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REVIEW ARTICLE

Exercise-Induced Inflammation and Immune Responses in Para-Athletes: Implications for Performance, Training, and Health

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Abstract. Exercise is a potent regulator of immune and inflammatory function, yet its impact in para-athletes remains insufficiently characterized due to the physiological diversity imposed by varying impairments. This narrative review synthesizes current evidence on exercise-induced inflammation and immunity across para-sport populations, examining both adaptive and maladaptive processes in relation to training, recovery, and performance. Para-athletes with spinal cord injury, limb deficiency, or cerebral palsy display distinct immunological signatures, influenced by autonomic disruption, altered muscle mass distribution, and neuromotor control deficits. While moderate exercise elicits beneficial cytokine responses and enhances immune surveillance, excessive or unaccustomed training can precipitate chronic low-grade inflammation, delayed recovery, and heightened infection susceptibility. Factors such as exercise modality, intensity, and environmental context further modulate these responses. Evidence supports targeted nutritional and rehabilitative interventions including omega-3 fatty acids, vitamin D optimization, antioxidant timing, and neuromuscular electrical stimulation as potential modulators of inflammatory balance. However, methodological limitations, small sample sizes, and inconsistent reporting hinder generalization. A paradigm shift toward impairment-specific, longitudinal, and mechanistic investigations is essential to advance clinical and applied knowledge in this field. Collectively, understanding the nuanced interplay between exercise, inflammation, and immunity in para-athletes can inform safer training strategies, enhance performance resilience, and support long-term health outcomes unique to this growing athletic population.

Keywords: Athletes with Disabilities; Inflammation; Immune System; Exercise Physiology; Sports Medicine



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Introduction

Exercise-induced inflammatory and immune responses represent a central physiological axis by which physical activity influences adaptation, recovery, performance, and long-term health. While the broad contours of exercise immunology have been elaborated in able-bodied populations, these paradigms cannot be assumed to generalize to athletes with permanent impairments because baseline immunophysiology, autonomic regulation, tissue exposure, comorbid burden and environmental interactions differ substantially across para-sport cohorts (Nieman & Wentz, 2019; Peake, Neubauer, Walsh, & Simpson, 2017). This issue has become clinically and operationally pressing: participation and competitiveness in para-sport have expanded globally, elite para-athletes now face intensified training and competition schedules, and recent position statements emphasize tailored health surveillance for para-athlete populations (Pinheiro et al., 2024). Importantly, evidence indicates that some impairment groups (notably persons with spinal cord injury and certain neuromotor disorders) carry altered baseline markers of systemic inflammation and immune function, and also display unique infection and morbidity profiles that may interact with training stress to influence availability and performance (Valido et al., 2023; Sterner et al., 2023). Consequently, a para-athlete-specific synthesis of exercise-induced inflammatory and immune responses is necessary for evidence-based training, recovery and medical care.

Clear definitions delimit the scope of this review. “Exercise-induced inflammation” is used to denote the stimulus-dependent, often transient mobilization and local/systemic signaling of inflammatory mediators (for example, interleukin-6 functioning as a myokine, tumor necrosis factor- α , and C-reactive protein) together with downstream processes of tissue repair and metabolic signaling that follow acute and repeated bouts of exercise. “Immune responses” refers to integrated innate and adaptive cellular and humoral adjustments changes in neutrophil and monocyte activation, lymphocyte trafficking and function (including natural killer activity), and mucosal immune markers that are modulated immediately after exercise and across training cycles. “Para-athletes” are defined as individuals with permanent physical or sensory impairments who train and compete in para-sport contexts (including spinal cord injury, limb amputation or deficiency, cerebral palsy and visual impairment among the internationally recognized Paralympic impairment classes). Throughout the review we maintain a strict para-athlete focus: inferences and recommendations are derived from studies in which para-athlete data are disaggregated and reported specifically, and comparative references to able-bodied cohorts are employed only when necessary for mechanistic clarification (Sellami, Puce, & Bragazzi, 2023).

Heterogeneity across para-athlete populations is both extensive and mechanistically relevant. The level and completeness of a spinal cord lesion exert profound effects on autonomic (sympathetic) outflow, thermoregulation and cortisol dynamics each of which modulates leukocyte redistribution and cytokine kinetics whereas in athletes with limb loss, differences in residual limb health, prosthetic interfaces and the amount of actively contracting muscle mass alter myokine release and local inflammatory signaling (Valido et al., 2023; Pinheiro et al., 2024). Neuromotor impairments such as cerebral palsy introduce variability in muscle recruitment patterns, spasticity and oxidative stress, all of which modify exercise tolerance and immune signaling. Additional modifiers common in some para populations include higher prevalence of

chronic low-grade inflammation related to skin integrity issues (pressure injuries), recurrent infections, medication burdens and altered body composition; these contextual factors can shift the balance between adaptive inflammatory signaling that supports repair and maladaptive chronic inflammation that undermines recovery and performance (Sterner et al., 2023). Recognizing and reporting these impairment-specific modifiers is therefore essential for meaningful exercise-immune research and for translating findings into practice.

This narrative review adopts a para-athlete-centric lens and a transparent, impairment-aware synthesis approach. We integrate human studies that directly examine exercise-induced inflammatory and immune phenomena in para-athlete cohorts, emphasize mechanism where human data permit, and explicitly grade evidence strength where small samples or methodological heterogeneity constrain inference. By confining evidence and interpretation to para-athlete populations and by foregrounding impairment-specific mechanisms, the review aims to deliver an actionable framework for researchers, clinicians and high-performance teams seeking to optimize both performance and health in para-sport.

Methodology

For this narrative review, a transparent and reproducible methodology was adopted to ensure rigor and clarity. Comprehensive searches were conducted across multiple bibliographic databases, including PubMed/MEDLINE, Embase, and Scopus, encompassing literature from database inception through May 1, 2026. Core search terms combined three conceptual domains: para-athlete populations (e.g., “para-athlete,” “Paralympic,” “spinal cord injury,” “amputation,” “cerebral palsy”), exercise modalities (e.g., “exercise,” “training,” “physical activity”), and inflammatory/immune outcomes (e.g., “inflammation,” “cytokine,” “immune response,” “myokine”). Boolean operators, truncation, and controlled vocabulary were employed where appropriate to maximize sensitivity while minimizing irrelevant retrievals. Searches were restricted to studies published in English. In addition, reference lists of all included studies, recent systematic reviews, and position statements from key disability-sport organizations were hand-searched to identify further eligible publications.

Eligibility criteria were defined a priori to maintain relevance and scientific integrity. Studies were included if they investigated human participants classified as para-athletes or athletes with specified permanent physical or sensory impairments, including spinal cord injury, limb amputation, cerebral palsy, or visual impairment, who were engaged in organized training or competition. Study designs considered eligible encompassed experimental trials, observational cohort or cross-sectional studies, mechanistic investigations, and case series; selected animal studies were included only if translationally applicable to para-athlete physiology. Studies were excluded when para-athlete data were combined with able-bodied participants in a way that precluded extraction of separate results. No restriction was imposed on exercise modality, duration, or intensity, but studies had to report at least one immune or inflammatory outcome, whether systemic (e.g., cytokines, leukocyte counts) or tissue-specific, with or without concurrent performance or clinical measures.

Study selection followed a two-step screening process. Titles and abstracts were initially screened for relevance, followed by full-text evaluation against inclusion criteria. Discrepancies in study eligibility were resolved by consensus among the review team. Data extracted from each included study encompassed

participant demographics, impairment type and classification, exercise modality, intensity, duration and frequency, immune and inflammatory outcome measures, clinical or performance endpoints, and relevant confounders such as medication use or comorbid conditions. Extracted data were recorded in a standardized spreadsheet to ensure consistency and traceability. To account for heterogeneity and variable study quality, a pragmatic risk-of-bias and methodological appraisal approach was applied. Elements were adapted from the NIH Study Quality Assessment Tools and Joanna Briggs Institute checklists, focusing on sample size adequacy, clarity of impairment characterization, appropriateness of immune/inflammatory measures, and transparency in reporting participant selection and confounding variables. Particular caution was applied when interpreting results from small cohorts, mixed impairment groups, or studies with limited follow-up or incomplete reporting.

Synthesis was conducted narratively, structured around thematic domains corresponding to major para-athlete classifications and immune/inflammatory mechanisms. Where possible, findings were tabulated to compare exercise modalities, intensity, and impairment-specific outcomes. Consistent patterns, discrepancies, and gaps in the literature were highlighted, with interpretive commentary provided in light of methodological limitations and mechanistic plausibility. No formal meta-analysis was undertaken due to anticipated heterogeneity in study design, participant characteristics, and outcome measures, and this limitation is transparently acknowledged.

Physiological foundations: key mechanisms linking exercise to inflammation and immunity in para-athletes.

Exercise exerts profound effects on both the innate and adaptive arms of the immune system, modulating cellular activity and systemic signaling in ways that facilitate adaptation and recovery, yet may also transiently increase susceptibility to infection if poorly managed. In para-athletes, these responses are further influenced by impairment-specific physiological constraints. Key innate immune responses to exercise include transient leukocytosis, neutrophil and monocyte activation, and natural killer (NK) cell mobilization. Adaptive responses encompass shifts in T- and B-lymphocyte numbers, trafficking patterns, and cytokine production. Central inflammatory mediators examined in para-athlete literature include interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), interleukin-10 (IL-10), and C-reactive protein (CRP). IL-6 is notable for its dual role as both a pro-inflammatory cytokine and a myokine released from contracting skeletal muscle, influencing glucose metabolism, lipid oxidation, and the balance of other cytokines (Peake, Neubauer, Walsh, & Simpson, 2017). TNF- α and CRP serve as systemic markers of basal or chronic inflammatory tone, whereas IL-10 represents anti-inflammatory signaling that mediates post-exercise resolution. NK cell cytotoxic activity and leukocyte trafficking patterns, particularly lymphopenia followed by rebound proliferation, provide functional insight into immune competence and recovery dynamics in para-athletes, with evidence suggesting both similarities and differences relative to able-bodied cohorts depending on impairment type and severity (Sellami, Puce, & Bragazzi, 2023; Valido et al., 2023). Para-athlete populations display marked heterogeneity that directly shapes immune and inflammatory responses to exercise. Autonomic dysfunction is a critical modifier in individuals with high-level spinal cord injury (SCI), where sympathetic nervous system impairment reduces catecholamine-mediated

leukocyte mobilization, blunts cortisol responses, and disrupts thermoregulatory mechanisms, collectively altering the magnitude and timing of immune activation (Agulló-Ortuño, Ruiz-Cordero, & Navarro-Martínez, 2024; Sterner & Sterner, 2023). Muscle mass and contractile activity represent another key determinant; wheelchair athletes or those with lower-limb amputations have reduced active muscle mass, which attenuates myokine release, particularly IL-6, thereby modulating downstream systemic anti-inflammatory signaling and metabolic effects (Derman, Badenhorst, & Blauwet, 2021; Sellami et al., 2023). Vascular shear and endothelial function are also affected by impairment-specific movement patterns. Reduced limb engagement leads to altered blood flow, lower shear stress, and potentially diminished endothelial cytokine production, which may affect leukocyte margination and local vascular immune signaling. Chronic comorbidities common in para-athlete cohorts—such as pressure ulcers, recurrent urinary tract or respiratory infections, metabolic dysregulation, and persistent low-grade inflammation further interact with exercise to modify immune responses (Valido et al., 2023; Bloom et al., 2020). Finally, emerging evidence suggests that alterations in the gut microbiome and mucosal barrier function, particularly following prolonged immobilization or reduced activity, may influence systemic immune tone, although data in para-athletes remain preliminary (Nieman & Wentz, 2019). Collectively, these factors underscore the necessity of impairment-specific mechanistic frameworks when interpreting exercise-induced immunophysiology in para-athletes, rather than extrapolating from able-bodied models.

Methodological considerations are critical in capturing accurate immune and inflammatory profiles in para-athletes. Timing of sample collection profoundly influences observed cytokine concentrations and cell counts, with distinct patterns emerging immediately post-exercise, within the first hour, and across the 1–24 h recovery period (Peake et al., 2017). Sampling site venous versus capillary also affects measurements, with some studies demonstrating differential cytokine kinetics or cellular profiles depending on site of collection. Assay selection introduces additional variability; ELISA, multiplex immunoassays, flow cytometry, and PCR-based quantification of cytokine gene expression may yield different sensitivity and specificity, which is particularly important given the generally small sample sizes typical in para-athlete research. Inter-individual heterogeneity within impairment subgroups further complicates interpretation, necessitating detailed reporting of lesion level, residual muscle mass, and functional classification to contextualize immune responses. When possible, longitudinal or repeated-measures designs, alongside individualized baselines, improve reliability and reduce confounding due to inter-subject variability. Finally, small cohort sizes and the rarity of some impairment classes often preclude conventional statistical power calculations, reinforcing the importance of cautious interpretation and transparent reporting of confidence intervals, effect sizes, and methodological limitations (Sellami et al., 2023; Sterner & Sterner, 2023).

By integrating these physiological and methodological considerations, researchers and practitioners can more accurately characterize the complex interactions between exercise, inflammation, and immune function in para-athletes. Understanding the interplay of innate and adaptive immune responses, impairment-specific modulators, and measurement constraints is essential not only for interpreting empirical findings but also for designing evidence-informed training programs, monitoring athlete health, and mitigating risk of maladaptive inflammatory or immunological outcomes. This foundation provides a mechanistic backdrop for the subsequent sections of this review, which examine empirical data on para-

athlete-specific exercise-induced inflammation, adaptive responses, and practical implications for performance optimization, recovery strategies, and clinical management. To ensure methodological transparency and reproducibility in para-athlete exercise-immunity research, studies should report participant characteristics, impairment-specific details, training context, sampling protocols, assay methods, and relevant confounders. A summary of recommended minimum reporting standards is provided in Table 1.

Table 1. Minimum reporting standards for exercise-immunity studies in para-athletes

Domain	Specific items
Participant characteristics	Age, sex, body composition (lean mass/fat mass), specific impairment type (e.g., spinal cord injury, amputation, cerebral palsy, visual impairment), lesion level/completeness for SCI, residual limb characteristics for amputees, functional classification (sport-specific class, mobility level)
Medication and supplement use	Immunomodulators, corticosteroids, antibiotics, anti-inflammatory drugs, nutritional supplements affecting immune/inflammatory status
Training and performance context	Training volume, intensity, modality, competitive level, recent exercise history (acute vs. chronic adaptations)
Sampling protocols	Timing relative to exercise (pre, immediate post, 1 h, 24 h, recovery), site of sampling (venous, capillary, tissue biopsy if applicable), repeated measures, longitudinal design details
Assay and measurement methods	Cytokine quantification (ELISA, multiplex, PCR), immune cell phenotyping (flow cytometry, functional assays), standardization/calibration details
Reporting of confounders	Sleep, nutrition, comorbidities, hydration, environmental conditions (temperature, altitude)

Empirical evidence: exercise-induced inflammatory and immune responses across para-athletes

Spinal Cord Injury (SCI) Para-Athletes. Para-athletes with spinal cord injury (SCI) represent one of the most extensively studied groups within adaptive sports immunophysiology, yet findings remain complex due to the degree of autonomic disruption and the heterogeneity of lesions. Baseline immune function in individuals with high-level SCI often reflects a pattern of immune dysregulation characterized by both suppression and low-grade inflammation. Elevated levels of pro-inflammatory cytokines such as TNF- α , IL-1 β , and IL-6 have been observed at rest, likely reflecting chronic tissue stress, metabolic dysregulation,

and recurrent infections (Sarro et al., 2017; Nash, 2022). Conversely, evidence suggests attenuated natural killer (NK) cell activity and impaired leukocyte trafficking, particularly in tetraplegic athletes, due to diminished sympathetic drive and reduced catecholamine-mediated mobilization (Leicht et al., 2021). Acute exercise responses in SCI para-athletes demonstrate distinct cytokine kinetics compared to able-bodied athletes. Studies using arm-crank ergometry or wheelchair racing indicate blunted IL-6 and IL-10 elevations post-exercise, alongside lower leukocyte and neutrophil counts (Paulson et al., 2015; Uth et al., 2018). These attenuated responses may result from reduced active muscle mass and limited myokine release, given the diminished skeletal muscle recruitment below the lesion level. Nonetheless, repeated exercise exposure can partially restore immune responsiveness. For example, 12-week endurance training in paraplegic athletes increased circulating anti-inflammatory IL-10 and decreased CRP concentrations, suggesting that chronic training mediates favorable immunomodulation despite baseline dysregulation (Haapanen et al., 2018; Leicht et al., 2021). A key mechanistic feature influencing these responses is autonomic interruption. High thoracic or cervical SCI disrupts sympathetic pathways, reducing adrenal catecholamine release and impairing thermoregulation, cortisol rhythms, and vascular tone (Jacobs & Nash, 2019). These autonomic deficits not only blunt acute immune activation but also increase vulnerability to respiratory and urinary tract infections, particularly during intensive training or competition phases (Totosy de Zepetnek et al., 2015). Overall, exercise in SCI para-athletes exerts dual effects acting as an anti-inflammatory stimulus when applied chronically, yet constrained by physiological limitations related to autonomic and muscular deficits. Optimizing training load, ensuring thermoregulatory control, and supporting recovery may therefore enhance immune resilience and reduce infection susceptibility in this population. The interplay of lesion level, training intensity, and individual immune variability remains a critical research frontier in para-sport physiology.

Limb amputation & unilateral/bilateral limb deficiency para-athletes. Amputee para-athletes (lower- and upper-limb loss) comprise a growing and heterogeneous subgroup within para-sport whose inflammatory and immune responses to exercise remain comparatively understudied. Available empirical work largely addresses functional capacity, cardiometabolic risk and broad hematological/biochemical markers rather than detailed cytokine kinetics, but several consistent observations and practical considerations emerge. First, amputee athletes commonly present with reduced fat-free mass and altered body composition, which influences resting metabolic rate and systemic inflammatory tone; adiposity and reduced lean mass are both linked to higher circulating CRP and IL-6 in broader cohorts, implying potential baseline differences that are relevant for training prescription in amputee athletes (Nowak, Marszałek, & Molik, 2022; Kurtoğlu et al., 2024). Second, studies of structured training in amputee populations show favorable shifts in cardiovascular fitness and hematological indices and occasional reductions in inflammatory surrogates after short-term programs, suggesting that chronic training can be immunomodulatory and cardioprotective in this group (Grecco et al., 2023; Kurtoğlu et al., 2024). Mechanistic drivers specific to the amputee context include altered muscle mass distribution and asymmetric loading, which modulate myokine release (e.g., IL-6) during locomotor activities and may reduce the systemic anti-inflammatory stimulus generated by contracting muscle mass compared with whole-body engagement. Prosthesis-related factors residual-limb skin health, socket fit, micromotion and localized mechanical stress create a milieu prone to local

inflammatory responses, callus formation or dermatitis, and recurrent tissue irritation; although these localized processes do not always translate to systemic cytokine spikes, they represent clinically important sources of inflammatory burden and infection risk (Tinney, Caldwell, & Lamberg, 2024). Match- and sport-specific analyses (e.g., amputee football running profiles) reveal that intermittent high-intensity efforts and sport demands produce cardiovascular and metabolic loads comparable to able-bodied analogues in many players, implying that exercise-induced biomarker dynamics (acute IL-6 rises, post-match CRP increases) are plausible but under-measured in this population (Muracki et al., 2023; Nowak et al., 2022). Data gaps and inconsistencies are notable: few studies report serial cytokine measures (pre, immediate post, 1 h, 24 h), and sample sizes are often small or limited to single sports. Consequently, direct evidence on acute cytokine kinetics and NK/lymphocyte functional shifts in amputee athletes remains scarce. Practically, clinicians and coaches should prioritize (1) preserving residual-limb health and prosthetic fit to minimize chronic local inflammation, (2) preserving lean mass through targeted strength and nutrition strategies to maximize beneficial myokine signaling, and (3) implementing longitudinal biomarker surveillance (e.g., hs-CRP, IL-6 where feasible) in athletes undergoing intensified training. Targeted, impairment-specific cytokine and immune functional studies stratified by level of amputation and sport are an urgent priority to move from inference to evidence-based recommendations for this expanding para-athlete group (Sellami, Puce, & Bragazzi, 2023; Kurtoğlu et al., 2024).

Cerebral Palsy and Neuromotor Impairments in Para-Athletes. Cerebral palsy (CP) represents a heterogeneous group of permanent movement disorders resulting from early brain injury, leading to varying degrees of motor dysfunction, spasticity, and coordination deficits (Rosenbaum et al., 2017). These neuromotor limitations significantly influence exercise tolerance, metabolic responses, and immune regulation in para-athletes with CP (Nooijen et al., 2016). While participation in sport offers functional and psychosocial benefits, underlying neurophysiological constraints and altered muscle activation patterns may modify inflammatory and immune responses to exercise compared with able-bodied athletes (Van der Slot et al., 2020). Studies investigating acute and chronic exercise effects in CP athletes remain limited, but emerging evidence indicates altered cytokine dynamics and systemic inflammation markers. For instance, Totosy de Zepetnek et al. (2018) observed blunted IL-6 and TNF- α responses following submaximal exercise in adolescents with CP, suggesting a dampened inflammatory reactivity potentially linked to reduced active muscle mass and impaired sympathetic activation. Similarly, Runciman et al. (2019) reported elevated baseline C-reactive protein (CRP) and IL-1 β levels in adults with spastic CP, indicative of low-grade systemic inflammation that might be partially mediated by sedentary behavior, muscle atrophy, and metabolic dysregulation. Regular training interventions, however, appear to mitigate these effects: a 12-week combined aerobic-resistance program reduced CRP and improved NK cell cytotoxicity in CP athletes (Kubo et al., 2021), underscoring the anti-inflammatory potential of structured physical activity. Mechanistically, autonomic imbalance and increased oxidative stress are key modulators of immune responses in CP. Impaired parasympathetic activity and chronic sympathetic overdrive contribute to pro-inflammatory signaling and endothelial dysfunction (Gould et al., 2018). Furthermore, muscle contractile

inefficiency and fiber-type alterations reduce myokine release especially IL-6 and irisin which normally mediate exercise-induced immune regulation (Pedersen & Febbraio, 2017). These physiological differences complicate the extrapolation of data from able-bodied cohorts to para-athletes with CP. From a practical standpoint, individualized training prescription that accounts for spasticity severity, motor control, and fatigue thresholds is critical. Periodized low-to-moderate intensity exercise with adequate recovery can optimize anti-inflammatory benefits while minimizing neuromuscular strain. Nevertheless, more mechanistic trials are warranted to elucidate dose–response relationships and to define optimal exercise modalities for enhancing immune resilience in this population (Keilani et al., 2020).

Visual Impairment, Intellectual Impairment, and Other Para-Sport Classes. Para-athlete populations with visual impairment (VI), intellectual impairment (II), or other less-studied classifications represent the most limited evidence base in exercise-immunity research. Unlike spinal cord injury or amputation, these groups rarely exhibit intrinsic physiological barriers to immune function; however, altered physical activity patterns, training adaptations, and comorbid conditions may influence inflammatory and immune responses (Fagher & Lexell, 2019). In visually impaired athletes, available studies suggest normal baseline immune profiles but demonstrate typical exercise-induced cytokine responses, including transient increases in IL-6, TNF- α , and IL-10 following aerobic or resistance sessions (Van der Linden et al., 2020). Training adaptations appear similar to able-bodied counterparts, although some evidence indicates slightly elevated oxidative stress markers, potentially reflecting altered gait mechanics, compensatory movement strategies, or increased energetic cost during locomotion (Gauthier et al., 2021). Athletes with intellectual impairment (e.g., para-athletes classified under the International Federation for Intellectual Disability Sport) show limited mechanistic data. Small-scale investigations report modest acute increases in pro-inflammatory markers post-exercise and improvements in anti-inflammatory profiles with structured multi-week programs (Rodriguez et al., 2022). Cognitive and behavioral factors such as exercise engagement, motivation, and adherence likely moderate physiological outcomes, emphasizing the need for tailored coaching and program design (Fagher & Lexell, 2019). Other para-sport classes, including athletes with multiple impairments or rare congenital disorders, remain largely unstudied. Existing evidence underscores knowledge gaps, particularly regarding serial cytokine profiling, NK cell function, and adaptive immune dynamics. Consequently, any translational application or performance guidance must be cautious and individualized, integrating medical, functional, and psychosocial considerations. Taken together, while VI and II para-athletes may not experience intrinsic autonomic or muscular limitations as seen in SCI or amputee populations, empirical data remain sparse, and mechanistic insights are inferred largely from able-bodied exercise immunology. High-quality, longitudinal studies with repeated biomarker measurements are urgently needed to establish evidence-based training and health recommendations in these understudied para-athlete populations

Exercise modality, intensity, volume, and contexts that modify immune responses in para-athletes

The relationship between exercise modality and immune regulation in para-athletes is complex, shaped by the interplay between impairment type, residual motor function, and physiological stress responses. Aerobic,

resistance, and high-intensity interval training (HIIT) paradigms each induce distinct inflammatory and immunomodulatory cascades, with implications for optimizing training and health outcomes in this unique athletic population.

Aerobic exercise remains the cornerstone of conditioning in most para-sport disciplines, yet the immune response to endurance-based exercise differs substantially depending on locomotor mode and lesion level. Wheelchair propulsion, the predominant aerobic modality among spinal cord-injured (SCI) athletes, elicits smaller active muscle mass recruitment and altered sympathetic drive, resulting in attenuated but prolonged cytokine responses compared with leg cycling (Paulson et al., 2015; Bartlett et al., 2021). IL-6 and IL-10 typically rise in proportion to exercise duration and relative intensity, reflecting both metabolic stress and anti-inflammatory counter-regulation (Pedersen & Febbraio, 2017). However, SCI athletes with higher lesions ($\geq T6$) display blunted catecholamine responses, potentially limiting leukocyte mobilization and delaying recovery (West et al., 2019). Training status also modulates immune resilience; well-trained wheelchair racers exhibit reduced baseline C-reactive protein (CRP) and a more rapid post-exercise normalization of IL-6 and TNF- α (Pinto et al., 2023). Conversely, sedentary individuals with comparable impairments often exhibit chronic low-grade inflammation and exaggerated cytokine reactivity to even moderate workloads (Gomes et al., 2020). The metabolic efficiency achieved through regular aerobic training therefore plays a crucial protective role against persistent inflammatory activation in para-athletes. Resistance exercise in para-athletes provokes more localized inflammatory and myokine responses relative to systemic alterations observed during endurance activity. Muscle contraction induces the release of myokines such as IL-6, IL-15, and irisin, which mediate communication between active muscle and immune organs (Pedersen, 2019). For athletes with SCI or amputation, reduced active muscle volume limits total cytokine output, yet repeated resistance sessions may still produce meaningful systemic anti-inflammatory adaptations (Bartlett et al., 2021). In wheelchair rugby or powerlifting athletes, acute bouts often yield transient increases in TNF- α and CRP due to localized muscle microdamage (Carty et al., 2020). Importantly, the adaptive remodeling that follows resistance training is associated with elevated IL-10 and diminished CRP, suggesting an evolving immune tolerance to muscular stress (Gillett et al., 2022). These findings indicate that properly periodized resistance training schedules allowing for adequate recovery between sessions may enhance immune robustness while avoiding chronic fatigue and overtraining risk. The interaction between load intensity and contraction type (isometric vs. dynamic) is another determinant of cytokine amplitude, particularly in athletes with impaired thermoregulation or limited autonomic feedback (Jacobs et al., 2018).

High-intensity interval training (HIIT) and repeated-sprint paradigms, though logistically challenging for many para-athlete subgroups, have recently gained attention for their potential efficiency and immune benefits. Short, intermittent bouts of maximal effort can evoke large but transient surges in pro-inflammatory cytokines (IL-6, IL-1 β , TNF- α), followed by compensatory anti-inflammatory signaling (IL-10, IL-1ra) during recovery (Tucker et al., 2017; Derman et al., 2020). In trained SCI athletes, 6–8 weeks of HIIT improved VO₂peak and reduced baseline CRP without compromising immune cell counts (Wouda et al., 2021). However, the same protocols in athletes with cerebral palsy or limb deficiency may impose

excessive oxidative stress if not matched to individual capacity (Fagher et al., 2023). Feasibility studies emphasize that adaptive equipment (e.g., arm ergometers with adjustable resistance) and careful workload monitoring are essential to avoid immune suppression. Importantly, sufficient recovery intervals between sessions typically ≥ 48 hours are required to restore leukocyte homeostasis and antioxidant reserves, especially in athletes with limited mobility or chronic comorbidities.

Beyond training modality and intensity, environmental and psychological contexts during Paralympic competition profoundly affect immune competence. Multi-day tournaments, travel across time zones, and classification-related stressors can synergistically promote transient immunosuppression (Pyne et al., 2016). Elevated cortisol levels, disrupted sleep, and exposure to novel pathogens heighten susceptibility to upper respiratory infections (URIs) among Paralympians, mirroring trends observed in able-bodied elite athletes but with amplified consequences due to preexisting immune dysregulation (Fagher et al., 2023). Evidence from the Tokyo 2020 and Beijing 2022 Paralympic Games indicates that illness incidence was notably higher among SCI and visually impaired athletes, particularly during travel and early competition days (Derman et al., 2022). Nutritional support (e.g., vitamin D, omega-3 fatty acids), psychological resilience programs, and pre-travel conditioning may mitigate these risks. Thus, immune health monitoring should be an integral part of para-athlete preparation and recovery cycles.

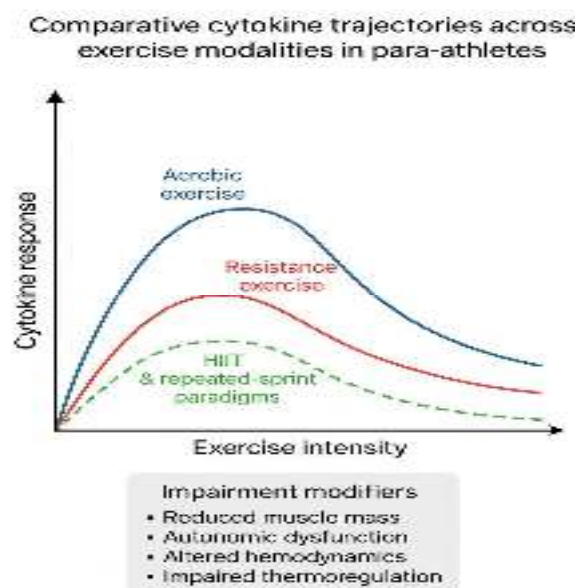


Figure 1. Comparative schematic of expected acute cytokine trajectories for different exercise modalities in para-athletes (qualitative curves), annotated with impairment modifiers.

As illustrated in Figure 3, different exercise modalities elicit distinct inflammatory and immune response trajectories in para-athletes. Aerobic exercise tends to induce a moderate, transient increase in cytokines such as IL-6 and IL-10, whereas resistance training produces more localized inflammatory signaling related to muscle repair and adaptation. In contrast, HIIT protocols generate sharp but short-lived spikes in systemic

cytokines, particularly in athletes with greater autonomic dysfunction or reduced muscle mass, emphasizing the importance of individualized recovery strategies

Schematic representation of qualitative cytokine kinetics (e.g., IL-6, TNF- α , IL-10) following different exercise modalities aerobic, resistance, and high-intensity interval training (HIIT) in para-athletes. The figure highlights how impairment-specific modifiers such as autonomic dysfunction, reduced active muscle mass, and altered vascular shear influence the magnitude and recovery pattern of inflammatory and immune responses. Curves are illustrative and not drawn to scale; they summarize typical acute response profiles observed in experimental studies among para-athlete cohorts.

Adaptive vs. maladaptive inflammation: training, recovery, and performance implications

Adaptive inflammatory signaling after exercise promotes repair, remodeling and functional gain, whereas maladaptive or chronic inflammation undermines recovery, predisposes to illness, and impairs performance capacity. Adaptive signaling is typically characterized by an acute, time-limited rise in pro-inflammatory mediators (for example, a transient increase in interleukin-6 [IL-6] with concomitant induction of anti-inflammatory cytokines such as IL-10 and interleukin-1 receptor antagonist) accompanied by leukocyte activation that resolves within hours to days and supports tissue repair and metabolic adaptation (Peake, Neubauer, Walsh, & Simpson, 2017). In contrast, maladaptive inflammation manifests as persistently elevated basal markers (e.g., high-sensitivity C-reactive protein [hs-CRP], chronic TNF- α elevation), sustained immune cell dysfunction (reduced natural killer cell cytotoxicity, impaired lymphocyte proliferation), and prolonged catabolic signaling that together impede muscle repair and increase susceptibility to infection and non-healing wounds. In para-athlete populations, the boundary between adaptive and maladaptive inflammation is shifted by impairment-specific biology: autonomic dysfunction after high spinal cord injury, ongoing local tissue irritation in amputees (residual-limb inflammation), or chronic low-grade inflammation associated with neuromotor disorders can convert otherwise beneficial exercise stimuli into disproportionate inflammatory burdens (Valido et al., 2023; Sterner & Sterner, 2023). Consequently, distinguishing transient, adaptive cytokine kinetics from persistent inflammatory tone is essential for clinically meaningful interpretation and safe training prescription in para-sport.

Empirical evidence links immune responses to both short-term performance and long-term health in para-athletes. Short-term, acute immune perturbations (for example, marked post-exercise lymphopenia or exaggerated IL-6 spikes without timely anti-inflammatory counterbalance) can coincide with transient decrements in neuromuscular function, impaired power output, and subjective fatigue during multi-day competitions (Paulson, Bishop, & Goosey-Tolfrey, 2015; Pinto et al., 2023). Over repeated cycles, inadequate recovery or persistent inflammatory activation correlates with greater illness incidence especially respiratory and urinary tract infections in athletes with SCI and with longer recovery times from musculoskeletal microtrauma (Totosy de Zepetnek et al., 2015; Derman et al., 2022). Longitudinal training interventions provide encouraging evidence that chronic, appropriately dosed exercise reduces basal inflammatory markers in several para-athlete groups: endurance or combined training programs lowered CRP and increased anti-inflammatory mediators in paraplegic and amputee athletes, and resistance protocols improved NK cell function in athletes with neuromotor disorders (Haapanen et al., 2018; Gillett

et al., 2022; Moro et al., 2020). Nonetheless, heterogeneity in lesion level, residual muscle mass, prosthetic issues, and comorbidities produces variable responses, such that group-level improvements may mask individual maladaptive trajectories. The clinical implication is clear: immune readouts can both reflect and predict performance capacity and health risk, but they must be interpreted within an impairment-aware framework and longitudinally rather than from isolated timepoints (Sellami, Puce, & Bragazzi, 2023; Valido et al., 2023).

Translating these insights into training and recovery practice requires a conservative, individualized approach. Periodization should explicitly integrate immune and recovery markers into load planning: macrocycles with progressive overload must be punctuated by active recovery or low-intensity blocks, and para-athletes with autonomic dysfunction or limited muscle mass may require longer recovery windows than able-bodied norms. Biomarker monitoring should prioritize feasible, informative measures baseline and serial hs-CRP for chronic inflammatory tone; point-of-care leukocyte counts; salivary immunoglobulin A (sIgA) for mucosal immunity where respiratory infection risk is high; and targeted cytokine panels (IL-6, IL-10) when laboratory resources permit (Peake et al., 2017; Pinto et al., 2023). Subjective measures (training distress scales, sleep quality, perceived recovery) often outperform single objective metrics for early detection of maladaptation and should be systematically collected (Saw, Main, & Gastin, 2016). Wearable proxies heart rate variability (HRV), sleep duration/efficiency, and movement-based training load provide continuous, noninvasive surveillance and are particularly useful when blood sampling is impractical. Decision rules for load modification should combine markers: for example, substantially elevated hs-CRP or sustained reductions in sIgA plus worsening subjective recovery would prompt de-loading and medical review in athletes with SCI or chronic wounds. Nutritional and sleep optimization, infection control (vaccination, hygiene), and prosthetic/residual-limb management are complementary strategies to reduce chronic inflammatory burden and support adaptive responses. Lastly, because cohort sizes are often small and interindividual variability large, multi-site registries and standardized reporting are essential to refine biomarker thresholds and evidence-based decision rules for para-athlete populations (Derman et al., 2021; Sellami et al., 2023). Monitoring immune and inflammatory responses in para-athletes requires a multimodal approach combining biomarkers, subjective assessments, and wearable data. Table 1 summarizes an evidence-based toolbox integrating feasible, impairment-specific monitoring domains for practical implementation in para-sport environments.

Table 1. Evidence-Based Monitoring Toolbox for Para-Athlete Training: Recommended Biomarkers, Monitoring Domains, and Practical Thresholds for Adaptive Load Management

Monitoring Domain	Specific Measure / Marker	Physiological Relevance	Recommended Frequency	Interpretation / Action Thresholds	Practical Considerations (Para-Athletes)
Core Blood Biomarkers	High-sensitivity C-reactive protein (hs-CRP)	Index of systemic low-grade inflammation	Baseline + every 4–6 weeks	>3 mg·L ⁻¹ suggests chronic inflammation or inadequate recovery	Elevated in chronic wounds, pressure ulcers, or infection risk in SCI athletes
	Full blood count (WBC, neutrophil, lymphocyte)	General immune activation and recovery status	Baseline + after heavy blocks	Leukocytosis (>10×10 ⁹ /L) or lymphopenia (<1.0×10 ⁹ /L) → potential immune stress	Adjust for medication use (e.g., antispasmodics, corticosteroids)
Mucosal Immunity	Salivary immunoglobulin A (sIgA)	Protection against upper respiratory infections	2–3× per week (non-invasive)	>30% drop from baseline → high infection risk	Ideal for multi-day competitions; easy home sampling
Cytokine / Myokine Panel (if available)	IL-6, IL-10, TNF-α	Balance between pro- and anti-inflammatory signaling	Selected sampling (training phase studies)	High IL-6 with low IL-10 → maladaptive stress	Interpretation must consider lesion level and muscle mass
Immune Function Tests	NK cell cytotoxicity, lymphocyte proliferation	Cellular immune competence	Research or clinical cases only	Reduced cytotoxicity → impaired defense	Use specialized labs; avoid post-competition sampling

Subjective Recovery Indicators	Daily wellness score (fatigue, sleep, mood, soreness)	Integrated perception of recovery status	Daily	Decline >2 points from baseline → monitor closely	Highly predictive of overtraining; requires athlete compliance
	Sleep quality and duration	Recovery and immune modulation	Daily (wearables or logs)	<6 h sleep for >3 nights → immune suppression risk	Adapt for travel and Paralympic events
Wearable Metrics / Physiological Proxies	Heart rate variability (HRV, RMSSD)	Autonomic recovery, parasympathetic tone	Daily or 3× weekly	>10% drop from baseline HRV → sympathetic dominance	Consider autonomic dysreflexia in SCI athletes
	Training load (session-RPE × duration)	External/internal load balance	Every session	Monotony index >2.0 or strain >6000 → high overload	Use to guide periodization in limited recovery windows
Contextual / Clinical Observations	Residual-limb skin condition, pressure ulcers	Chronic inflammatory source	Weekly medical review	New lesions → immediate load modification	Multidisciplinary coordination essential
	Infection frequency (URTI, UTI)	Immune system burden	Track per month/season	>2 infections/season → investigate overtraining	Vaccination and hygiene support critical

Therapeutic and nutritional interventions to modulate inflammation and immunity in para-athletes

Exercise-induced immune modulation is not solely a matter of training load and recovery: nutritional, pharmacological and adjunctive therapeutic strategies form a critical layer in shaping adaptive inflammatory signaling and supporting immune resilience in para-athletes. Nutritional strategies represent the most accessible avenue. For example, long-chain omega-3 polyunsaturated fatty acids (PUFAs, EPA/DHA) have been shown to reduce pro-inflammatory cytokines and support leukocyte function in

athletic populations, although direct trials in para-athletes remain rare (Thielecke & Nestel, 2020). Nonetheless, review data indicate that athletes with physical impairments (e.g., spinal cord injury) often have sub-optimal omega-3 status and may benefit from supplementation of $\sim 1\text{--}3\text{ g day}^{-1}$ EPA+DHA, with caution regarding bleeding risk in those with pressure-ulcer histories or antiplatelet therapy (Ritz et al., 2020; Shaw et al., 2021). Vitamin D deficiency is highly prevalent among para-athletes (especially wheelchair users and amputees) due to limited sun exposure and altered metabolic status; observational data link serum 25-hydroxyvitamin D $< 50\text{ nmol L}^{-1}$ with higher infection incidence and elevated systemic CRP, prompting many practitioners to aim for levels $> 75\text{ nmol L}^{-1}$ in this group (Ghazzawi et al., 2025). Protein intake and timing also merit special attention: in athletes with spinal cord injury, reduced active muscle mass, lower energy expenditure and metabolic derangements demand adjusted protein guidelines (Flueck & Perret, 2021). Although conventional able-bodied athlete targets ($1.6\text{--}2.2\text{ g}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$) may offer a starting point, para-athlete specialists counsel tailoring based on lean mass, residual muscle recruitment and renal status. Antioxidant and polyphenol interventions (e.g., quercetin, cocoa flavanols) show promise in reducing oxidative-stress-mediated inflammation, yet may blunt training adaptation when used chronically; thus, their application in para-athlete contexts requires phase-specific prescription (Stojic et al., 2023).

Pharmacological and medical considerations are equally pivotal. Para-athletes often rely on medications spasticity treatments (e.g., baclofen), corticosteroids, antispasmodics or frequent antibiotic courses for urinary tract or skin infections that may interact with immune and inflammatory readouts. Routine vaccination, notably for influenza and SARS-CoV-2, is indispensable given elevated infection risk in spinal cord injury and neuromotor impairment cohorts (Sellami, Puce, & Bragazzi, 2023). While protective responses are generally intact, autonomic dysregulation may attenuate antibody kinetics or mucosal immunity, necessitating post-vaccination monitoring in some cases (Sellami et al., 2023). Antibiotic-induced gut dysbiosis is an under-recognized factor in immune perturbation; co-administration of probiotics may restore gut-immune homeostasis, though data in highly trained para-athletes remain limited. Non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids can suppress acute inflammatory responses and mask training-induced cytokine changes; thus, in immune monitoring protocols these must be noted as confounders and accounted for when interpreting biomarker kinetics (Valido et al., 2023).

Training-based, rehabilitation and adjunct modalities constitute a third pillar of intervention. Cryotherapy and cold-water immersion have been used post-competition in wheelchair rugby and amputee sprinters to reduce muscle soreness and IL-6 kinetics; however, over-use may blunt the training-dependent myokine signaling required for adaptation (Gomes et al., 2020). Heat therapy and thermoneutral immersion may be particularly relevant for athletes with spinal cord injury who's thermoregulatory and perfusion capacity is compromised; preliminary work indicates enhanced IL-10 release and improved capillary perfusion in paralyzed limbs after repeated exposure (Blackport, 2022). Compression garments, widely used in able-bodied sport for venous return and edema management, show utility for reducing lower-limb swelling in amputee athletes and thus lowering secondary inflammatory burden (Flueck & Perret, 2021). Neuromuscular electrical stimulation (NMES) has emerged as a viable modality in para-athletes for

inducing muscle contraction in de-innervated or under-used limbs; small pilot studies in paraplegic populations show reduced TNF- α , improved muscle oxidative properties and increased lean mass after 8–12 weeks of NMES combined with resistance training (Li et al., 2025). Implementation demands careful oversight to avoid skin irritation, and pulse parameters may need adjustment in athletes with sensory deficits. In sum, therapeutic and nutritional interventions offer significant promise for modulating inflammation and immunity in para-athletes however, they must be individualized, contextualized and integrated with training load, recovery and health status. Table 2 summarizes key interventions, evidence levels specific to para-athlete cohorts, recommended usage guidelines and safety caveats. This integrated approach enhances the capacity to support adaptive immune responses, minimize infection risk, and facilitate performance and health outcomes in para-sport

Table 2. Summary of nutritional, pharmacological, and adjunct interventions to modulate inflammation and immunity in para-athletes (evidence-based, 2015–2024)

Category / Intervention	Evidence in Para-Athletes (2015–2024)	Mechanism / Clinical Rationale	Recommended usage / Practical notes	Safety caveats & limitations	Key references (examples)
Omega-3 (EPA/DHA)	Limited/indirect — few para-athlete studies; Shaw et al. (2021) systematic review identified fish-oil use in small para-athlete samples; direct RCTs in PARA cohorts are sparse.	Anti-inflammatory (\downarrow NF- κ B), modulate eicosanoid balance, support membrane function.	Consider 1–3 g EPA+DHA \cdot day ⁻¹ for 6–12 wk when low dietary intake; integrate with meals.	Bleeding risk (monitor with anticoagulant therapy), heterogenous formulations.	Shaw et al., 2021; Ritz et al., 2020
Vitamin D (25(OH)D correction)	Moderate (observational + trial protocols in SCI) — deficiency common in wheelchair users; VitD-SCI protocol and observational data support correction.	Supports innate immunity, antimicrobial peptides, modulates inflammation.	Screen; replete to target ≥ 75 nmol \cdot L ⁻¹ when deficient; individualized dosing.	Monitor Ca ²⁺ ; evidence for direct performance/immune endpoints in PARA limited.	Hertig-Godeschalk et al., 2021 (protocol); Wang et al., 2023

Protein (intake & timing)	Moderate (PARA-SCI evidence & guidance) — Flueck & Perret (2021) provide PARA-SCI-specific recommendations (≥ 1.2 g·kg ⁻¹ /day minimal; higher for hypertrophy).	Supports muscle repair, myofibrillar synthesis, myokine production relevant for immune cross-talk.	Individualize by lean mass and training; distribute protein across day; leucine-rich post-exercise feeding.	Adjust for renal/medical status; energy balance considerations.	Flueck & Perret, 2021
Antioxidants / Polyphenols (quercetin, cocoa flavanols)	Limited (pilot / translational) — few small para-athlete reports; broader athlete literature indicates benefit for oxidative stress but possible blunting of adaptation if chronic.	Scavenge ROS, modulate Nrf2/NF- κ B pathways, support endothelial health.	Use phase-specifically (e.g., travel, acute heavy load); avoid chronic megadoses.	May blunt training adaptations; product variability.	Stojic et al., 2023; Shaw et al., 2021
Vaccination (influenza, SARS-CoV-2)	Strong (observational/recommendation) — recommended for PARA; evidence supports protective value and reduction in training/competition loss.	Prevents infection-related immune burden and performance loss.	Ensure up-to-date vaccination, schedule away from key competitions when possible.	Local/systemic reactivity; immunogenicity may be modestly altered in some SCI cases — monitor.	Sellami, Puce, & Bragazzi, 2023
Antibiotics \pm Probiotics	Common clinical practice; limited PARA trial data — antibiotics often used for recurrent	Antibiotics alter gut microbiota \rightarrow	Use probiotics during/after antibiotic	Strain/product heterogeneity; limited RCTs	Valido et al., 2023; Shaw et al., 2021

	UTIs in SCI; probiotics proposed to mitigate dysbiosis.	mucosal immunity; probiotics may restore balance.	courses (strain-specific).	in elite PARA athletes.	
NSAIDs / Corticosteroids	Well-documented pharmacologic effects; PARA usage common for spasticity/pain	Strong anti-inflammatory effect; corticosteroids suppress cellular immunity.	Restrict to clinical indications; record in biomarker studies as confounder.	Mask biomarker signals; long-term harms (immune suppression, metabolic).	Valido et al., 2023
Cold therapy / cryotherapy	Limited PARA studies; applied in wheelchair/amputee sports — some field reports and small trials show reduced soreness and short-term IL-6 attenuation.	Reduces local inflammation, edema; analgesic effects.	Short, targeted use post-competition (<10–15 min); avoid routine overuse.	May blunt adaptive signaling if overused; practical/logistic issues.	Gomes et al., 2020 (para-sport contexts); Griggs et al., 2019 (heat/thermoregulation review)
Heat therapy / thermal immersion	Emerging evidence (SCI relevance) — thermotherapy may improve perfusion and anti-inflammatory signaling in paralyzed limbs (preliminary).	Increases peripheral blood flow, can enhance IL-10 release and nutrient delivery.	Consider 1–3×/week; monitor autonomic response in high-level SCI.	Thermoregulatory risks in SCI (autonomic dysfunction).	Griggs et al., 2019; Dorrian et al., 2023

Compression garments	Limited PARA data; mechanistic rationale — reduces edema in residual limbs, may lower local inflammatory stimulus.	Improves venous return, reduces interstitial cytokine accumulation.	Use post-exercise/travel; ensure prosthetic compatibility.	Risk of pressure/skin breakdown in stump areas; fitting critical.	Flueck & Perret, 2021
Neuromuscular electrical stimulation (NMES/FES)	Moderate (SCI clinical and sport studies) — FES cycling and NMES improve aerobic fitness, lean mass and show reductions in inflammatory markers in small studies.	Mimics contractile activity → myokine release, muscle metabolism enhancement.	Combine with resistance training 2–3×/week; 8–12 wks. protocols reported.	Skin irritation, contraindications (implanted stimulators), careful parameterization required.	Dorrian et al., 2023; Research on FES cycling (workshop reports)

Health outcomes, infection risk, and long-term considerations

Paralympic and para-athlete cohorts face a distinctive constellation of health risks in which infection incidence, vaccine responsiveness, chronic inflammation and cardiometabolic comorbidity interact to influence both availability for training and long-term health. Surveillance during major international competitions documents a measurable burden of illness: at the Tokyo 2020 Paralympic Games the overall incidence of illness was 4.2 illnesses per 1,000 athlete-days, with particularly high incidence in wheelchair sports, underscoring the acute vulnerability of some impairment groups to infection during travel and competition cycles (Derman et al., 2022). Infection risk in para-athletes is amplified by impairment-specific factors impaired cough and pulmonary mechanics, neurogenic bladder with recurrent urinary tract infections, and skin breaches so that respiratory and genitourinary illnesses are common causes of time loss from training (Totony de Zepetnek et al., 2015; Derman et al., 2022).

Vaccine responses in people with physical impairments are generally protective but can be modified by underlying immune dysregulation. Evidence from vaccination status studies and targeted assessments in spinal cord injury (SCI) populations indicates that standard immunogenicity is usually achieved for routine vaccines (e.g., influenza, SARS-CoV-2), although autonomic dysfunction, chronic inflammation and medication use (e.g., corticosteroids) can attenuate magnitude or kinetics of responses in some individuals, motivating post-vaccination monitoring in higher-risk athletes (Bigford & Garshick, 2022; Sellami, Puce, & Bragazzi, 2023). Importantly, vaccination prevents infection-related training loss and should be

integrated into athlete medical planning, with timing optimized to minimize interference with competition and training peaks (Derman et al., 2022).

Chronic inflammatory burden is a pervasive concern in many para-athlete subgroups and acts as a nexus between immune function, metabolic health and performance. Individuals with chronic SCI commonly display systemic low-grade inflammation elevated CRP and pro-inflammatory cytokines that contributes to increased cardiometabolic risk, including dyslipidemia, insulin resistance and accelerated atherosclerotic processes; these conditions are reported across cohort studies and reviews and represent major long-term health priorities beyond sport performance (Bigford & Garshick, 2022; Nash, 2018). Training and structured exercise can reduce basal inflammatory markers in several para-athlete groups, yet heterogeneity in lesion level, body composition and comorbidities means benefits are variable and individualized monitoring is essential (Haapanen et al., 2018; Valido et al., 2023).

Skin integrity and pressure injuries uniquely mediate inflammatory load in those with paralysis or sensory loss. Pressure injuries are both a source and consequence of chronic inflammation: local tissue breakdown fosters persistent cytokine production and bacterial colonization, which in turn promote systemic inflammatory signaling and impair wound healing (Vecin & Gater, 2022). For para-athletes this interaction has immediate performance implications wounds limit training, increase infection risk and may necessitate antibiotic courses that perturb the gut-immune axis and long-term consequences if recurrent lesions sustain pro-inflammatory milieu that exacerbate cardiometabolic risk (Vecin & Gater, 2022; Valido et al., 2023). Multidisciplinary prevention (pressure-relieving seating, frequent repositioning, prosthetic socket optimization) and rapid treatment of skin breaches are therefore central to minimizing immune burden in para-sport cohorts.

In summary, the health landscape for para-athletes is defined by an intersection of elevated infection risk in specific impairment classes, largely preserved but sometimes modified vaccine responsiveness, and a pervasive tendency to chronic low-grade inflammation that links to cardiometabolic disease and to complications of skin integrity. These realities mandate tailored medical planning proactive vaccination, targeted infection surveillance around travel and competition, longitudinal inflammatory and metabolic monitoring, and robust skin-care protocols to protect both short-term performance and long-term health in para-sport athletes.

Knowledge gaps, methodological challenges, and research priorities

The research landscape on exercise-induced inflammation and immunity in para-athletes exhibits several critical gaps and methodological challenges that limit current knowledge and practical application. The predominant issue is small sample size: many studies are single-site investigations or pilot trials involving fewer than thirty participants, which reduces statistical power and limits subgroup analyses by impairment type, lesion level, sex, or competitive status. Heterogeneous cohorts exacerbate interpretive difficulties because studies often combine disparate impairment groups (e.g., spinal cord injury, amputation, cerebral palsy) without disaggregated reporting, masking impairment-specific physiology. Inconsistent reporting of essential descriptors such as lesion level and completeness in SCI, residual-limb characteristics in amputees, functional classification, medication use, and exact training load metrics further undermines comparability. Longitudinal data are rare: most investigations emphasize acute responses to single exercise bouts or short

interventions, whereas clinically meaningful questions require seasonal and multi-year surveillance to detect patterns of chronic inflammation, infection susceptibility, and training adaptation. Women para-athletes are under-represented, reflecting broader gender imbalances in sport science; this absence hinders understanding of sex-specific immune trajectories and the interaction of hormonal status with training-induced inflammation. Additionally, inconsistent laboratory methods (varying assay platforms, sampling times, and biospecimen types) and inadequate accounting for confounders (sleep, nutrition, recent infections, and medications) lead to conflicting results and impede meta-analytic efforts.

To address these limitations, the field should prioritize concrete, scalable study designs. Large multicenter prospective cohorts established through consortia of rehabilitation and sport science centers would enable adequately powered subgroup analyses and harmonized protocols. Such consortia should implement standardized participant phenotyping, including validated measures of body composition, residual muscle mass, autonomic function testing where relevant, and precise impairment classification. Nested within cohorts, mechanistic randomized controlled trials targeting specific impairment strata (for example, HIIT versus moderate-intensity training in paraplegic athletes or NMES combined with resistance training in complete SCI) would elucidate causal pathways while maintaining external validity. Adaptive trial designs and crossover methods can maximize power in small populations, and platform trial infrastructures could efficiently test multiple interventions (nutritional, pharmacological, rehabilitative) against shared control arms. Translational animal models remain useful for mechanistic exploration but should be selected judiciously and designed to mirror key features of human impairments; findings must be validated in human translational studies prior to clinical application.

A unified standard for reporting is necessary. Minimum datasets should mandate clear participant descriptors (age, sex, impairment type and classification, lesion level/completeness, prosthetic status), training history and contemporaneous load metrics (session-RPE, duration), medication and supplement use, comorbidity profiles, and environmental exposures. Core biological outcomes should include readily accessible measures full blood count, hs-CRP, and salivary IgA for mucosal immunity augmented by targeted cytokine panels (IL-6, IL-10, TNF- α) and functional assays (NK cell cytotoxicity) when resources permit. Standardized biospecimen timing (pre-exercise, immediate post, 1 h, and 24 h) and assay platform documentation are essential for comparability. Finally, investment in data sharing, open-access registries, and pre-registration of study protocols will accelerate cumulative knowledge building and enable robust meta-analyses that can inform evidence-based guidelines for training and health management in para-athletes. Collectively, these steps will strengthen translational impact for para-sport. Globally.

Conclusions and clinical/practical implications

The collective evidence reviewed throughout this article underscores that para-athletes exhibit unique inflammatory and immune dynamics shaped by their underlying impairments, training demands, and adaptive physiological responses. While exercise remains a potent modulator of immune health, its effects in this population are neither uniform nor directly comparable to those observed in able-bodied athletes. Spinal cord injury, limb deficiency, cerebral palsy, and other forms of impairment introduce distinct autonomic, vascular, and metabolic alterations that fundamentally influence the inflammatory milieu and

recovery processes. Thus, uncritical extrapolation from general athletic populations risks both scientific misinterpretation and clinical misapplication. For researchers, the central message is the urgent need for rigorously designed, adequately powered, and impairment-specific investigations that incorporate standardized biomarkers, transparent reporting, and longitudinal follow-up. Future studies must integrate multidimensional assessments linking molecular, functional, and behavioral outcomes to capture the true complexity of inflammation and adaptation in para-sport. Collaboration across institutions and disciplines will be essential to overcome sample size limitations and ensure diversity across impairment classes and sex. For clinicians, careful interpretation of immune and inflammatory markers is paramount, considering baseline physiological differences and common comorbidities in para-athletes. Medical management should prioritize individualized recovery monitoring, timely vaccination, and proactive strategies to minimize infection risk and chronic low-grade inflammation. Nutritional and rehabilitative interventions including optimized omega-3 intake, vitamin D sufficiency, controlled protein timing, and adjunct modalities such as neuromuscular stimulation can serve as valuable components of an integrative approach. For coaches and performance staff, training programs should balance overload with adequate recovery, using objective and subjective monitoring tools to detect early signs of maladaptation. Load adjustments, cross-training, and scheduled rest periods should be personalized, recognizing the variable inflammatory responses between and within impairment categories.

Finally, for policy makers and governing bodies, the advancement of para-sport medicine demands institutional support for research infrastructure, accessible biomarker testing, and evidence-based health monitoring protocols. In sum, the goal is not merely to mirror able-bodied sport science but to build a distinct, evidence-driven framework that safeguards health while optimizing performance in the para-athlete community.

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