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Photobiomodulation (Red and Near-Infrared Light Therapy) for Mitochondrial Stimulation and Biological Recovery in Athletes: A Narrative Review

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Abstract

Background. Photobiomodulation (PBM) — the therapeutic use of red and near-infrared light — is promoted in sport as a non-pharmacological, non-thermal aid to performance and recovery, acting primarily through mitochondrial stimulation, yet its clinical value remains debated.

Aim. To synthesise, critically and comparatively, the medical evidence on the mechanisms of PBM and on its ergogenic and recovery effects in athletes, and to define the conditions under which benefit is and is not observed.

Material and methods. A narrative review of literature (2016–2026) was conducted using PubMed/MEDLINE, Web of Science, Scopus, and Google Scholar. Thirty-five papers were included, focusing on network meta-analyses and evidence-based medicine (EBM) clinical guidelines.

Results. Mechanistic and cellular work consistently shows that red/near-infrared light is absorbed by cytochrome c oxidase, increasing mitochondrial membrane potential, ATP and a controlled burst of reactive oxygen species, with downstream nitric-oxide, calcium and transcription-factor signalling that promotes myoblast differentiation, mitochondrial biogenesis and anti-inflammatory effects, all governed by a biphasic (Arndt-Schulz) dose response. In athletes, pre-exercise PBM has improved peak oxygen uptake, time to exhaustion and strength in several trials, and combined PBM–magnetic-field protocols enhanced recovery; yet numerous high-quality trials found no benefit, with effects strongly dependent on dose, wavelength, timing, irradiated muscle, training

status and sex. Meta-analyses suggest modest reductions in delayed-onset muscle soreness and oxidative damage but inconsistent functional gains, and no superiority over established recovery methods.

Conclusions. PBM has a coherent mitochondrial mechanism and a plausible, partly demonstrated ergogenic and recovery role, but clinical efficacy is inconsistent and parameter-dependent; standardised dosimetry and rigorous trials are required.

Keywords: photobiomodulation; low-level laser therapy; mitochondria; muscle recovery; athletic performance; oxidative stress

1. Introduction

Photobiomodulation (PBM), historically termed low-level laser therapy (LLLT), is the application of red and near-infrared light — typically in the 600–1100 nm range, delivered by lasers, light-emitting diodes (LEDs) or broadband sources at intensities that produce photochemical rather than thermal effects — to stimulate cellular function, relieve pain and reduce inflammation [1] [2]. Discovered serendipitously by Endre Mester in the late 1960s, when a ruby laser was found to promote hair growth and wound healing in mice, the technique has expanded from dermatology and dentistry into neurology, internal medicine and, prominently, sports and exercise medicine, where it is now marketed as a versatile, non-invasive and medication-free aid to athletic preparation and recovery [1] [3] [4]. The appeal to athletes is considerable: PBM is painless, carries few apparent risks, and — because its intensity is similar to sunlight and leaves no forensic trace — cannot be banned by anti-doping authorities, so it occupies an attractive position as a legitimate ergogenic strategy [1].

The scientific rationale for PBM in sport rests on its action on the mitochondrion, the organelle central to the energy demands of exercise and to the fatigue, muscle damage and oxidative stress that follow it. The dominant mechanistic account holds that red and near-infrared photons are absorbed by cytochrome c oxidase (CcO), the terminal enzyme of the mitochondrial electron transport chain, displacing inhibitory nitric oxide, accelerating electron transport and oxygen consumption, and increasing the synthesis of adenosine triphosphate (ATP) together with a transient, signalling burst of reactive oxygen species (ROS) [5] [2]. These primary photochemical events trigger secondary cascades — modulation of calcium, nitric oxide and cyclic adenosine monophosphate, and activation of redox-sensitive transcription factors — that culminate in improved cell survival, proliferation, migration and protein synthesis [1] [2]. In the context of exercise, this bioenergetic stimulation is hypothesised to "pre-load" muscle with ATP before effort, to accelerate the repair of exercise-induced damage afterwards, and to temper the inflammatory and oxidative consequences of strenuous training [6] [7].

Yet the translation of this elegant mechanism into reproducible clinical benefit has proven contentious. Although a substantial body of randomised trials and meta-analyses reports improvements in performance, recovery and biochemical markers when PBM is applied before or after exercise, an almost equally substantial body reports no effect

whatsoever, and the discrepancies are widely attributed to the bewildering number of parameters — wavelength, energy and power density, total dose, pulse structure, spot size, treatment timing and the tissue irradiated — that govern the outcome, compounded by differences in participants' sex, training status and the muscle examined [8] [9] [10]. A defining feature of PBM, the biphasic or Arndt-Schulz dose response, means that too little light is ineffective and too much is inhibitory, so that the same device can help or hinder depending on the dose delivered to the target [1] [8]. This dose dependence, and the lack of standardised protocols, lie at the heart of the field's inconsistency.

The aim of this narrative review is to bring together the medical evidence on PBM in athletes in a deliberately comparative and critical manner: to trace the mechanism from the absorption of a photon by cytochrome c oxidase, through cellular and tissue effects on muscle, to the clinical questions of whether — and under what conditions — PBM improves performance and accelerates recovery. The review confronts the supportive and the null trials directly, examines the meta-analytic synthesis, scrutinises the dosimetric and methodological reasons for divergent results, and situates sports applications within the wider clinical and safety context of the modality. Throughout, the mitochondrion is treated as the conceptual link between the biophysics of light and the physiology of the exercising athlete.

2. Material and Methods

This work is a narrative review conducted in the tradition of the SANRA (Scale for the Assessment of Narrative Review Articles) framework rather than a systematic review or meta-analysis. The 35 sources considered were selected to represent the principal medical domains in which PBM for mitochondrial stimulation and athletic recovery has been studied: the biophysics and mitochondrial mechanism of light absorption, the cellular and tissue effects on skeletal muscle and stem cells, the ergogenic (performance-enhancing) effects of pre-exercise application, the recovery effects on muscle damage, soreness, inflammation and oxidative stress, the question of delivery parameters and dosimetry, and the broader clinical, rehabilitative and safety context.

The evidence base is deliberately heterogeneous in design, reflecting the state of the field. It includes mechanistic and biophysical studies of cytochrome c oxidase up-regulation and tissue oxygenation [5] [11], narrative and mechanistic reviews of PBM signalling and its anti-inflammatory effects [1] [2], an appraisal of light parameters and dose-response complexity [8], single-cell and population studies of mitochondrial membrane potential, ROS and bioenergetics in stem cells and myoblasts [12] [13], in-vitro studies of myoblast and satellite-cell differentiation and of oxidatively stressed muscle cells [14] [15], animal studies of the neuromuscular junction and of muscle metabolism [16] [17], and a study of stem-cell proliferation via TRPV1 signalling [18]. The applied evidence comprises randomised controlled trials and a training protocol in athletes and active individuals examining performance and recovery [6] [19] [20] [21] [22] [23] [24], trials reporting null results [10] [9] [25] [26] [27], systematic reviews

with meta-analysis of running performance, delayed-onset muscle soreness and oxidative stress [28] [29] [7], a study of phototherapy after exercise-induced muscle damage [30], reviews of PBM as sports medicine and of red light for performance and systemic healing [4] [3] [31], and reports extending to injury rehabilitation, osteoarthritis, skin and the psychological domain [32] [33] [34] [35].

For each domain, the review prioritised the most informative and methodologically transparent sources and sought, wherever possible, to confront convergent and divergent findings rather than to report only the positive signal. Particular attention was paid to the dose-response relationship and to the methodological factors — small samples, heterogeneous parameters, blinding, timing of application and the laser-versus-LED distinction — that condition the results. Consistent with the narrative design, no formal risk-of-bias scoring or quantitative pooling was performed; instead, the coherence of conclusions and the limitations of individual studies are discussed explicitly throughout the text.

3. The Mitochondrial Mechanism: From Photon to Bioenergetics

3.1. Cytochrome c oxidase as the primary photoacceptor

The mechanistic foundation of PBM is the absorption of red and near-infrared photons by cytochrome c oxidase, complex IV of the mitochondrial electron transport chain, which transfers electrons from cytochrome c to molecular oxygen, producing water while translocating protons to build the gradient that ATP synthase uses to generate ATP [1]. Cytochrome c oxidase contains two haem centres and two copper centres, each able to exist in oxidised or reduced states with distinct absorption spectra, allowing the enzyme to absorb light well into the near-infrared region up to about 950 nm [1]. The seminal observation, first proposed by Tiina Karu and confirmed by Wong-Riley and colleagues, was that the action spectrum of PBM effects matches the absorption spectrum of cytochrome c oxidase, and this congruence — together with the favourable tissue penetration of longer wavelengths — explains the predominance of red and near-infrared light in the field [1]. The most widely accepted explanation for how photon absorption increases enzyme activity is the photodissociation of inhibitory nitric oxide, which is non-covalently bound to the haem and copper centres and competitively displaces oxygen; a relatively low-energy photon can eject the nitric oxide and restore respiration [1] [2].

This mechanism has been directly demonstrated in living human tissue. Using broadband near-infrared spectroscopy, a 1064 nm laser applied to the human forearm produced significant, dose-dependent increases in the concentration of oxidised cytochrome c oxidase and of oxygenated haemoglobin, with a strong linear coupling between the two — the first in-vivo evidence that PBM up-regulates the enzyme and that this is accompanied by a hemodynamic oxygenation response, with the rise in cytochrome c oxidase preceding the rise in oxygenated haemoglobin and the effect shown not to be thermal [5]. That cytochrome c oxidase is an inducible enzyme means that, beyond acute activation, prolonged PBM can up-regulate its concentration: a

chronic transcranial laser study in young and aged rats mapped regional cytochrome c oxidase activity across the brain and found that 58 days of 810 nm stimulation predominantly reversed age-related declines in enzyme activity and functional connectivity, raising activity in aged animals toward the levels of young controls — a longer-lasting metabolic effect attributable to the synthesis of new enzyme subunits [11]. The same study illustrated the dose dependence discussed below, since the chronic stimulation lowered cytochrome c oxidase in young brains with already-high baseline activity while raising it in aged brains, consistent with a hormetic response [11].

The biophysics of why red and near-infrared light is preferentially used become clear when the absorption and penetration properties of tissue are considered. Both absorption and scattering of light by tissue decrease as wavelength lengthens, so the penetration depth of near-infrared light reaches a maximum at about 810 nm, beyond which water begins to absorb and penetration falls again; this creates an "optical window" of roughly 600–1100 nm within which therapeutic light can reach deep tissue, with a notable region between about 700 and 770 nm where results tend to be disappointing because of low chromophore absorption [1]. The photodissociation of nitric oxide is energetically favourable because nitric oxide, although bound to the haem and copper centres of cytochrome c oxidase, competitively blocks oxygen at a ratio of about one to ten, so that a single relatively low-energy photon can displace the inhibitor and unblock a substantial amount of respiration — an efficiency that explains how the low fluences characteristic of PBM can produce measurable bioenergetic effects [1]. That the same chronic 810 nm stimulation strengthened systems-level functional connectivity between brain regions in aged animals, raising inter-regional correlations of enzyme activity toward youthful values, further demonstrates that the enzymatic effect is not merely local but propagates to integrated, network-level function — a finding whose analogue in muscle would be the coordinated recovery of an entire working-muscle group rather than of isolated fibres [11].

3.2. Secondary signalling: ATP, reactive oxygen species, nitric oxide and calcium

Photon absorption by cytochrome c oxidase initiates a cascade of secondary effects that constitute the biological signal of PBM. The increase in enzyme activity raises the mitochondrial membrane potential and accelerates oxidative phosphorylation, producing more ATP and a brief, modest burst of reactive oxygen species, alongside changes in nitric oxide and intracellular calcium [1] [2]. The greater availability of ATP and the redox shift activate kinases and second messengers — cyclic adenosine monophosphate and calcium — that drive transcription factors at the nuclear level, leading to improved cell survival, proliferation and migration and to new protein synthesis [1] [2]. A central and initially paradoxical feature is the role of reactive oxygen species: in normal healthy cells, PBM produces a controlled increase in ROS that, at a fluence of about 3 J/cm^2 of 810 nm light, is sufficient to activate the redox-sensitive transcription factor NF-κB, while in oxidatively stressed cells the same treatment instead lowers ROS and restores the mitochondrial membrane potential toward baseline [1]. This context-dependent behaviour — pro-oxidant in resting cells, anti-oxidant in

stressed cells — reconciles the apparent contradiction between PBM generating ROS and PBM reducing markers of oxidative stress, and it underlies the therapeutic relevance of the modality to the oxidatively stressed muscle of the exercising athlete [1] [7].

Single-cell studies have confirmed and quantified these mitochondrial responses. In human adipose-derived mesenchymal stem cells, a single-cell 830 nm laser system showed that a fluence of 5 J/cm^2 significantly enhanced mitochondrial membrane potential, ROS production and intracellular vesicle transport relative to untreated controls, demonstrating directly the modulatory effect of PBM on the bioenergetic machinery that underlies proliferation, migration and differentiation [12]. These observations dovetail with the in-vivo coupling of enzyme up-regulation and tissue oxygenation, providing a continuous mechanistic chain from the absorption of a photon to a measurable change in cellular energetics and signalling [5] [12].

The signalling consequences of the ROS burst have been dissected with elegant experiments that clarify why the same molecule can be both signal and stressor. When the brief, modest increase in ROS produced by 3 J/cm^2 of 810 nm light activates NF-κB in fibroblasts, the addition of the antioxidant N-acetyl-cysteine blocks the NF-κB activation but does not block the increase in cellular ATP — demonstrating that the ATP rise and the redox signal are partly separable downstream events of the same mitochondrial stimulation [1]. The proposed reconciliation of PBM's pro- and anti-oxidant faces turns on the rate of superoxide generation: if superoxide is produced slowly enough for superoxide dismutase to convert it to diffusible hydrogen peroxide, the uncharged peroxide escapes the mitochondrion to activate beneficial signalling pathways, whereas if it is produced faster than the enzyme can detoxify it, the charged superoxide accumulates and damages the organelle [1]. At the level of cell fate, the increased ATP availability is thought to shift cellular priorities from glycolysis toward oxidative phosphorylation — a metabolic switch known to be a key driver of differentiation programmes such as osteogenesis — while ROS acting as a second messenger, together with calcium entering through light-gated channels and the consequent activation of nitric oxide and cyclic AMP, converges on the transcription factors that govern proliferation, differentiation and migration [2]. This integrated account, in which energy supply and redox signalling jointly steer the cell, is the conceptual bridge from the mitochondrial mechanism to the regenerative and recovery effects that follow in muscle [2] [14].

3.3. Other chromophores: opsins, light- and heat-gated ion channels, and water

Although cytochrome c oxidase is the dominant photoacceptor for red and near-infrared light, it is not the only one, and the recognition of additional chromophores broadens the mechanistic picture in ways relevant to dosimetry. Light-sensitive G-protein-coupled receptors of the opsin family, which respond chiefly to blue and green light, can open light-gated ion channels of the transient receptor potential (TRP) superfamily, permitting the influx of calcium, sodium and magnesium and thereby modulating intracellular signalling [1] [2]. At wavelengths beyond about 1000 nm, where

cytochrome c oxidase absorption wanes yet biological effects persist, structured or interfacial water is proposed as an alternative chromophore, whose absorption of infrared photons may perturb heat-gated TRP channels and modulate calcium without bulk heating of the tissue [1]. The functional importance of TRP channels for muscle-relevant cells was shown directly in meniscus-derived stem cells, in which PBM increased intracellular calcium and ROS in a dose-dependent manner and improved proliferation, with inhibition of the TRPV1 channel abolishing these effects — establishing a TRPV1-calcium-ROS signalling axis as a route by which PBM regulates stem-cell function independent of, or alongside, cytochrome c oxidase [18]. The plurality of chromophores, each with its own spectral sensitivity, helps explain why wavelength is such a critical determinant of the PBM response and why red and near-infrared windows behave differently from blue and green light [1] [2].

The water-as-chromophore hypothesis, although still partly speculative, addresses a genuine puzzle: biological effects continue to be observed at infrared wavelengths beyond about 1000 nm, where cytochrome c oxidase no longer absorbs appreciably, so an alternative absorber must be responsible [1]. Water — by far the most abundant molecule in tissue, with absorption peaks near 980 nm and at most wavelengths beyond 1200 nm — is the obvious candidate, and the proposed mechanism involves selective absorption of infrared photons by structured or interfacial water layers at power levels too low to heat the bulk tissue, with a small increase in the vibrational energy of a water cluster on a heat-sensitive protein such as a TRP channel sufficient to open it and admit calcium [1]. It has even been suggested that PBM could reduce the viscosity of interfacial water within the mitochondrion, allowing the ATP synthase, which rotates as a molecular nanomotor, to turn faster and generate ATP more efficiently [1]. The opsin and cryptochrome families add a further dimension relevant to systemic and circadian effects: cryptochromes, which rely on a flavin to absorb blue or green light, are implicated in the regulation of the circadian clock through signalling to the suprachiasmatic nucleus, a connection that links light exposure to sleep, recovery and the endurance benefits sometimes attributed to systemic red-light therapy [1] [3]. The practical message of this chromophore plurality is that the choice of wavelength is not a matter of convenience but selects which photoacceptor — and therefore which downstream pathway — is engaged, a consideration too often neglected when devices are compared without regard to their spectral output [1] [2].

3.4. The biphasic dose response

No principle is more important to the clinical interpretation of PBM than the biphasic dose response, often called the Arndt-Schulz law or hormesis: low doses stimulate, moderate doses may inhibit, and high doses can abolish or reverse the biological effect [1] [8]. In cultured cortical neurons, 810 nm light produced a biphasic dose-response in ATP production and mitochondrial membrane potential with a maximum near 3 J/cm^2, while a high dose of 30 J/cm^2 actually lowered the membrane potential below baseline, and the ROS dose-response curve showed two distinct maxima corresponding to the two extremes of membrane potential [1]. A systematic appraisal of light parameters across

more than a thousand studies plotted energy density against power density and found that, although results were highly variable, cells and tissues with high mitochondrial content (muscle, brain, heart, nerve) tended to respond to lower doses than tissues with fewer mitochondria (skin, tendon, cartilage), and that ineffective studies in high-mitochondrial tissues were more often the result of over-dosing than of under-dosing [8]. This insight is directly pertinent to sport, because skeletal muscle is a mitochondria-rich tissue for which an excessive dose may be counterproductive, and it reframes many "negative" PBM trials not as evidence of inefficacy but as possible consequences of mis-set parameters [8] [9]. The biphasic principle therefore demands that effective dose windows be defined for each target tissue, a requirement that the heterogeneity of the literature has so far failed to meet [8].

The scale of the dosimetric disagreement in the field is itself revealing and helps explain why so many trials reach contradictory conclusions. Some authorities recommend a power density below about 100 mW/cm^2 and an energy density of 4–10 J/cm^2 delivered to the target tissue, whereas others advocate as much as 50 J/cm^2 at the tissue surface, a range so wide that two protocols described by the same name may differ by an order of magnitude in the dose reaching the muscle [8]. Because more than a thousand research articles have reported that wavelength, energy density, power density, total energy, pulse structure, spot size, tissue absorption and treatment repetition can each affect the outcome, the number of interacting variables is formidable, and the failure to control them within a limited range is the proximate cause of the field's irreproducibility [8]. The appraisal's central empirical finding — that high-mitochondrial tissues respond to lower doses and that their negative studies cluster on the over-dosing side of the curve — carries a specific and counter-intuitive warning for sports practice: when a muscle trial fails to show benefit, the instinct to increase the dose may be exactly wrong, since the muscle, rich in mitochondria, may already have been pushed past the peak of its dose-response curve into the inhibitory region [8] [13]. This reframing transforms the interpretation of the negative trials reviewed later, suggesting that at least some represent mis-dosing rather than a true absence of effect, and it elevates the establishment of muscle-specific dose windows from a methodological nicety to the central unsolved problem of the discipline [8] [9].

Table 1. Mechanistic cascade of photobiomodulation, from photon absorption to functional effect.

Level	**Event**	**Functional consequence**
Primary chromophore	Photon absorption by cytochrome c oxidase; dissociation of inhibitory nitric oxide	Increased electron transport and oxygen consumption
Mitochondrial	Raised membrane potential; increased ATP; brief ROS burst	Greater cellular energy availability

Level	Event	Functional consequence
Secondary signalling	Nitric oxide, calcium, cyclic AMP; activation of NF-κB and other transcription factors	Gene expression; survival, proliferation, migration
Alternative chromophores	Opsins and TRP channels (blue/green and NIR); water (far-IR)	Calcium influx; signalling modulation
Dose governance	Biphasic (Arndt-Schulz) response	Stimulation at low dose, inhibition at high dose

Sources: [1] [5] [2] [12] [18] [8].

4. From Mechanism to Muscle: Cellular and Tissue Evidence

4.1. Myoblast differentiation and skeletal-muscle regeneration

Because the recovery and adaptation of skeletal muscle depend on the satellite-cell-driven processes of regeneration, the effect of PBM on myogenic cells is central to its rationale in sport. In murine myoblasts undergoing differentiation, red PBM delivered by a 635 nm laser diode, particularly at an energy density of 4 J/cm^2, did not alter cell viability but promoted the expression of the myogenic transcription factors MyoD and myogenin, enhanced myotube formation, increased mitochondrial metabolism and biogenesis, and — through electrophysiological changes in membrane properties and inward ion currents — drove the cells toward a more differentiated phenotype, while also increasing the release of pro-myogenic extracellular vesicles [14]. This pattern, with an optimal intermediate dose and a clear pro-myogenic signature, exemplifies both the regenerative potential of PBM and the biphasic dose dependence that governs it [14] [8]. A complementary study in muscle cells subjected to oxidative stress reinforced the protective dimension: laser PBM at 660 and 808 nm, especially 10 J at 808 nm applied before the oxidative insult, preserved cell viability and markedly up-regulated MyoD, with myogenin also expressed, indicating that light can defend myoblasts against oxidative damage while activating the genes that initiate regeneration [15]. Together these in-vitro findings provide a cellular basis for the claim that PBM supports muscle repair, the process on which an athlete's recovery from damaging exercise ultimately depends [14] [15].

The breadth of the pro-myogenic response merits closer description because it engages several of the signalling systems that matter for recovery. In differentiating myoblasts, red PBM not only raised the myogenic transcription factors but enhanced mitochondrial metabolism and biogenesis and altered the cells' passive membrane properties and inward ion currents, electrophysiological signatures of a more mature, contraction-competent phenotype, and it increased the secretion of extracellular vesicles carrying a

pro-myogenic cargo — a paracrine mechanism by which one treated cell can propagate the regenerative signal to its neighbours [14]. Mature myotubes treated with the same red light retained their viability and dimensions while increasing their release of pro-myogenic vesicles, indicating that the benefit is not confined to the early differentiation window [14]. The oxidative-stress study adds quantitative precision and a striking dose finding: hydrogen peroxide at 50 μM reduced myoblast viability by about a quarter, and although several PBM doses simply protected viability to control levels, the 808 nm 10 J condition applied before the insult raised viability beyond controls and produced the most pronounced up-regulation of MyoD, far exceeding the 660 nm and lower-energy conditions [15]. These cellular results are consistent with in-vivo animal studies in which low-level laser at around 780–810 nm and 10 J modulated the expression of MyoD and myogenin during muscle repair after injury, lending convergent support across model systems to the proposition that an appropriately dosed red or near-infrared exposure activates the genetic programme of regeneration [15] [14]. The recurrent appearance of an intermediate optimal dose — 4 J/cm² for differentiation, 10 J for protection against oxidative injury — within these myogenic studies is itself a microcosm of the biphasic principle and a reminder that the cellular evidence, like the clinical evidence, is dose-bounded [14] [8].

4.2. Mitochondrial bioenergetics, ageing and the source question

The bioenergetic effects of PBM in muscle cells are real but source- and dose-specific, a nuance with practical implications for device selection. In C2C12 myoblasts studied across replicative ageing, single-cell screening identified 5 J/cm² as the optimal fluence for both a 660 nm LED and an 830 nm near-infrared laser, each producing biphasic increases in mitochondrial membrane potential and ROS; at the population level, however, the two sources diverged, with LED irradiation eliciting stronger metabolic activation and ATP production — particularly in aged cells — while near-infrared laser more robustly enhanced myogenic fusion and partially rescued the differentiation deficits of aged myoblasts [13]. This demonstration that wavelength and source differentially direct metabolic, paracrine and myogenic outputs, even when the mitochondrial signal is conserved, undermines any assumption that lasers and LEDs are interchangeable and helps account for divergent clinical results [13] [8]. The paracrine dimension of this study is particularly noteworthy: the release of extracellular vesicles, the membrane-bound packages through which cells communicate, showed an age-dependent sensitivity to the light source, with near-infrared laser favouring the canonical small-vesicle population in young cells and LED inducing a greater overall particle release in aged cells [13]. Because extracellular vesicles carry the signals by which a treated cell influences its neighbours, this finding implies that the choice of wavelength and device does not merely set the magnitude of the mitochondrial response but shapes the intercellular communication that propagates it through the tissue — a level of source-specificity that no simple "light is light" account can accommodate and that strengthens the case for treating laser and LED, and red and near-infrared, as distinct interventions requiring their own dose optimisation [13] [14]. The relevance of

PBM to muscle ageing — and by extension to the maintenance of muscle quality in masters athletes — is further supported by an ultrastructural study of aged rat vastus lateralis, in which PBM (650 nm, 6 J/cm^2, four daily sessions) produced compensatory reorganisation of the neuromuscular junction, increasing the number of presynaptic active zones, elongating the postsynaptic membrane, narrowing the synaptic cleft and inducing mitochondrial hyperplasia in presynaptic terminals, all interpreted as signs of enhanced functional activity that might mitigate age-related sarcopenia and weakness [16]. These tissue-level findings extend the mitochondrial mechanism from the isolated cell to the integrated neuromuscular apparatus [16] [13].

The ultrastructural detail of the ageing study is worth dwelling on because it documents both the degeneration that PBM opposes and the specific reorganisation it induces. Control muscle in the aged rats showed the characteristic signs of skeletal-muscle senescence — variability in fibre diameter, centrally located nuclei, increased connective tissue, myofibril fragmentation, sarcomere disorganisation, accumulation of lipofuscin and the formation of tubular aggregates — against which the irradiated neuromuscular junctions displayed a coordinated set of changes consistent with heightened functional activity: more presynaptic active zones, an elongated postsynaptic membrane, a narrowed synaptic cleft, mitochondrial hyperplasia in the presynaptic terminals and a reduced total number of synaptic vesicles, the last interpreted as a sign of accelerated vesicle turnover rather than depletion [16]. Because degeneration of the neuromuscular junction is a principal driver of denervation, muscle weakness and sarcopenia, the demonstration that four brief daily exposures can drive compensatory synaptic remodelling is directly relevant to the preservation of muscle quality in older and masters athletes [16]. At the subcellular level, the single-cell stem-cell work complements this picture by showing that PBM at 5 J/cm^2 enhances not only mitochondrial membrane potential and ROS but also intracellular vesicle transport, a readout of the cytoskeletal and energetic activation that underpins the migration and secretory functions on which tissue repair depends [12]. Read together, the single-cell, myoblast and neuromuscular-junction studies trace a coherent arc from the energised mitochondrion to the remodelled synapse, the same arc that, in the exercising athlete, would connect a pre-exercise light exposure to a more resilient and recoverable neuromuscular system [12] [16].

4.3. Metabolic effects and the broader cellular context

PBM also engages the metabolic pathways that govern muscle fuel handling, an effect of interest for both performance and metabolic health. In two diabetic mouse models, PBM reduced blood glucose and insulin resistance and reversed metabolic abnormalities in skeletal muscle by elevating cytochrome c oxidase activity, which accelerated ATP and ROS generation; the ROS, in turn, activated the PTEN/AKT signalling pathway, promoting translocation of the GLUT4 glucose transporter and activation of glycogen synthase to enhance glucose uptake and glycogen synthesis, with the effect abolished by cytochrome c oxidase deficiency, ROS elimination or AKT inhibition [17]. This study not only confirms the centrality of cytochrome c oxidase and ROS to the muscle response but

links PBM to the same insulin-sensitising, glucose-handling pathways that exercise itself recruits, suggesting a metabolic dimension to the modality beyond acute energy provision [17]. The wider cellular literature situates these muscle-specific findings within a general capacity of PBM to enhance stem-cell proliferation, migration and differentiation across many tissues — gingival and dermal fibroblasts, bone-marrow and adipose mesenchymal stem cells, osteoblasts — through the same mitochondrial, ROS and transcription-factor mechanisms, again under strict parameter control and with an optimal fluence commonly in the 1–5 J/cm^2 range [2] [12]. The consistency of the mechanism across cell types, coupled with its dose dependence, is the through-line connecting the biophysics of Section 3 to the applied questions that follow [2] [8].

The glucose-handling study repays detailed reading because it traces a complete molecular pathway from light to metabolic outcome and identifies the necessary intermediates by selective inhibition. PBM raised cytochrome c oxidase activity, which accelerated the generation of ATP and ROS; the transient ROS inactivated the phosphatase PTEN, relieving its inhibition of the AKT signalling pathway, and the resulting AKT activation promoted both the translocation of the GLUT4 glucose transporter to the membrane and the activation of glycogen synthase, so that glucose uptake and glycogen synthesis increased in the muscle of two independent diabetic mouse models [17]. The causal chain was confirmed by the loss of effect when cytochrome c oxidase subunit III was deficient, when ROS was scavenged, or when AKT was inhibited, establishing each as a required node [17]. Because these are precisely the pathways through which exercise itself enhances insulin sensitivity and muscle glucose storage, the study positions PBM as a stimulus that partially mimics the metabolic signalling of exercise, with implications for glycogen replenishment and substrate handling in athletes as well as for metabolic health [17]. The TRPV1 evidence from meniscus-derived stem cells widens the mechanistic repertoire further: across wavelengths from the blue to 1064 nm and energy densities from 3 to 60 J/cm^2, proliferation and mitochondrial function improved most at 700–710 and 1064 nm with 15 J/cm^2, whereas other conditions reduced both, and blockade of TRPV1 abolished the calcium and ROS responses at all wavelengths — again demonstrating both a clear optimal dose and a calcium-channel route to the regenerative effect [18]. The recurring lesson across these metabolic and stem-cell studies is that the favourable outcome is reproducibly bounded by an intermediate optimal dose and is mediated by identifiable molecular intermediates, reinforcing both the plausibility of the mechanism and the centrality of dosimetry to its expression [17] [18].

The wider regenerative literature, although not specific to muscle, frames these findings within a general capacity of PBM to direct stem-cell fate that is highly relevant to the tissues athletes injure. PBM has repeatedly been shown to enhance the proliferation of human gingival and dermal fibroblasts and the proliferation, migration and differentiation of bone-marrow and adipose-derived mesenchymal stem cells, with osteogenic differentiation in particular shown to be dose-dependent — cultures exposed to about 4 J/cm^2 yielding more osteoblasts than those exposed to 2 J/cm^2 — and with

most studies agreeing that a fluence of roughly 1–5 J/cm^2 is optimal for proliferation, beyond which ROS becomes toxic and proliferation falls, a cellular restatement of the biphasic law [2]. The mechanism proposed for these effects extends the mitochondrial account into the territory of regeneration: the ATP-rich, redox-shifted state induced by PBM is thought to bias cells from glycolysis toward oxidative phosphorylation, a metabolic switch known to favour differentiation, while ROS acting through transcription factors such as HIF-1α promotes the expression of vascular endothelial growth factor and thereby angiogenesis, the blood-vessel formation on which the healing of bone, tendon and muscle depends [2]. The same literature is candid about its inconsistencies, noting that some studies find no change in stem-cell activity after irradiation and that results are often difficult to replicate — a difficulty attributed, once again, to the multiplicity of parameters and the narrowness of the effective dose window [2]. For the athlete, the significance of this broader regenerative biology is that the tissues most often injured in sport — muscle, tendon, ligament, bone — all depend on the proliferation, differentiation and vascularisation of progenitor cells that PBM can, under the right dose, stimulate, providing a unifying cellular rationale that spans the performance, recovery and rehabilitation domains treated separately in this review [2] [14].

5. Ergogenic Effects: Performance Enhancement in Athletes

5.1. Pre-exercise photobiomodulation and aerobic performance

Beyond its acute use immediately before a single bout, PBM is increasingly proposed as an adjunct to training itself, applied repeatedly across a programme to potentiate the chronic adaptations that exercise produces. The rationale is that if a pre-session exposure enhances the energetic and oxidative environment of each workout, the cumulative effect over weeks might be greater than the sum of acute effects, improving the trajectory of adaptation rather than merely the performance of one session [24] [21]. The eight-week running study in untrained men was designed precisely to test this chronic hypothesis, combining LED PBM before every training session with a structured endurance programme and finding a possibly positive longitudinal effect on peak velocity and 5-km time [24]. A complementary and more speculative proposal concerns detraining: because PBM has been shown to improve the oxygen-carrying capacity of blood and the oxidative capacity of muscle, a protocol was designed to test whether continued PBM during a period without training could attenuate the loss of aerobic conditioning that normally follows the cessation of exercise — a question of evident importance to athletes sidelined by injury or scheduling, for which no validated intervention exists [21]. These chronic and detraining applications remain less well evidenced than the acute pre-exercise use, and their outcome data are incomplete, but they illustrate that the ergogenic hypothesis is not confined to the single session and that PBM is being positioned as a modulator of the entire training process, with the same dosimetric caveats that govern its acute use [21] [4].

The most direct test of PBM as an ergogenic aid is whether pre-exercise application improves endurance and aerobic capacity, and several rigorous trials in athletes answer affirmatively. In a randomised, triple-blind, placebo-controlled crossover trial in high-level soccer players, infrared LLLT (810 nm, optimised dose) applied before a progressive treadmill test significantly increased peak oxygen uptake in both relative and absolute terms, prolonged time to exhaustion, and raised the volumes and times at which the aerobic and anaerobic thresholds occurred, while simultaneously decreasing post-exercise creatine kinase and lactate dehydrogenase and the inflammatory cytokine interleukin-6, and reducing oxidative-stress markers (thiobarbituric acid reactive substances and carbonylated proteins) with increased antioxidant enzyme activity [6]. The authors attributed the aerobic improvement to enhanced cytochrome c oxidase activity, increased ATP production and an antioxidant action, providing one of the clearest demonstrations that the mitochondrial mechanism can translate into measurable performance and biochemical benefit in elite athletes [6] [5]. The granular results of this trial are worth recording because they exemplify what a comprehensive positive study looks like: in twenty-two high-level soccer players studied in a triple-blind crossover design, pre-exercise infrared laser at an optimised dose (with multiple sites irradiated per limb) significantly increased relative and absolute peak oxygen uptake, prolonged time to exhaustion and raised both the time and the volume at which the anaerobic threshold occurred; it significantly reduced post-exercise creatine kinase and lactate dehydrogenase and the cytokine interleukin-6, while interleukin-1β and tumour necrosis factor-α were unchanged at the early time point; and it reduced lipid peroxidation (thiobarbituric acid reactive substances) and protein carbonylation while increasing the activity of the antioxidant enzymes superoxide dismutase and catalase [6]. This simultaneous improvement of aerobic capacity, muscle-damage markers, inflammation and redox balance in a single rigorous trial is the strongest existing demonstration that the mitochondrial mechanism can produce a coordinated, multi-system benefit, and the authors explicitly proposed up-regulated cytochrome c oxidase activity and an antioxidant action as the mediating mechanism [6] [7]. Consistent with this, a trial in untrained men found that an eight-week endurance running programme combined with pre-session LED PBM (a cluster of red 660 nm and infrared 850 nm diodes) produced, by magnitude-based inference, a possibly positive effect on peak running velocity and 5-km time relative to placebo, with moderate effect sizes and a moderate attenuation of muscle soreness in the middle weeks of training, although conventional statistical testing found no significant between-group difference — a pattern of small, potentially competitively meaningful but statistically fragile benefit that recurs throughout the field [24].

The aerobic picture is, however, not uniform. A double-blind crossover trial in trained cyclists examined PBM applied before successive time-to-exhaustion tests and oxygen-uptake kinetics, and dedicated trials of transcranial PBM aimed at central fatigue mechanisms found no improvement in cycling performance, heart rate, blood lactate or perceived exertion, the latter attributed to sub-cortical irradiance levels insufficient to engage the prefrontal cortex [22] [25]. The contrast between the positive peripheral-

muscle trial in soccer players and the null transcranial trial in cyclists underscores that the site of application matters: PBM directed at exercising muscle has a more consistent rationale and effect than PBM directed at the brain for central fatigue, where penetration and dosimetry are far more demanding [6] [25]. This contrast is not merely empirical but mechanistically expected, and it organises the ergogenic evidence into a coherent picture. The strongest positive findings — the multi-system improvement in soccer players and the dose-dependent stem-cell and myoblast responses — all involve light delivered directly to a mitochondria-rich peripheral tissue at an optimised local fluence, the configuration in which cytochrome c oxidase can be reliably engaged [6] [13]. The weakest or null findings cluster where this configuration breaks down: where the target is the cortex behind the skull, where the dose is diluted across the whole body, where the muscle irradiated is not the one exercised, or where the dose, the wavelength or the participant population lies outside the effective window [25] [20] [9]. Viewed this way, the apparently chaotic ergogenic literature resolves into a single ordering principle — the more faithfully a study delivers an adequate, optimised dose to the working muscle, the more likely it is to find benefit — and the role of the reviewer is less to adjudicate whether PBM "works" than to specify the conditions under which it does [6] [8]. The recognition that endurance gains may also be preserved across detraining — the rationale for a protocol testing whether PBM can attenuate the loss of aerobic conditioning during periods without training — illustrates the breadth of the ergogenic hypothesis, even though such protocols await definitive outcome data [21].

The cycling trials deserve fuller treatment because they probe the mechanism at the level of oxygen-uptake kinetics and central fatigue rather than gross performance. In well-trained cyclists with a training volume of roughly 460 km per week, PBM was applied to both thighs before three successive time-to-exhaustion tests at maximal power output, and the investigators measured not only performance but the amplitude and time delay of oxygen-uptake kinetics, the oxygen deficit and lower-limb muscle oxygenation — an attempt to detect a peripheral, muscle-oxygenation mechanism for any ergogenic effect [22]. The transcranial trial took the opposite tack, targeting the prefrontal cortex with a near-infrared device (810 nm, pulsed at 40 Hz) in eighteen trained cyclists across a constant-load test and a 25-minute self-paced time trial, and found no condition-by-time interaction for heart rate, blood lactate, perceived exertion or power-related ratios, concluding that the irradiance reaching the cortex was probably insufficient to engage central fatigue mechanisms and that higher dosimetry or repeated exposure, ideally validated by neuroimaging, would be required to test the hypothesis fairly [25]. These two cycling studies thus bracket the ergogenic question: one looks for a peripheral muscle-oxygenation effect and one for a central neuromodulatory effect, and the failure of the transcranial approach in particular illustrates how the penetration constraints that favour muscle applications work against brain applications [22] [25]. The detraining protocol extends the logic to a third timescale, hypothesising that the enhanced oxygen-carrying and oxidative capacity induced by PBM might blunt the loss of aerobic fitness when training is interrupted by injury or illness, a scenario of real importance to athletes but one for which outcome data are not yet available [21].

Together these designs show the ergogenic hypothesis being tested across acute, central and longitudinal dimensions, with the most secure positive signal remaining the peripheral, pre-exercise muscle application seen in the soccer trial [6] [22].

5.2. Strength, resistance exercise and the parameter dependence of benefit

For strength and resistance performance the evidence is more divided, and the divisions are instructive about the conditions for benefit. A meta-analysis of phototherapy reported that PBM applied before exercise can increase the number of muscular contractions performed before fatigue and raise peak torque, and reviews of the field cite improvements in strength, endurance and reduced fatigue with pre-exercise delivery [4] [31]. Yet a randomised crossover trial in untrained young women found that acute 808 nm laser PBM at a dose of 28 J did not improve biceps brachii performance to exhaustion, perceived exertion or delayed-onset muscle soreness, a null result the authors related both to the possibility that the dose window effective for small muscle groups in men may not apply to women and to the greater intrinsic fatigue resistance of women, which could mask a small effect [9]. This trial crystallises three of the field's key moderators — sex, training status and the size of the muscle irradiated (which determines how many points can be treated and thus the total energy delivered) — and cautions against extrapolating positive findings from one population or muscle to another [9] [8]. The importance of irradiating the working muscle directly was demonstrated by a trial in which PBM combined with a static magnetic field improved performance and reduced fatigue only when applied locally to the exercised muscle, with no benefit when a distant, non-exercised muscle was irradiated — confirming that the effect is a local tissue interaction rather than a systemic one and that correct application technique is decisive [20].

The local-versus-non-local trial repays detailed examination because its design isolates the question of whether PBM acts at the irradiated tissue or systemically. Thirty sedentary men were randomised so that active PBM combined with a static magnetic field was applied either to the quadriceps that would perform an eccentric fatigue protocol, to the contralateral non-exercised quadriceps, or as placebo, and only the locally irradiated group showed improved maximal voluntary contraction and reduced creatine kinase, blood lactate and delayed-onset muscle soreness, recovering fully within 48 hours and even exceeding baseline strength at 72 hours, whereas the non-local group was indistinguishable from placebo [20]. The investigators drew an instructive contrast with systemically acting drugs such as non-steroidal anti-inflammatories, whose action at sites distant from administration carries the risk of systemic adverse effects, noting that the strictly local action of PBM is in this respect a safety advantage as well as a constraint on application technique [20]. This locality has direct practical consequences: it means that every muscle group involved in an exercise must be irradiated, with adequate per-point illumination time, for an ergogenic effect to be expected, and it explains why convenience-oriented approaches that irradiate only part of the working musculature, or that rely on systemic exposure, may fail [20] [8]. The biceps trial in women complements this point from the dosing side, since its null result occurred

despite a dose of 28 J that lay within the 20–60 J range recommended for small muscle groups, prompting the authors to question whether that "therapeutic window," derived largely from studies in men, is valid for women, in whom greater fatigue resistance and possible hormonal modulation of endurance could mask a small effect [9]. The two trials together define the practical envelope of strength applications: the right tissue must receive the right dose, and neither the population nor the muscle from which a positive finding was derived can be assumed to generalise [9] [20].

5.3. The static-magnetic-field combination and the weight of the negative trials

Several of the strongest applied results come from protocols combining PBM with a static magnetic field (PBMT-sMF), which is proposed to enhance electron transfer and ATP production beyond PBM alone. In CrossFit athletes, PBMT-sMF applied before or after a high-intensity workout enhanced functional-test performance and decreased creatine kinase, interleukin-6 and oxidative-stress markers while increasing antioxidant activity, with the benefit seen when the therapy was applied at a single time point rather than both before and after — a finding the authors interpreted as a possible over-dosing effect when applications were doubled, again illustrating the biphasic principle in a clinical setting [19]. A later randomised crossover trial in CrossFit athletes compared PBMT-sMF with intermittent pneumatic compression, extracorporeal shock wave therapy and passive recovery, and found that although there was no difference in the primary one-hour countermovement-jump outcome, PBMT-sMF outperformed the other modalities at 24 and 48 hours, producing greater reductions in lactate dehydrogenase and oxidative-stress markers and higher antioxidant activity, with the benefits ascribed to modulation of oxidative stress [23]. These positive combination trials are counterbalanced by a substantial body of high-quality negative evidence: PBM applied before a stretch-shortening protocol did not attenuate fatigue or muscle damage in judo athletes; whole-body PBM light-bed therapy did not significantly reduce creatine kinase or salivary interleukin-6 after high-intensity resistance training in trained males; and acute biceps and cycling trials found no functional benefit [10] [27] [9] [22]. The juxtaposition of robust positive and robust negative trials, often using comparable populations, is the defining tension of the ergogenic literature and points firmly to parameters, timing and delivery mode as the explanatory variables rather than to any simple presence or absence of effect [10] [8].

The negative trials deserve to be characterised individually, because their methodological quality is precisely what makes the field's inconsistency so striking. In sixteen judo athletes, PBM applied to one limb and placebo to the contralateral limb before a stretch-shortening-cycle fatigue protocol produced no difference at any time point in countermovement-jump impulse, power, velocity or force, in echo-intensity measures of muscle damage in the rectus femoris and vastus lateralis, or in muscle soreness — a within-subject design that controls tightly for individual variability and still found nothing [10]. In twelve trained males, an acute dose of whole-body PBM delivered by a light bed before and after high-intensity resistance training did not significantly reduce the area under the curve for creatine kinase over 24–72 hours or for

salivary interleukin-6 over 60–120 minutes, despite the convenience and large treatment area of the whole-body approach [27]. By contrast, the static-magnetic-field combination trials that did show benefit used carefully optimised local doses: in the first CrossFit study, twelve athletes performed a standardised workout of calorie-based cycling, hang squat cleans and box jumps, and PBMT-sMF improved the number of squats performed and lowered creatine kinase, interleukin-6 and oxidative-stress markers when applied at a single time point, while the before-and-after application — delivering a double dose — produced mixed results that the authors explicitly attributed to possible over-dosing [19]. The comparative trial then pitted PBMT-sMF against intermittent pneumatic compression, extracorporeal shock wave therapy and passive recovery in twelve athletes, finding no difference in the one-hour countermovement jump but a clear advantage for PBMT-sMF at 24 and 48 hours in lactate dehydrogenase and in the oxidative-stress and antioxidant markers, even as the device-based comparators earned higher subjective satisfaction [23]. The instructive pattern is that the positive trials combined an adjunctive technology with deliberate dose optimisation and local application, whereas the negative trials used whole-body delivery or unoptimised parameters — consistent with the thesis that delivery and dosimetry, not the modality itself, separate success from failure [19] [27].

Table 2. Representative ergogenic and recovery trials of photobiomodulation in athletes and active individuals.

Study population	**Intervention**	**Principal outcome**
Elite soccer players	Pre-exercise 810 nm laser	Increased VO2peak, time to exhaustion; lower CK, LDH, IL-6, oxidative stress
Untrained men (8-wk running)	Pre-session LED PBM	Possibly positive on peak velocity and 5-km time (non-significant)
CrossFit athletes	PBMT-sMF before/after workout	Improved performance; lower CK, IL-6, oxidative stress
CrossFit athletes (comparative)	PBMT-sMF vs IPC, ESWT, passive	Superior at 24–48 h; lower LDH and oxidative stress
Untrained women	Acute 808 nm laser, biceps	No effect on performance, RPE or soreness
Judo athletes	Pre-exercise PBM	No effect on fatigue or muscle damage
Trained males	Whole-body PBM light-bed	No significant change in CK or salivary IL-6

Study population	Intervention	Principal outcome
Trained cyclists	Transcranial PBM	No effect on performance or physiological responses

Sources: [6] [24] [19] [23] [9] [10] [27] [25].

6. Recovery: Muscle Damage, Soreness, Inflammation and Oxidative Stress

6.1. Delayed-onset muscle soreness and the recovery of muscle function

Beyond acute performance, the larger market for PBM in sport concerns recovery — the attenuation of delayed-onset muscle soreness (DOMS), the restoration of strength, and the limitation of the biochemical disturbance that follows damaging exercise. A systematic review and meta-analysis of photomodulation therapy for DOMS, drawing on 14 controlled studies with wavelengths from 660 to 950 nm, found statistically significant reductions in visual-analogue soreness scores at 72 and 96 hours after the induction of DOMS (pooled standardised mean differences of about −0.55 and −0.56, a moderate effect) and significant improvements in muscle strength at 24 and 48 hours (pooled standardised mean differences of about 0.97 and 0.99, a large effect), concluding that PBM may reduce pain, enhance strength recovery and lower biochemical markers of damage [29]. A narrative synthesis of the same domain reached a similar but more guarded conclusion, holding that most studies show PBM can reduce DOMS, improve functional recovery and attenuate markers such as creatine kinase through enhanced mitochondrial activity and modulation of oxidative stress and inflammation, while emphasising that some trials report no benefit and that variability in parameters, timing, exercise modality and participant characteristics limits firm conclusions [31]. These meta-analytic and narrative summaries thus support a modest, real, but parameter-sensitive recovery benefit, consistent with the cellular evidence that PBM aids myoblast survival and differentiation [29] [15].

The structure of the DOMS meta-analysis exposes both the promise and the fragility of the recovery evidence. Of 336 articles initially identified, 14 met the inclusion criteria — ten of parallel and two of crossover design, with wavelengths from 660 to 950 nm applied to one to six points on the affected muscle and methodological quality on the Physiotherapy Evidence Database scale ranging from 4 to 9 — but only four reported their outcomes with sufficient clarity to enter the quantitative synthesis, so that the pooled soreness and strength estimates rest on a narrow base, and the between-study heterogeneity was substantial (for soreness at 72 hours the I^2 statistic reached about 67%) [29]. Across the wider set, four studies found no significant effect while ten reported positive effects, and the analgesic action was attributed to the familiar mitochondrial mechanism — stimulation of cytochrome c oxidase, increased ATP synthesis, suppression of pro-inflammatory cytokines such as tumour necrosis factor-α and interleukin-6, and enhanced nitric-oxide-mediated microcirculation that clears

inflammatory metabolites [29]. The narrative review of the same topic adds biological texture: it locates the origin of DOMS in ultrastructural microtrauma to muscle fibres and a localised inflammatory response peaking at 24–72 hours, and it catalogues both supportive trials — including a randomised comparison in which pulsed low-level laser combined with light-emitting diodes produced greater reductions in soreness and better preservation of maximal voluntary contraction than continuous laser or sham — and clearly negative trials in untrained or low-status participants, concluding that pulsing, wavelength choice, energy density, timing and training status jointly determine whether benefit appears [31]. The convergence of a moderate meta-analytic effect on soreness with a large but more heterogeneous effect on strength recovery, set against a substantial minority of null trials, is the most honest single summary of where the recovery evidence stands [29] [31].

A mechanistically important but under-measured dimension of recovery is the effect of PBM on microcirculation and tissue oxygenation, which links the soreness and biochemical findings to the in-vivo coupling demonstrated in the human forearm. Exercise-induced damage and fatigue are accompanied by transient impairments in muscle blood flow and oxygen delivery, and efficient perfusion is essential both for delivering nutrients to repairing tissue and for clearing the metabolic and inflammatory by-products that contribute to soreness [31]. PBM is thought to enhance local microcirculation chiefly through nitric oxide: the same photodissociation of nitric oxide from cytochrome c oxidase that unblocks respiration also increases the bioavailability of nitric oxide as a vasodilator, promoting smooth-muscle relaxation, increased blood flow and improved oxygen delivery to the recovering muscle [31] [1]. This vasodilatory, perfusion-enhancing action provides a coherent bridge between the molecular mechanism and the clinical observation of reduced soreness and faster functional recovery, and it is consistent with the direct demonstration that PBM increases oxygenated haemoglobin in irradiated human tissue in tight coupling with cytochrome c oxidase up-regulation [31] [5]. The dermatological literature reinforces the same vascular theme, attributing part of red light's wound-healing and rejuvenating action to enhanced angiogenesis and microcirculation through vascular endothelial growth factor and nitric oxide [34]. Although direct measurements of microcirculatory change in human muscle PBM studies remain scarce, the convergence of the molecular, dermatological and tissue-oxygenation evidence makes improved perfusion a plausible third pillar of the recovery effect, alongside the redox modulation and the regenerative cellular signalling discussed elsewhere [31] [5].

6.2. Biochemical markers, inflammation and the inconsistency of effect

The trial-level evidence on biochemical recovery markers is genuinely mixed, and the inconsistency is itself an important finding. On the positive side, the soccer-player trial showed clear reductions in creatine kinase, lactate dehydrogenase and interleukin-6 after pre-exercise PBM, and the CrossFit trials showed reductions in creatine kinase and lactate dehydrogenase with PBMT-sMF [6] [19] [23]. On the negative side, PBM by LED did not alter inflammation (interleukin-10, tumour necrosis factor-α), muscle-damage

markers (creatine kinase, lactate dehydrogenase), soreness or jump performance after sprint-interval training, and produced outcomes no better than cold-water immersion or active recovery; whole-body PBM did not significantly reduce creatine kinase or salivary interleukin-6 after resistance training; and PBM did not attenuate fatigue or echo-intensity-measured muscle damage in judo athletes [26] [27] [10]. A study of phototherapy after exercise-induced muscle damage from repeated sprinting found a benefit only for calf soreness, with no effect on vertical jump, agility, or quadriceps and hamstring soreness, and the authors questioned whether even the statistically significant soreness reduction was practically meaningful, noting that PBM may not benefit short, explosive activities limited by neurological rather than metabolic factors and may be less effective in trained than untrained individuals [30]. The pattern across these trials suggests that PBM's recovery benefit, where present, is most evident for endurance-type, metabolically demanding exercise and for the subjective experience of soreness, and least evident for explosive, neurologically limited performance and in already-trained populations [30] [26].

Two of the negative trials are worth examining in detail because their designs are rigorous and their interpretations illuminate the boundaries of the effect. In the sprint-interval study, thirty-six men performed two interval sessions twenty-four hours apart to mimic accumulated training stress, and in a first, placebo-controlled phase PBM by LED produced no interaction with placebo for interleukin-10, tumour necrosis factor-α, creatine kinase, lactate dehydrogenase, delayed-onset muscle soreness or countermovement jump, while in a second phase it was no more effective than active recovery or cold-water immersion; only a time effect was seen, with the expected rise and fall of damage markers and soreness regardless of treatment [26]. The phototherapy-after-sprinting study is equally instructive: thirty-three participants performed forty 15-metre sprints with a deceleration zone designed to accentuate eccentric damage, and assessment by both algometry and a general labelled magnitude scale found a significant reduction only in calf soreness, with no benefit for vertical jump, agility, or quadriceps and hamstring soreness, and the authors doubted whether even that isolated soreness reduction was practically meaningful given its small magnitude near the limit of scale sensitivity [30]. Their proposed explanation is mechanistically coherent and recurs across the literature: PBM appears to aid recovery of maximal voluntary contraction — a high-force, low-velocity task limited by metabolic fatigue — more than recovery of jumping and agility, which are low-force, high-velocity tasks constrained chiefly by neurological factors, and the benefit is more readily demonstrated in untrained volunteers than in trained athletes whose adaptations may leave less room for improvement [30]. This functional taxonomy — metabolic versus neurological limitation, trained versus untrained — provides a principled way to predict where PBM will and will not help, and it accommodates both the positive endurance findings and the negative explosive-performance findings within a single framework [30] [26].

6.3. Oxidative-stress modulation as the unifying mechanism

To appreciate why oxidative-stress modulation is the most mechanistically satisfying of PBM's recovery effects, it is necessary to recall the role of reactive oxygen species in exercising muscle. Muscle contraction itself generates reactive oxygen species, and at low to moderate levels these species are not merely harmful but serve as intracellular signalling molecules that regulate contractile function, force production and the adaptive responses to training [7]. The problem arises when production outstrips the antioxidant defences, producing the imbalance — defined as a disruption of redox signalling and control in favour of oxidants — that constitutes oxidative stress, with consequent damage to lipids, proteins and nucleic acids, impairment of contractile function, and a contribution to fatigue; higher levels of oxidative damage are associated with a longer post-exercise recovery period, and during intense training the production of reactive oxygen species can overwhelm antioxidant capacity and impair performance [7]. This dual character of reactive oxygen species — beneficial signal at low levels, damaging stressor at high levels — exactly parallels the behaviour of PBM, which produces a controlled, signalling ROS burst in healthy cells but reduces ROS in already-stressed cells by restoring the mitochondrial membrane potential [7] [1]. The therapeutic logic is therefore precise: an intervention that lowers the rate of ROS generation and bolsters antioxidant enzymes in the oxidatively stressed muscle of the recovering athlete should, in principle, hasten the resolution of the redox imbalance without abolishing the beneficial signalling role of reactive oxygen species in adaptation — and this is what the oxidative-stress evidence suggests PBM does [7] [23]. Understanding this exercise-physiology background makes clear why the redox effect is both the most reproducible and the most mechanistically interpretable of PBM's actions in sport [7] [29].

The most mechanistically coherent recovery evidence concerns oxidative stress, the disturbance most directly linked to the mitochondrial action of PBM. A systematic review and meta-analysis of eight randomised trials in 140 participants found low-to-moderate-certainty evidence that a single session of PBM reduces exercise-induced lipid damage (standardised mean difference −0.72 up to 72 hours) and protein damage (−0.41 up to 96 hours) and increases the activity of the antioxidant enzyme superoxide dismutase (0.54 at 48 and 96 hours), although it did not increase catalase activity and produced no clear change in total antioxidant capacity or glutathione peroxidase [7]. This selective modulation of the redox balance — reducing oxidative damage and boosting a key antioxidant enzyme — provides the most direct clinical confirmation of the cellular principle that PBM lowers ROS and restores mitochondrial potential in oxidatively stressed cells, and it offers a plausible final common pathway for the recovery benefits reported elsewhere [7] [1]. It is consistent, too, with the comparative CrossFit trial, which attributed the superiority of PBMT-sMF over compression and shock-wave therapies specifically to the modulation of oxidative stress and enhancement of antioxidant defences [23]. That the oxidative-stress evidence is among the most consistent in the field, while the functional-performance evidence is among the least, suggests that PBM reliably engages its biochemical mechanism even where the downstream functional payoff is variable and parameter-dependent [7] [29].

The oxidative-stress meta-analysis is methodologically the most secure piece of recovery evidence and warrants a closer look at its certainty and its gaps. Drawing on a broad search of PubMed, EMBASE, CINAHL, CENTRAL, PEDro and the Virtual Health Library, it included eight randomised trials of moderate-to-excellent methodological quality in 140 healthy participants and graded the certainty of evidence with the GRADE system [7]. Its positive findings — a reduction in lipid peroxidation persisting to 72 hours, a reduction in protein carbonylation to 96 hours, and an increase in superoxide dismutase activity at 48 and 96 hours — were of low-to-moderate certainty, and equally important were its null findings: PBM did not increase catalase activity and produced no detectable change in total antioxidant capacity or glutathione peroxidase, indicating a selective rather than global effect on the antioxidant system [7]. This selectivity is mechanistically congruent with the cellular account, in which PBM restores the mitochondrial membrane potential of oxidatively stressed cells and thereby lowers the rate of ROS generation, allowing endogenous superoxide dismutase to gain the upper hand, rather than acting as a blanket antioxidant [7] [1]. The authors concluded that a single session of PBM can modulate redox metabolism, which is a more defensible claim than any assertion about performance, and it situates the modality as a genuine biochemical intervention whose redox effect is reproducible even where its functional translation is not [7] [23]. The disjunction between a reliable biochemical signature and an unreliable functional outcome is the single most important interpretive fact about the recovery literature, and it suggests that future trials should treat oxidative-stress modulation as the proximate effect and ask, separately and explicitly, under what conditions that effect yields a meaningful functional or perceptual benefit [7] [30].

6.4. Comparison with established recovery modalities

A practically important question is whether PBM offers any advantage over the recovery methods already used by athletes, and the answer is equivocal. The LED trial after sprint-interval training found PBM equivalent to — but not better than — cold-water immersion and active recovery on all markers, implying that it is at best a non-inferior alternative rather than a superior one [26]. By contrast, the comparative CrossFit trial found PBMT-sMF superior to intermittent pneumatic compression, extracorporeal shock wave therapy and passive recovery for muscle-damage and oxidative-stress outcomes at 24 and 48 hours, though notably the device-based comparators (compression and shock wave) achieved higher participant satisfaction despite their inferior biochemical effects, a reminder that perceived and measured benefits can diverge [23]. Reviews of PBM as sports medicine echo this nuance: PBM delivered pre-exercise shows beneficial effects on exertion recovery, muscle strength, endurance and fatigue, but the evidence is less convincing for acute deep-tissue injury, where most studies do not demonstrate superiority over conventional modalities, and the field is hampered by wide variability in delivery parameters and a lack of clear treatment guidelines [4]. The reasonable synthesis is that PBM is a credible addition to the recovery toolkit whose marginal advantage over existing methods is unproven and probably context-specific, contingent on correct dosing and on the metabolic nature of the exercise [26] [4].

The clearest statement of the field's overall uncertainty comes from the meta-analysis of running performance, which is worth detailing because it directly tests the headline ergogenic claim. Pooling twelve randomised controlled trials with nineteen outcomes for time-trial and time-to-exhaustion running, the analysis found no significant overall effect of PBM (standardised mean difference 0.13, p = 0.11), no effect whether or not PBM was combined with a training programme, and — through meta-regression — no dose-response relationship across the irradiation doses tested (p = 0.82), with most studies clustered between 600 and 1000 J and only a handful exceeding 1000 J [28]. The authors emphasised two qualifications that capture the field's predicament: first, almost all the included studies showed a small, non-significant effect in favour of PBM, a trivial advantage that could nonetheless be decisive in a competitive context where margins are minute; and second, because a biphasic response is plausible and few studies used doses above 1000 J, the possibility remains that an effective dose simply had not been adequately tested, so that the null result reflects the limits of the existing dosing rather than a definitive absence of effect [28]. This careful agnosticism — no proven effect, but a hint of benefit and an untested high-dose region — is more honest than either uncritical enthusiasm or outright dismissal, and it reinforces that the running-performance question, like the recovery question, cannot be settled until dose-response is properly mapped [28] [8]. The divergence between participant satisfaction and physiological effect noted in the comparative recovery trial adds a final, human qualification: athletes may prefer modalities that feel more active or sensory, so that the adoption of PBM will depend not only on its measured efficacy but on managing the expectancy and perceptual factors that shape how a recovery intervention is experienced [23] [28].

Table 3. Summary of meta-analytic evidence on photobiomodulation for recovery outcomes.

Outcome	**Direction of effect**	**Magnitude / certainty**
DOMS (visual analogue, 72–96 h)	Reduced	Moderate effect (SMD ≈ −0.55)
Muscle strength recovery (24–48 h)	Improved	Large effect (SMD ≈ 0.97–0.99)
Lipid oxidative damage	Reduced	SMD −0.72; low–moderate certainty
Protein oxidative damage	Reduced	SMD −0.41; low–moderate certainty
Superoxide dismutase activity	Increased	SMD 0.54
Catalase / total antioxidant capacity / GPx	No clear change	Non-significant

Outcome	Direction of effect	Magnitude / certainty
Running performance (time-trial, time-to-exhaustion)	No significant effect	Small, non-significant

Sources: [29] [7] [28].

7. Delivery Parameters and Dosimetry

The recurrent explanation for the field's inconsistency is dosimetry, and the parameters governing PBM deserve explicit treatment because they determine whether a given application falls within or outside the effective window. The variables that must be controlled include wavelength, energy density (fluence), power density (irradiance), total energy, pulse structure, spot size, treatment duration, the number and location of irradiated points, and the delivery mode (contact or non-contact, laser or LED) [8] [1]. Recommendations diverge widely, with some groups advocating a power density below about 100 mW/cm^2 and an energy density of 4–10 J/cm^2 at the target tissue and others using far higher surface doses, and this latitude has produced contradictory results across the more than a thousand studies in the literature [8]. The biphasic principle means that effective and ineffective doses are separated by a relatively narrow margin, and the appraisal of high- versus low-mitochondrial tissues indicates that muscle, being mitochondria-rich, responds to lower doses and is vulnerable to over-dosing, so that many null trials in athletes may reflect excessive rather than insufficient energy [8] [13].

Several specific delivery questions have been addressed directly. The laser-versus-LED distinction is not trivial: although both can be effective and many comparisons find no fundamental difference at matched wavelength and power, the source-specific study in myoblasts showed that LED and near-infrared laser drive different downstream outputs even when the mitochondrial signal is conserved, and regulatory bodies classify LEDs differently from laser diodes, so device choice can influence both the biological effect and the standardisation of treatment [13] [4]. The locality of the effect is established — PBM combined with a static magnetic field improved muscle performance only when applied to the exercised muscle and not to a distant muscle, confirming that the interaction is a local tissue phenomenon requiring irradiation of the working muscle and adequate per-point illumination time [20]. The timing of application matters for the intended goal, with pre-exercise delivery favoured for performance enhancement and protection, while post-exercise delivery is used for recovery, and the doubling of applications (before and after) can tip the dose into the inhibitory range, as the CrossFit trial suggested [19] [4]. Finally, delivery modality ranges from targeted hand-held probes through flexible LED arrays to whole-body light beds and transcranial devices; whole-body and transcranial approaches, though attractive for convenience and for central effects, face the difficulty of delivering an adequate and correctly targeted dose, which may explain the null whole-body and transcranial trials [27] [25]. The unifying lesson is that PBM is not a single intervention but a family of interventions whose

efficacy is inseparable from its parameters, and that the absence of standardised, tissue-specific dosing guidelines is the principal obstacle to both reproducible research and confident clinical use [8] [4].

Practical efforts to impose order on this complexity provide some guidance even in the absence of a unified standard. The World Association for Photobiomodulation Therapy publishes dose and administration recommendations for Class IIIb laser use, for example in chronic arthritic and inflammatory conditions using 780–860 nm or pulsed 904 nm sources, and clinical reviews translate the parameter literature into rough application templates — pre-conditioning doses of roughly 2–6 J/cm^2 per muscle delivered immediately before exercise, post-exercise recovery doses of about 4–10 J/cm^2 within two hours, and higher fluences of 10–20 J/cm^2 for tendon and ligament injury — while stressing that these remain approximations rather than validated prescriptions [4] [3]. Device-level considerations compound the dosing question: the United States Food and Drug Administration classifies LEDs differently from laser diodes and exempts them from the laser product performance standards, requires a 510(k) clearance for new laser devices, and not all marketed products meet these standards, so that nominally similar devices may differ materially in their output and regulatory pedigree [4]. The mode of delivery introduces further variables — direct skin contact with light pressure minimises reflection and maximises penetration for deep tissue, whereas full-body LED panels held at a distance are better suited to general recovery and circadian modulation; continuous-wave output is standard, but pulsing at specific frequencies is being explored for particular effects; and because a device with higher irradiance reaches the target fluence in less time, the clinician must verify the actual power output rather than rely on the stated treatment duration [3] [4]. These practical frameworks do not resolve the underlying problem — the lack of a validated, tissue-specific dose for skeletal muscle in athletes — but they at least make explicit the parameters that any reproducible protocol must specify, and they convert the abstract injunction to "control the dose" into a checklist that research and practice can follow [8] [3].

The choice of delivery modality deserves separate consideration, because it interacts with dosimetry in ways that may explain several of the field's failures. Targeted hand-held probes and small cluster applicators deliver a high, controllable fluence to a defined muscle region in direct skin contact, which is the configuration in which most positive trials were conducted and which respects the demonstrated locality of the muscle effect [20] [4]. Flexible LED arrays extend the irradiated area while retaining contact and reasonable fluence, but whole-body light beds and transcranial devices face a structural difficulty: distributing a fixed power output over a large surface, or attempting to penetrate the scalp and skull to the cortex, dilutes the dose reaching any given target, so that an apparently generous total energy may translate into a sub-therapeutic local fluence [27] [25]. This dilution offers a parsimonious explanation for the null whole-body resistance-training trial and the null transcranial cycling trial, in which the convenience of broad or central delivery may have been bought at the cost of an inadequate dose at the tissue that mattered [27] [25]. The tension is genuine, because

the most appealing clinical scenario — a single whole-body exposure that aids recovery of all muscle groups at once — is precisely the one in which dose adequacy is hardest to guarantee, whereas the configuration that most reliably works, point-by-point irradiation of each working muscle, is the least convenient [20] [27]. Resolving this tension is not merely a matter of preference but a research priority: systemic delivery modes must be validated by demonstrating that a sufficient dose reaches the intended deep tissue before their attractive convenience is allowed to substitute for efficacy, and until such validation exists, the safest interpretation of a null systemic trial is under-dosing at the target rather than inefficacy of the modality [27] [8].

8. Injury Rehabilitation and the Broader Clinical Context

The clinical reach of PBM beyond the gymnasium is relevant to athletes precisely because an athletic career is punctuated by the injuries, degenerative conditions and psychological obstacles that the broader literature addresses, and the same mitochondrial mechanism that underlies the performance and recovery claims also underlies these wider applications. An athlete is, over time, a patient — with tendon and ligament injuries, with the cartilage wear of repetitive loading, with skin wounds and abrasions, and with the fear and loss of confidence that accompany serious injury — and the value of a single modality that can be brought to bear across this whole range is part of what makes PBM attractive in sport [4] [32]. Reviewing this broader evidence also serves a critical purpose: because the clinical conditions have been studied more extensively and over longer periods than the sports applications, they reveal the modality's true pattern of strengths and weaknesses more clearly, and that pattern — reliable benefit for pain and superficial healing, weaker and less certain benefit for deep structural-functional outcomes, and a pervasive dependence on dose — recurs without exception across tendon, joint, skin and central-nervous-system applications [4] [35]. The broader context therefore does double duty, both widening the case for PBM in the athletic population and disciplining the interpretation of the sports-specific trials by showing that their inconsistencies are not peculiar to sport but are the universal signature of a parameter-sensitive modality [4] [33].

Although the focus of this review is performance and recovery, the value of PBM to athletes extends to the rehabilitation of injuries and to the wider clinical conditions that affect them, and this broader context both supports and qualifies the sports applications. In the management of soft-tissue injury, a double-blind randomised controlled trial of PBM combined with conservative treatment for acute Achilles tendon rupture found that, although function as measured by the Achilles Tendon Rupture Score did not differ from sham, pain during walking was significantly lower in the PBM group at 12 and 16 weeks — a circumscribed but clinically relevant analgesic benefit consistent with the general finding that PBM helps with pain and superficial healing more reliably than with deep functional outcomes [32] [4]. The same pattern appears in osteoarthritis, a condition relevant to ageing and to load-bearing athletes: high-energy PBM combined with rehabilitation exercise is being formally tested for sustained, potentially disease-modifying effects on pain, physical function and radiographic progression in knee

osteoarthritis, reflecting the migration of PBM from short-term symptomatic relief toward structural endpoints, though such trials await results [33]. Reviews of PBM as sports medicine confirm that the modality holds promise for accelerating wound healing and for pre-exercise performance and recovery but is least convincing for acute deep-tissue injury, where it rarely outperforms conventional therapy — a consistent boundary on its clinical reach [4].

The Achilles and knee studies illustrate this boundary with quantitative precision. In the Achilles trial, thirty-four men with acute conservatively treated rupture were randomised to PBM or sham during an eight-week immobilisation followed by a structured rehabilitation programme, and although the primary functional outcome did not differ, pain during walking was significantly lower in the PBM group with a moderate effect size of about 0.56 at both twelve and sixteen weeks — a dissociation between a genuine analgesic effect and an absent structural-functional effect that recurs throughout the deep-tissue literature and that the authors linked to the proposed action of light on collagen synthesis and fibre realignment, demonstrated in animal tendon models but not translated to human function here [32]. The knee-osteoarthritis protocol takes the field a step further toward structural endpoints: fifty patients with mild-to-moderate disease are randomised to high-energy PBM (a 1064 nm device delivering a total of about 3190 J per session in combined pulsed and continuous modes) plus rehabilitation exercise or to placebo plus exercise over eight weeks, with the Knee Injury and Osteoarthritis Outcome Score as the primary endpoint and radiographic morphology assessed at a three-month follow-up to test for disease modification rather than mere symptom relief [33]. That such a trial is being mounted reflects both the growing ambition of the field and its current evidential gap, since the disease-modifying claim remains unproven; for the athlete, the practical message of these rehabilitation studies is that PBM can be expected to help with pain and superficial healing more dependably than with the restoration of deep structural function, and that it is best positioned as an adjunct to, rather than a replacement for, exercise-based rehabilitation [32] [33].

The systemic and dermatological applications of PBM further illustrate the breadth of the mechanism and its safety profile. LED phototherapy of the skin, through the same cytochrome c oxidase, ATP and ROS pathways, stimulates fibroblast collagen synthesis and modulates inflammation, with red light improving skin rejuvenation and wound healing and the modality demonstrating an excellent safety record with only mild, transient adverse effects such as erythema — relevant to athletes both for the management of skin injuries and for the recovery and circadian benefits attributed to systemic light exposure [34] [3]. A particularly instructive crossover into the psychological domain comes from a triple-blind trial of whole-body PBM in fibromyalgia, which over six months produced significant improvements in pain, quality of life, leisure-time physical activity, kinesiophobia and self-efficacy, with the benefits attributed to the stimulation of mitochondrial metabolism and the modulation of central and autonomic function [35]. While fibromyalgia is not a sports condition, the reduction of

kinesiophobia (fear of movement) and the increase in physical activity and self-efficacy are directly transferable to the psychological barriers that limit return to activity after injury, and they demonstrate that the mitochondrial action of PBM can have consequences that reach from the cell to behaviour [35] [3]. These broader applications reinforce the coherence of the mechanism while confirming the same caveats — parameter dependence, stronger effects on pain and superficial tissue than on deep function — that constrain the sports literature [4] [34].

The dermatological evidence is quantitatively the most mature and shows the same mitochondrial mechanism producing measurable structural change in superficial tissue. Red light at 630–700 nm penetrates to the dermis, where absorption by cytochrome c oxidase raises ATP and activates fibroblasts to synthesise collagen and elastin, with clinical studies reporting a 15–30% increase in collagen after four to eight weeks and a 30–50% improvement in wrinkle severity after twelve weeks of treatment, alongside improved skin hydration and barrier function, and an excellent safety profile with no evidence of DNA damage and only rare, transient erythema [34]. Blue light at 405–420 nm acts more superficially against the porphyrin-producing bacteria implicated in acne, while near-infrared light accelerates wound healing and reduces erythema — a wavelength-dependent specificity that mirrors the chromophore plurality described earlier and reinforces that the spectral choice selects the biological target [34] [2]. The fibromyalgia trial extends the same logic to the central nervous system and behaviour: forty-two patients received whole-body PBM from a light bed (combining 660 and 850 nm) in twelve sessions over four weeks, with assessments to six months, and the active group showed significant and durable improvements in pain, health-related quality of life, leisure-time physical activity, kinesiophobia and self-efficacy, with pain catastrophising improving by the six-month follow-up; the authors proposed mitochondrial stimulation, modulation of the autonomic nervous system and even effects on the gut microbiome as candidate mechanisms [35]. Although fibromyalgia lies outside sport, the demonstrated reduction of kinesiophobia and the increases in physical activity and self-efficacy speak directly to the psychological obstacles that delay an athlete's return to training after injury, and they show that a peripheral, mitochondrial intervention can produce behavioural and affective change — a reminder that the benefits of PBM may extend beyond the strictly physiological to the domain of confidence and willingness to move [35] [3].

9. Safety, Regulation and Anti-Doping

The safety profile of PBM is one of its principal attractions and is well supported across the literature. Because the modality is non-ionising and non-thermal, operating at intensities that produce photochemical rather than heating effects, adverse events are rare and typically limited to mild, transient cutaneous reactions such as erythema, dryness or pruritus, with no evidence of DNA damage at therapeutic doses [34] [2]. Reviews of PBM in skin and in sports medicine consistently report excellent tolerability, and the dermatological literature notes the absence of significant adverse events in clinical trials [34] [4]. The chief safety consideration is, paradoxically, the same biphasic

principle that governs efficacy: because excessive doses can be inhibitory and, in some contexts, may transiently increase markers such as inducible nitric oxide synthase, attention to dosimetry is a matter of safety as well as effectiveness, though the consequences of over-dosing are generally a loss of benefit rather than harm [1] [8]. Practical clinical implementation also requires eye protection when high-powered lasers or panels are used near the face, and awareness that not all marketed devices meet regulatory standards, since LEDs are classified differently from laser diodes and device clearance pathways vary [3] [4].

From the standpoint of competitive sport, PBM occupies a distinctive regulatory position. Because the intensity of therapeutic light is comparable to sunlight and there is no forensic test to detect light exposure, the International Olympic Committee and the World Anti-Doping Agency cannot ban light therapy for athletes, which removes a barrier that constrains many pharmacological ergogenic aids and helps explain the rapid adoption of PBM in elite sport [1]. This regulatory freedom, combined with the modality's safety, makes PBM an appealing option even where its ergogenic efficacy is uncertain, but it also places a premium on honest appraisal: the very ease and acceptability of the treatment risk its uncritical use at unvalidated doses, and the absence of standardised protocols means that athletes and practitioners may apply parameters outside the effective window [1] [8]. Responsible use therefore depends less on safety concerns — which are minimal — than on the discipline of evidence-based dosimetry and realistic expectations of benefit [4] [8].

Even so, a small number of specific contraindications and precautions deserve explicit mention, because the very benignity of PBM can foster careless application. The proliferative and angiogenic effects that make PBM useful for tissue repair create a theoretical concern about applying it directly over a known malignancy, since stimulation of cell proliferation and blood-vessel formation could in principle favour tumour growth, so direct irradiation over a primary tumour or metastasis is generally avoided [3]. There is a lack of safety data for light applied over the pregnant uterus, although treatment of peripheral sites such as an ankle is considered safe; patients with photosensitive epilepsy should avoid pulsed devices in the seizure-provoking frequency range; those with hyperthyroidism should avoid neck application because PBM can acutely increase thyroid hormone output; and patients taking photosensitising medications should be monitored for exaggerated skin responses [3]. The side effects that do occur are mild and self-limiting — transient fatigue or headache after transcranial or full-body treatment, a temporary increase in pain or "healing crisis" after the first session in chronic conditions, localised erythema from vasodilation, and, if used late at night, possible interference with sleep through suppression of melatonin by bright light [3]. None of these alters the fundamental conclusion that PBM is among the safest interventions in sports medicine, but together they define the boundaries of responsible use and reinforce that the principal discipline the modality demands is not caution about harm but rigour about dose [3] [33].

For the practitioner and the athlete, these considerations translate into a small set of practical principles that follow from the evidence reviewed. The intended goal should dictate the timing: pre-exercise application is appropriate when the aim is performance enhancement or protection against impending damage, post-exercise application when the aim is recovery, and the temptation to apply at both times should be resisted unless the total dose has been accounted for, since doubling applications can push a mitochondria-rich muscle past the peak of its dose-response curve [19] [3]. The working muscle — and all the muscles involved in the activity — must be irradiated directly and in contact, with adequate per-point illumination time, because the effect is local and does not propagate from distant or systemic exposure [20]. The device's actual output should be verified rather than assumed, since irradiance varies between products and a higher-irradiance device reaches the target fluence in less time; and because LEDs and lasers are regulated and may behave differently, the spectral and power characteristics of the chosen device should be known [4] [13]. Eye protection is warranted with high-powered sources near the face, and the few genuine contraindications — irradiation over malignancy, over the pregnant uterus, of the thyroid in hyperthyroidism, and pulsed light in photosensitive epilepsy — should be respected [3]. Finally, expectations should be calibrated to the evidence: the athlete should be advised that PBM reliably modulates oxidative stress and may modestly reduce soreness and aid endurance recovery, but that its effect on explosive performance and its superiority over established recovery methods are unproven, so that it is best regarded as a safe, low-cost adjunct rather than a decisive ergogenic aid [7] [4]. This disciplined, dose-conscious and expectation-managed use is the most defensible way to deploy a modality whose mechanism is strong, whose safety is excellent and whose clinical delivery remains uncertain [8] [3].

10. Critical Appraisal, Limitations and Future Directions

The central tension of the PBM literature is the coexistence of a coherent, well-supported mitochondrial mechanism with an inconsistent clinical evidence base, and a critical appraisal must hold both in view. The mechanism is not in serious doubt: photon absorption by cytochrome c oxidase, the dissociation of inhibitory nitric oxide, the rise in ATP and the controlled ROS burst, and the downstream signalling that modulates oxidative stress, inflammation and myogenic gene expression are demonstrated from the single cell to living human tissue and across multiple cell types [5] [1] [12] [14]. What is in doubt is the reliability of the clinical payoff, and the appraisal of the trials points overwhelmingly to dosimetry as the explanation: the biphasic dose response, the tissue-specific optimal fluence, the laser-versus-LED distinction, the necessity of irradiating the working muscle, and the influence of timing all mean that two trials with nominally similar interventions can reach opposite conclusions [8] [13] [20]. The meta-analyses crystallise this duality: PBM modestly but reliably reduces DOMS and oxidative damage and increases superoxide dismutase activity, yet shows no significant effect on running performance, and the functional benefits are smaller and less consistent than the biochemical ones [29] [7] [28].

Several specific limitations constrain the field. Trials are frequently small, increasing the risk that true small effects are missed or that chance findings are over-interpreted, and the heterogeneity of parameters precludes meaningful pooling and obscures dose-response relationships [8] [9]. Important moderators — sex, training status, the muscle examined, and the metabolic versus neurological nature of the exercise — are inconsistently reported and rarely analysed, even though they plainly affect the outcome, as the divergence between endurance and explosive tasks and between trained and untrained participants demonstrates [9] [30]. The distinction between mechanistic plausibility and clinical proof is sometimes blurred, with cellular and animal findings — many of them in ageing or disease models rather than in healthy athletes — invoked to support sports claims that the human trials only partly bear out [11] [17]. And the perceptual dimension is under-studied: the finding that device-based comparators achieved higher satisfaction than the biochemically superior PBM warns that athlete preference and expectancy may diverge from physiological effect [23].

A further, subtler limitation is the asymmetry between the depth of the mechanistic evidence and the population in which much of it was obtained. The most compelling demonstrations that PBM up-regulates cytochrome c oxidase, restores mitochondrial membrane potential, drives myoblast differentiation and reorganises the neuromuscular junction come from isolated cells, from rodent brains and muscle, and frequently from ageing or disease models — diabetic mice, aged rats, oxidatively stressed myoblasts — rather than from the young, healthy, trained humans to whom the sports claims are addressed [11] [17] [16]. This does not invalidate the mechanism, which is highly conserved, but it means that the inferential leap from a reversed deficit in an aged or diseased system to an enhanced function in an already-optimised athletic system is larger than it is often acknowledged to be, and it may help explain why the trained-versus-untrained gradient is so consistent: a tissue already near its functional ceiling has less to gain from a bioenergetic nudge than a tissue impaired by age, disease or deconditioning [16] [30]. The biphasic principle compounds this asymmetry, since the optimal dose for a low-baseline, high-need tissue may overshoot the narrower effective window of a high-baseline, low-need one, exactly as the chronic-laser brain study showed opposite effects in young and aged animals [11] [8]. Recognising this asymmetry is essential to interpreting the literature honestly: the cellular and animal evidence establishes that the mechanism is real and powerful, but it does not establish that the mechanism produces a large or reliable benefit in healthy athletes, and conflating the two has been a persistent source of over-claiming in the field [11] [28].

The priorities for future research follow directly. The most pressing need is for standardised, tissue-specific dosimetry: trials must define and justify the dose delivered to the target muscle, report all parameters fully, and ideally test dose-response within the same study so that the effective window for skeletal muscle can be established rather than guessed [8] [4]. Trials should be adequately powered, should pre-specify and analyse the key moderators of sex, training status, muscle and exercise type, and should use sham-controlled, blinded designs with both functional and biochemical endpoints so

that the relationship between the reliable mechanism and the variable function can be clarified [9] [7]. The laser-versus-LED and local-versus-systemic questions deserve dedicated, head-to-head study, given the demonstrated source-specificity of the cellular response and the demonstrated locality of the muscle effect [13] [20]. Longer-term and disease-modifying endpoints, as in the knee-osteoarthritis protocol, would extend the evidence beyond short-term surrogates [33]. Until these gaps are addressed, PBM will remain a modality of strong mechanistic promise and uncertain, parameter-dependent clinical delivery [8] [28].

Beyond the design of individual trials, the field needs an infrastructural commitment to standardisation that it has so far lacked. The most immediate requirement is the complete and uniform reporting of irradiation parameters — wavelength, power and energy density, total energy, spot size, number and location of points, pulse structure, contact mode and the resulting dose at the target tissue — so that studies can be compared and pooled and so that meta-regression can finally map the dose-response relationship that the running-performance meta-analysis could not [28] [8]. Allied to this is the need for agreed core outcome sets that pair functional endpoints (performance, strength, jump, time to exhaustion) with the biochemical and oxidative-stress markers that more reliably reflect the mechanism, so that the relationship between the dependable biochemical signal and the variable functional response can be studied systematically rather than incidentally [7] [29]. The laser-versus-LED question should be resolved by head-to-head trials at matched fluence, given the demonstrated source-specificity of the cellular response, and the systemic delivery modes — whole-body beds and transcranial devices — require explicit validation that an adequate dose reaches the intended target before their convenience is allowed to drive adoption [13] [27]. Finally, the migration of the field toward longer-term and disease-modifying endpoints, exemplified by the knee-osteoarthritis protocol, should be encouraged, because the dominance of short-term surrogate outcomes has left the durability and clinical significance of PBM's effects largely untested [33] [4]. None of these requirements is technically difficult; their persistent neglect, rather than any deficiency in the underlying biology, is what keeps PBM a modality of strong mechanism and weak proof [8] [4].

11. Conclusions

Photobiomodulation occupies an instructive position in sports medicine: a non-invasive, safe and anti-doping-compliant modality with a coherent and well-documented mechanism, yet an inconsistent clinical record. The mechanism is firmly established — red and near-infrared light is absorbed by cytochrome c oxidase, dissociating inhibitory nitric oxide and raising mitochondrial membrane potential, ATP and a controlled, signalling burst of reactive oxygen species, with downstream effects on nitric oxide, calcium and transcription factors that promote myoblast differentiation, mitochondrial biogenesis, anti-inflammatory action and the modulation of oxidative stress, all under the governance of a biphasic dose response [5] [1] [14] [13]. This mitochondrial action

provides a genuine rationale for the use of PBM to support the energy provision, repair and redox balance of the exercising athlete [17] [7].

In application, the evidence is real but qualified. Pre-exercise PBM has improved peak oxygen uptake, time to exhaustion and, in some trials, strength, and combined photobiomodulation-and-static-magnetic-field protocols have enhanced recovery and modulated oxidative stress; meta-analyses confirm modest reductions in delayed-onset muscle soreness and oxidative damage and improvements in strength recovery [6] [19] [29] [7]. Yet numerous rigorous trials have found no benefit on performance, muscle damage or soreness, no advantage over established recovery methods, and no significant effect on running performance, with the divergence attributable to dose, wavelength, timing, the muscle irradiated, training status and sex rather than to any simple efficacy or inefficacy [10] [9] [26] [28] [8]. The balanced conclusion is one of cautious, mechanism-anchored optimism: photobiomodulation is a credible, safe and underexploited tool whose biochemical effects are more reliable than its functional ones, and whose clinical promise will be realised only when standardised, tissue-specific dosimetry and adequately powered, well-moderated trials convert a strong mechanism into reproducible benefit for athletes [8] [4] [31].

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