Issue Rehabil. Orthop. Neurophysiol. Sport Promot. 2026; 54: 17–26. ISSN 2300-0767

DOI: 10.5114/IRONS/218480

REVIEW ARTICLE

EXERCISE-BASED STRATEGIES TO ENHANCE BDNF AND COGNITIVE RECOVERY AFTER STROKE: A CLINICAL REVIEW

STRATEGIE OPARTE NA ĆWICZENIACH FIZYCZNYCH W CELU ZWIĘKSZENIA STĘŻENIA BDNF I POPRAWY FUNKCJI POZNAWCZYCH PO UDARZE MÓZGU: PRZEGLĄD KLINICZNY

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ABSTRACT

Introduction

Stroke is one of the leading causes of death and long-term disability worldwide, with a growing number of survivors living with cognitive impairment. Aerobic exercise is recognised as a promising non-pharmacological intervention supporting neuroplasticity and functional recovery after stroke. Brain-derived neurotrophic factor (BDNF) plays a key role in activity-dependent plasticity and has been proposed as a potential biomarker of rehabilitation success; however, clinical evidence regarding exercise-induced changes in BDNF remains inconsistent.

Aim

This clinical narrative review summarises clinical studies evaluating the effects of structured aerobic training on circulating BDNF levels and cognitive outcomes in stroke survivors.

Materials and methods

A literature search identified five eligible clinical trials.

Results

Across these studies, aerobic exercise interventions were associated with cognitive or functional improvements; however, heterogeneity in exercise-intensity prescription precluded identification of a single optimal training intensity. In contrast, high-intensity exercise protocols were linked to increases in circulating BDNF concentrations but did not demonstrate clearly superior cognitive outcomes compared with moderate-intensity training. Chronic BDNF responses to aerobic training were heterogeneous. One study reported an increase in resting BDNF levels, two studies observed no significant change, one study reported a decrease, and one study demonstrated BDNF increases only following high-intensity interval training. In most trials, changes in circulating BDNF were not associated with improvements in cognitive or clinical outcomes.

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Authors reported no source of funding
Authors declared no conflict of interest

Date received: 24th January 2026
Date accepted: 23rd February 2026

A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation, D – Writing the article, E – Critical revision of the article, F – Final approval of the article

Conclusions
These findings indicate that aerobic exercise is an effective component of post-stroke rehabilitation aimed at improving cognitive function. However, resting circulating BDNF shows substantial variability and does not consistently reflect cognitive recovery, suggesting that BDNF alone is not a reliable long-term biomarker of exercise-induced cognitive improvement after stroke. Aerobic training remains a practical and clinically beneficial strategy, although the optimal training intensity and dose remain uncertain.

Keywords: neuroplasticity, BDNF, aerobic training, stroke survivors, cognitive function

Introduction

Stroke remains the second leading cause of death and the third leading cause of death and disability combined worldwide [1]. Global estimates from the World Stroke Organisation indicate that from 1990 to 2021 incident strokes increased by approximately 70%, stroke-related deaths by 44%, and the prevalence of stroke survivors by 86%, with the majority of deaths and disability-adjusted life-years occurring in low- and middle-income countries [1]. Advances in acute care have reduced early mortality; however, this has led to a growing population of stroke survivors living with long-term motor and cognitive impairments, which substantially reduce independence and quality of life.

Cognitive impairment is highly prevalent after stroke, affecting domains such as attention, executive function, memory, and processing speed [2]. Post-stroke cognitive deficits are associated with poorer functional outcomes, an increased risk of institutionalisation, and higher mortality [2]. As pharmacological options for cognitive recovery remain limited, clinical guidelines increasingly emphasise the role of non-pharmacological strategies, particularly structured exercise, in secondary prevention and rehabilitation [2].

From a neurobiological perspective, the brain retains a remarkable capacity for structural and functional reorganisation – neuroplasticity – throughout life. Physical exercise is one of the most potent physiological stimuli of neuroplastic mechanisms [3]. Experimental and human studies have shown that aerobic exercise can promote neurogenesis, synaptogenesis, and angiogenesis, increase cerebral blood flow, and enhance functional connectivity within cognitive networks [3]. These changes are mediated, at least in part, by the activity-dependent release of neurotrophins, among which brain-derived neurotrophic factor (BDNF) plays a central role.

BDNF supports neuronal survival, dendritic growth, and long-term potentiation, and is critically involved in learning and memory. In animal models, both acute and chronic aerobic exercise robustly increase BDNF expression in the hippocampus and cerebral cortex, accompanied by improved cognitive performance [3]. In humans, meta-analyses have shown that a single bout of exercise elevates peripheral BDNF concentrations in healthy adults in an intensity- and duration-dependent manner, and that repeated high-intensity training can produce chronic increases [4].

In stroke survivors, systematic reviews and meta-analyses indicate that exercise interventions improve both motor and cognitive outcomes [2]. A recent meta-analysis focusing specifically on BDNF in post-stroke populations found that high-intensity aerobic exercise can increase circulating BDNF concentrations, suggesting a potential link between exercise dose, neurotrophin response, and neuroplasticity [5]. However, individual clinical trials report mixed findings, with some demonstrating parallel improvements in cognition and BDNF, and others

showing cognitive benefits without measurable changes in resting BDNF levels [6].

Given the growing interest in BDNF as both a therapeutic target and a biomarker of recovery after stroke, it is important to clarify how exercise parameters such as intensity and duration relate to chronic BDNF levels and cognitive outcomes in clinical populations. Therefore, this review focuses on clinical trials that combined structured aerobic training with measurement of circulating BDNF and cognitive assessment in stroke survivors.

Aim

The aim of this review was to synthesise clinical studies evaluating how different aerobic training parameters, particularly exercise intensity and training duration, influence chronic circulating BDNF levels and cognitive outcomes in stroke survivors, and to critically appraise the suitability of resting BDNF concentration as a biomarker of rehabilitation success.

Materials and methods

Literature search and study selection

A structured literature search of PubMed, Scopus, and the Cochrane Library was conducted covering the period from 2013 to 2025. Search terms combined controlled vocabulary and free-text words related to stroke, exercise, and neurotrophins, including: stroke, ischaemic stroke, aerobic exercise, training, physical activity, cognition, cognitive impairment, BDNF, and rehabilitation. Reference lists of relevant systematic and narrative reviews were also screened to identify additional clinical trials.

Inclusion and exclusion criteria

Studies were eligible if they met the following criteria:

- adult participants with a history of ischaemic or haemorrhagic stroke;
- a clearly defined aerobic or predominantly aerobic training protocol (continuous or interval);
- measurement of circulating BDNF (serum or plasma) before and after a multi-session training programme (chronic response);
- assessment of cognitive outcomes (global cognition or specific domains) and/or validated functional measures with a cognitive component;
- clinical trial design (randomised or non-randomised), published in a peer-reviewed journal in English.

Exclusion criteria were as follows:

- animal or preclinical studies;
- studies assessing only acute BDNF responses to a single exercise bout without a multi-session intervention;
- interventions combining exercise with pharmacological agents specifically targeting BDNF (e.g. selective serotonin reuptake inhibitors), where the effects of exercise could not be isolated;
- narrative reviews, editorials, and case reports.

This work was planned as a clinical narrative review rather than a fully systematic review. Consequently, no formal Preferred Reporting Items for Systematic Reviews and Meta-Analyses flowchart, risk-of-bias assessment, or quantitative meta-analysis of effect sizes was performed. Instead, trials were analysed descriptively, with particular attention to exercise intensity, training duration, stroke phase, cognitive outcomes, and patterns of chronic BDNF change.

Results

Five clinical trials met the inclusion criteria. They varied in stroke phase (early subacute to chronic), sample size (n = 23–76), training modality (cycle ergometer, treadmill, mixed tasks), exercise intensity [low, moderate, high, and high-intensity interval training (HIIT)], and intervention length (3–18 weeks, typically 3–4 sessions per week). Cognitive assessment tools included the Addenbrooke's Cognitive Examination–Revised (ACE-R), Addenbrooke's Cognitive Examination, third

edition (ACE-III), Raven's Progressive Matrices, and broader clinical scales.

The ACE-R and ACE-III are brief, 100-point cognitive screening batteries assessing attention/orientation, memory, verbal fluency, language, and visuospatial abilities, and are widely validated for the detection of cognitive impairment in neurological populations [7,8]. Fluid intelligence was assessed using Raven's Progressive Matrices, a non-verbal multiple-choice test of abstract reasoning composed of matrix-completion items of increasing difficulty, commonly used as a measure of fluid intelligence [9]. Broader clinical scales included functional and motor assessments, such as the Barthel Index and the Rivermead Motor Assessment, which evaluate independence in activities of daily living and motor performance in stroke survivors [6].

The key features and outcomes of each study are summarised in Table 1, and below.

Characteristics of included studies

El-Tamawy et al. [10] – BDNF concentrations and cognitive function increased

In a randomised trial of 30 patients after ischaemic stroke, El-Tamawy *et al.* [10] compared standard physical therapy alone with physical therapy moderate-intensity aerobic training (bicycle ergometer exercise). The intervention group trained 3 times per week for 8 weeks.

Cognitive function, assessed using the ACE-R, improved significantly more in the aerobic training group than in the control group. Serum BDNF concentrations increased significantly after training in the aerobic group compared with both baseline values and the control group. Importantly, a moderate positive correlation was observed between changes in ACE-R scores and changes in BDNF concentrations (r = 0.53).

Table 1. Characteristics and key findings of included clinical trials

No.	Study	*n*	Stroke phase	Intensity	Duration	Cognitive outcome	BDNF outcome	BDNF – cognition relation
1	El-Tamawy *et al.* [10]	30	Chronic	NR	8 wks, 3 ×/wk	ACE-R ↑	↑ Significant	Positive correlation
2	Ploughman *et al.* [11]	52	Chronic	60–80% VO_2peak	10 wks, 3 ×/wk	Fluid intelligence ↑	↔ No change	No association
3	Hsu *et al.* [12]	23	Chronic	HIIT: 80/40% VO_2peak; MICT: 60% VO_2peak	12–18 wks, 2–3 ×/wk	Data unclear	↑ (HIIT)	No data
4	Górna *et al.* [6]	44	Early subacute	MICT: 70–80% VO_2max; LICT: 50–60% VO_2max	3 wks, 4 ×/wk	ACE-III ↑	↓ Significant	No association
5	De Las Heras *et al.* [13]	76	Early subacute	MICT: 65–80% PPO; HIIT: 85–100% PPO	8 wks, 3 ×/wk	Clinical ↑	↔ No change	No association

ACE-III – Addenbrooke's Cognitive Examination, third edition; ACE-R – Addenbrooke's Cognitive Examination–Revised; BDNF – brain-derived neurotrophic factor; HIIT – high-intensity interval training; LICT – low-intensity continuous training; MICT – moderate-intensity continuous training; NR – not reported; PPO – peak power output; VO_2max – maximal oxygen uptake; VO_2peak – peak oxygen uptake; ↑ = increase/improvement; ↓ = decrease; ↔ = no change; wks = weeks; ×/wk = times per week

This study provides the clearest evidence that, under specific conditions, moderate-intensity aerobic training can simultaneously enhance cognition and increase resting BDNF levels in stroke survivors.

Ploughman et al. [11] – cognitive function increased and was not significantly associated with BDNF response, but was significantly associated with insulin-like growth factor 1

Ploughman *et al.* [11] randomised 52 individuals with chronic stroke into four groups in a 10-week programme combining aerobic exercise or low-intensity activity with cognitive training or computer-based games. Aerobic training sessions were conducted 3 times per week at a target heart-rate zone corresponding to 60–80% peak oxygen uptake (VO_2peak).

The greatest improvements in fluid intelligence, assessed using Raven's matrices, were observed in the group receiving combined aerobic and cognitive training. However, resting the BDNF levels and acute BDNF responses to exercise were not significantly altered by the intervention. Instead, the magnitude of the insulin-like growth factor 1 (IGF-1) response to exercise at baseline explained a substantial proportion of the variance in cognitive improvement, suggesting a more prominent role for IGF-1 than BDNF in mediating training-related cognitive effects in this context.

This trial demonstrates robust cognitive improvements and highlights IGF-1 as a plausible alternative biomarker, but does not support a simple association between chronic BDNF levels and cognitive gains.

Hsu et al. [12] – HIIT significantly increased serum BDNF, but cognition data were limited

Hsu *et al.* [12] conducted a randomised controlled trial in 23 patients more than 2 years after stroke, comparing moderate-intensity continuous training (MICT, approximately 60% VO_2peak) with HIIT (intervals at approximately 80% VO_2peak alternated with low-intensity bouts). Both interventions lasted 12–18 weeks and were performed 2–3 times per week.

Serum BDNF concentrations increased significantly following HIIT, but to a greater extent than after MICT. HIIT also produced superior improvements in cardiorespiratory fitness and indicators of frontal cortical oxygenation. However, the published report provides limited detail on cognitive outcomes, and no explicit correlation between changes in BDNF concentrations and cognitive measures was reported.

This study supports meta-analytic evidence indicating that high-intensity aerobic exercise can increase circulating BDNF levels in stroke survivors, but does not demonstrate a clear cognitive advantage of HIIT over moderate-intensity exercise.

Górna et al. [6] – cognition increased, BDNF decreased, with no significant association between them

Górna *et al.* [6] randomised adults after a first ischaemic stroke in the regenerative–compensatory phase to either MICT or low-intensity continuous training (LICT), both combined with standard neurorehabilitation. The intervention lasted three weeks and consisted of four 45-minute cycling sessions per week in addition to daily rehabilitation.

Cognitive performance, assessed using ACE-III, improved to a greater extent in the MICT group than in the low-intensity group, although confidence intervals around between-group differences were wide. Unexpectedly, serum BDNF concentrations decreased significantly in the MICT group compared with the LICT group (between-group difference of approximately −2.2 ng/ml). No significant associations were observed between changes in BDNF concentrations or other neurotrophins and changes in cognitive, functional, or fitness outcomes.

This trial demonstrates that short-term moderate-intensity training can improve cognition even when chronic BDNF levels decrease, further challenging the concept of a straightforward positive relationship between BDNF and cognitive recovery after stroke.

De Las Heras et al. [13] – cardiorespiratory fitness improved, BDNF concentrations did not change significantly and were not associated with fitness outcomes

In one of the larger recent studies, De Las Heras *et al.* [13] investigated 76 patients in the early subacute phase (< 3 months) after a first ischaemic stroke. Participants were randomised to an 8-week cardiovascular exercise programme (initial continuous training followed by HIIT) plus standard care, or to standard care alone.

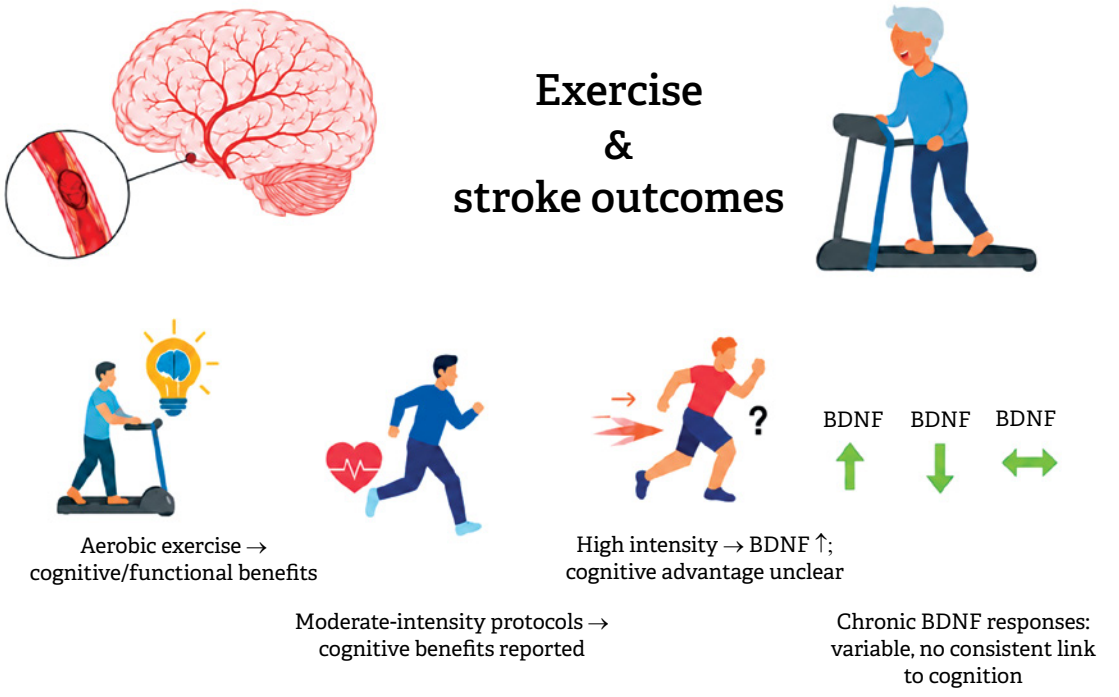
Cardiorespiratory fitness and selected clinical outcomes improved in the exercise group. However, neither chronic nor acute serum BDNF concentrations changed significantly over the 8-week intervention period, and changes in BDNF were not associated with changes in fitness or clinical outcomes.

This study indicates that even relatively intensive and prolonged exercise programmes in early subacute stroke may not necessarily alter resting BDNF concentrations, despite clear functional benefits.

Overall patterns

Taken together, these 5 trials demonstrate that:

- aerobic exercise consistently improves cognitive or functional outcomes after stroke [6,10–13];
- exercise intensity was prescribed using heterogeneous physiological parameters across studies, and the available evidence does not allow a single optimal training intensity to be identified;



↑ = *increase/improvement;* ↓ = *decrease;* ↔ = *no change*

Figure 1. How exercise intensity shapes cognitive recovery and brain-derived neurotrophic factor (BDNF) profiles in stroke survivors

- high-intensity training can increase circulating BDNF concentrations but does not appear to provide superior cognitive benefits [5];
- chronic BDNF responses to exercise are highly heterogeneous (increase, decrease, or no change) and frequently show no association with cognitive outcomes [6,10–13].

A synthesis of these patterns is presented in Figure 1.

Discussion

Aerobic exercise as a cognitive rehabilitation strategy

The consistent observation across heterogeneous trials that aerobic exercise improves cognitive or related functional outcomes aligns with broader evidence from mixed stroke populations and other neurological conditions. Systematic reviews report that exercise interventions, particularly those incorporating aerobic components, can enhance global cognition and executive function in stroke survivors with cognitive impairment [2].

Benefits were reported after both short and longer exercise programmes; however, the heterogeneity of the available studies precludes conclusions regarding an optimal intervention duration [6,10–13]. These findings support the practical recommendation that stroke rehabilitation programmes should include regular, supervised aerobic training as a core component to support cognitive recovery, provided that cardiovascular status and safety considerations are appropriately assessed.

Why does chronic BDNF behave so inconsistently?

Only one study (El-Tamawy *et al.* [10]) demonstrated that increases in resting BDNF corresponded with cognitive improvements and a positive correlation, whereas several other studies found that BDNF levels remained stable or decreased despite gains in cognitive or functional performance.

Several factors may explain this discrepancy:

1. **Peripheral versus central BDNF:** Peripheral BDNF is largely stored in platelets, and serum concentrations may reflect platelet activation as much as brain-derived secretion [4]. This phenomenon complicates the interpretation of resting serum BDNF as a marker of central neuroplasticity.
2. **Timing and matrix of sampling:** Differences in blood matrix (serum vs. plasma), timing relative to the last exercise session, circadian rhythm, and fasting state can all influence measured BDNF concentrations [4].
3. **Stroke phase and comorbidities:** Baseline BDNF levels are often reduced after stroke and may be modulated by depression, inflammation, and cardiometabolic comorbidities [14]. Consequently, training-related changes may be modest or obscured by underlying biological variability.
4. **Genetic variability:** The *BDNF Val66Met* polymorphism, examined by De Las Heras *et al.* [13], can affect activity-dependent secretion of BDNF and may contribute to inter-individual differences in responsiveness [14].

Meta-analytic evidence in non-stroke populations supports the notion that acute exercise reliably increases BDNF concentrations, particularly at higher intensities and longer durations, whereas chronic changes are smaller and more variable [4]. Ashcroft *et al.* [5] reported that, in stroke survivors, high-intensity aerobic training programmes can increase circulating BDNF on average; however, the effects are heterogeneous and highly dependent on the specific protocols used. Collectively, these data suggest that resting BDNF is a variable and context-dependent indicator. It likely reflects only a fraction of the complex biological processes underlying exercise-induced recovery and may be more informative as part of a broader biomarker panel than as a stand-alone measure.

Role of intensity: clinical and mechanistic considerations

High-intensity aerobic exercise appears to be a potent stimulus for acute BDNF release across different populations [5]. Hsu *et al.* [12] demonstrated that in chronic stroke patients, HIIT induces larger increases in serum BDNF concentrations than MICT and yields superior improvements in VO_2peak and cortical oxygenation. Similar patterns have been reported in other neurological conditions and in healthy adults.

Nevertheless, the trials reviewed here do not demonstrate that high-intensity protocols yield consistently superior cognitive outcomes compared with well-designed moderate-intensity programmes, particularly when sample sizes are limited and cognitive outcome measures vary. Moreover, high-intensity training may be less feasible or acceptable in patients with significant cardiovascular comorbidity, fatigue, or balance impairments.

Moderate-intensity aerobic training has been associated with cognitive benefits in some studies and may offer a practical balance between feasibility and tolerability. However, differences in the methods used to prescribe exercise intensity preclude defining a specific optimal intensity target based on the currently available evidence.

Beyond BDNF: towards multimarker and multidomain assessment

The observation by Ploughman *et al.* [11] that IGF-1 responses – but not BDNF – predicted improvements in fluid intelligence underscores the importance of considering alternative or complementary biomarkers. IGF-1 plays a crucial role in neurogenesis, synaptic plasticity, and angiogenesis and may interact with BDNF signalling pathways [14]. Vascular endothelial growth factor (VEGF) and other growth factors also contribute to vascular and structural adaptations induced by exercise [3].

Recent narrative reviews have argued that exercise-based stroke rehabilitation research should move towards multimarker and multidomain frameworks that integrate [14]:

- neurotrophins (BDNF, IGF-1, VEGF, glial cell line-derived neurotrophic factor);
- markers of inflammation and oxidative stress;
- neuroimaging metrics (structural and functional connectivity);
- detailed cognitive and functional assessments;
- patient-reported outcomes.

Such approaches may better capture the complexity of exercise-induced neuroplasticity and help identify more stable and clinically useful biomarker composites than any single analyte measured at rest.

Clinical implications

For clinicians involved in stroke rehabilitation, the key messages from this review are as follows:

- aerobic exercise should be considered a core component of cognitive rehabilitation after stroke and integrated with task-specific and cognitive training;
- moderate-intensity aerobic training may represent a practical option for appropriately selected patients; however, current evidence does not support a single optimal intensity or training dose for cognitive recovery after stroke;
- HIIT may be an option for selected, medically stable patients to enhance cardiorespiratory fitness and acutely increase BDNF concentrations; however, its added value for cognition over moderate-intensity exercise remains uncertain;
- routine measurement of resting BDNF concentrations is not currently justified for clinical decision-making, given their variability and the lack of consistent association with cognitive outcomes.

Limitations of the evidence base

The conclusions of this review are constrained by several limitations of the underlying literature:

- modest sample sizes in individual trials, reducing statistical power to detect moderate effects and interactions;
- considerable heterogeneity in cognitive outcome measures and test batteries, complicating direct comparisons;
- limited reporting of both neurotrophin and cognitive outcomes in several trials, with some studies focusing primarily on functional or fitness measures;
- heterogeneity in stroke phase, lesion characteristics, comorbidities, and rehabilitation contexts, which may mask or modify BDNF responses.

These limitations highlight the need for larger, adequately powered trials employing harmonised protocols and outcome measures.

Conclusions

1. **Aerobic exercise and cognition:** Structured aerobic training can improve cognitive and functional outcomes in stroke survivors and is feasible in routine clinical practice; however, the optimal training intensity and dose remain uncertain.
2. **Chronic BDNF as a biomarker:** Resting BDNF responses to exercise are highly variable and rarely correlate with cognitive gains. Consequently, BDNF alone is insufficient as a long-term biomarker of stroke rehabilitation success.
3. **Future directions:** Future research should integrate BDNF with other neurotrophins (e.g. IGF-1 and VEGF), standardise sampling procedures and cognitive outcome reporting, compare moderate- vs. high-intensity exercise in larger cohorts, and combine biological markers with neuroimaging and clinical assessments to develop multimodal predictive models of recovery.

Until such evidence becomes available, clinicians can prescribe aerobic exercise as an effective and accessible strategy to support cognitive recovery after stroke, with exercise intensity individualised according to clinical status and tolerance, while recognising that routine measurement of resting BDNF concentrations is unlikely to provide meaningful information for day-to-day clinical decision-making.

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