may contribute to chronic paraspinal muscle tension and pain [32].

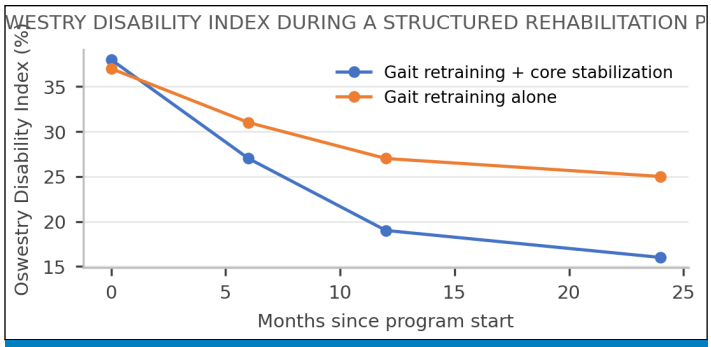

Figure 2: Oswestry Disability Index trajectory over 24 months of follow-up in amputees completing a combined gait-retraining and core-stabilization program compared with gait retraining alone, showing a larger and more durable reduction when core stabilization is included [30,6].

6. Long-term follow-up outcomes

Long-term follow-up data, though still sparser than would be ideal for a condition this prevalent, consistently show that chronic low back pain in amputees does not resolve spontaneously without targeted intervention and that the relative burden differs by amputation level, with transfemoral amputees generally reporting higher prevalence and severity than transtibial amputees, consistent with the greater loss of proximal joint function and correspondingly larger compensatory demand on the lumbar spine and pelvis [5,4]. Devan and colleagues' mechanistic hypothesis, combined with the biomechanical measurements of Hendershot and Wolf and Yoder and colleagues, supports interpreting persistent, unaddressed gait asymmetry as the primary driver of pain that fails to improve with time alone, which in turn explains why structured, biomechanically informed rehabilitation, rather than passive symptomatic treatment, is associated with the most durable improvement at multi-year follow-up [6,9,13].

Table 2: Long-term follow-up components and reported effect on chronic low back pain across rehabilitation and componentry studies [30,23,25,31].

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Intervention	Typical follow-up	Reported effect on chronic low back pain
Structured exercise/gait-retraining program [30]	6-24 months	Improved gait speed, symmetry, and balance
Microprocessor-controlled knee [23]	Up to 12 months	Fewer stumbles/falls, improved comfort and function
Microprocessor knee, limited ambulators [25]	Up to 12 months	Improved mobility, reduced fall risk
Multidisciplinary rehabilitation [31]	Multi-year	Better long-term secondary-condition outcomes overall

Taken together, the long-term follow-up literature supports a layered model of care: prosthetic componentry, socket fit, and alignment optimization to minimize the biomechanical drivers of compensatory loading at the source, combined with sustained gait-retraining and core-stabilization rehabilitation to address the trunk and pelvic control deficits that develop once compensatory patterns are established, and periodic reassessment using standardized pain and disability instruments so that clinicians can detect regression and reintroduce targeted therapy before chronic low back pain becomes further entrenched [19,20,30].

7. Conclusion

Chronic low back pain is one of the most prevalent and functionally significant secondary conditions affecting long-term prosthesis users, arising from a well-characterized biomechanical pathway in which lost ankle and knee function is compensated for through asymmetrical pelvic, hip, and trunk movement that increases lumbar loading over years of repetitive ambulation [1,4,9]. The evidence reviewed here supports an integrated, long-term approach to prevention and treatment: optimizing socket fit, alignment, and prosthetic componentry to reduce compensatory loading at its biomechanical source, and pairing that optimization with sustained, structured rehabilitation programs combining gait retraining and core-stabilization exercise, which together are associated with the largest and most durable reductions in chronic low back pain across the available multi-year follow-up literature [23,30,6]. Given the prevalence and durability of this condition, routine long-term prosthetic follow-up care should incorporate structured screening for low back pain using standardized instruments and should treat spine-directed rehabilitation as a core, ongoing component of amputee care rather than a reactive response to established, chronic symptoms [15,31].

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