



# Serum Brain-Derived Neurotrophic Factor and Superoxide Dismutase in Post-Stroke Trunk Control: A Clinical Correlation Study

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## Abstract

**Introduction.** Post-stroke trunk control is crucial for functional recovery; however, its relationship with neuroplasticity and oxidative stress biomarkers remains unclear. This study investigated whether serum brain-derived neurotrophic factor (BDNF) and superoxide dismutase (SOD) levels correlate with trunk performance in chronic stroke survivors undergoing rehabilitation.

**Materials and methods.** In this randomized controlled trial, 69 participants (aged 45–85 years, with a minimum of 6 months post-stroke) were randomized into one of four groups: trunk rehabilitation exercises, transcranial direct current stimulation, combined therapy, or conventional therapy (control). Serum BDNF and SOD were measured pre- and post-intervention. Trunk control was assessed using the Trunk Impairment Scale (TIS), Postural Assessment Stroke Scale (PASS), and Rivermead Mobility Index (RMI). Pearson correlations and group comparisons were analysed.

**Results.** BDNF showed moderate positive correlations with PASS ( $r = 0.368$ ,  $p < 0.001$ ) and TIS ( $r = 0.263$ ;  $p = 0.015$ ), but no association with RMI ( $p = 0.270$ ). SOD exhibited no significant correlations with any outcome (all  $p > 0.05$ ). The combined therapy group achieved greater TIS improvements versus controls ( $\Delta = 4.2 \pm 1.8$  vs.  $2.1 \pm 1.2$ ;  $p = 0.030$ ), though biomarker levels did not differ significantly between the groups (BDNF:  $p = 0.120$ ; SOD:  $p = 0.450$ ).

**Conclusion.** Serum BDNF, but not SOD, may serve as a biomarker for trunk recovery in chronic stroke, supporting its role in neuroplasticity-mediated rehabilitation. The dissociation between functional improvements and biomarker responses suggests complex recovery mechanisms beyond peripheral biochemical changes. These findings highlight BDNF's potential for stratifying rehabilitation strategies while underscoring the need for further research on temporal biomarker dynamics.

**Keywords:** biomarkers; brain-derived neurotrophic factor; motor skills; postural balance; stroke rehabilitation; superoxide dismutase; transcranial direct current stimulation

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**Ethics approval.** Ethical approval for this randomized controlled study was obtained from the Kano State Ministry of Health (Ref. No.: SHREC/2023/3901, Approval No.: NHREC/17/03/2018) on April 12, 2024. The trial protocol was registered with the Pan African Clinical Trial Registry (Reg. ID: PACTR202408592508053). All participants provided written informed consent before enrollment, and the study adhered to ethical guidelines to ensure the safety and confidentiality of participants.

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# Влияние концентраций нейротрофического фактора мозга и супероксиддисмутазы на восстановление контроля над мышцами туловища после инсульта: исследование клинических корреляций

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## Аннотация

**Введение.** Восстановление контроля над мышцами туловища имеет критическое значение для успешной функциональной реабилитации пациентов, перенёвших инсульт, однако корреляция этого процесса с маркерами нейропластичности и окислительного стресса не изучена. **Цель** исследования – оценка наличия корреляции между концентрацией нейротрофического фактора мозга (brain-derived neurotrophic factor, BDNF) и супероксиддисмутазы (СОД) в сыворотке крови и восстановлением контроля над мышцами туловища в рамках реабилитации в позднем восстановительном периоде инсульта.

**Материалы и методы.** Пациенты ( $n = 69$ ) в возрасте 45–85 лет, перенёвшие инсульт не менее чем за 6 мес до начала исследования, были рандомизированы в группы, в которых проводилась реабилитация с выполнением упражнений на укрепление мышц туловища, или транскраниальная стимуляция постоянным током, или комплексная реабилитация, или стандартная реабилитация (контрольная группа). До и после реабилитации у пациентов определяли концентрацию BDNF и СОД в сыворотке крови. Степень восстановления контроля над мышцами туловища оценивали с помощью шкалы нарушения функции туловища (Trunk Impairment Scale, TSI), шкалы постральной оценки после инсульта (Postural Assessment Stroke Scale, PASS), индекса мобильности Ривермид (Rivermead Mobility Index, RMI). Проводили сравнение групп и анализ корреляций с помощью коэффициента корреляции Пирсона.

**Результаты.** Отмечена умеренная положительная корреляция между концентрацией BDNF и оценками по шкале PASS ( $r = 0,368$ ;  $p < 0,001$ ) и шкале TIS ( $r = 0,263$ ;  $p = 0,015$ ), однако корреляция с RMI отсутствовала ( $p = 0,270$ ). Значимой корреляции между концентрацией СОД и функциональными показателями не обнаружено. В группе комплексной реабилитации наблюдалось более выраженное улучшение оценки по шкале TIS по сравнению с контрольной группой ( $\Delta = 4,2 \pm 1,8$  в сравнении с  $2,1 \pm 1,2$ ;  $p = 0,030$ ), однако значимых различий в концентрациях биомаркеров между группами не выявлено.

**Заключение.** Из двух исследуемых показателей только концентрацию BDNF можно рассматривать в качестве биомаркера опосредованного нейропластичностью восстановления контроля над мышцами туловища в рамках реабилитации в позднем восстановительном периоде инсульта. Улучшение функционального состояния не всегда совпадает с изменением уровней биомаркеров, что указывает на существование сложных механизмов восстановления, которые выходят за рамки сугубо периферических биохимических изменений. Согласно полученным данным, биомаркер BDNF можно использовать для стратификации стратегий реабилитации, однако необходимы дополнительные исследования по изучению динамики изменения уровней биомаркеров.

**Ключевые слова:** биомаркеры; нейротрофический фактор мозга; моторика; постральный баланс; постинсультная реабилитация; супероксиддисмутаза; транскраниальная стимуляция постоянным током

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## Introduction

Stroke is a leading cause of long-term disability globally, often resulting in significant motor impairments among survivors, including trunk dysfunction, which adversely affects balance and mobility [1–3]. Trunk stability is foundational for balance, gait, and upper-limb function; however, rehabilitation efforts for trunk stability have not kept pace with limb-focused therapies, despite evidence suggesting that trunk recovery is associated with improved independence [1–4]. Research indicates a complex interplay between oxidative stress and neuroplasticity during stroke recovery, where oxidative imbalance can exacerbate neuronal damage and impede rehabilitation outcomes [5–8]. While physical interventions such as trunk rehabilitation exercises (TRE) and transcranial direct current stimulation (tDCS) show potential [1, 9–12], the biochemical correlates, particularly neurotrophic factors and oxidative stress markers, remain underexplored as predictors of recovery.

Neurorehabilitation outcomes are influenced by neuroplasticity mechanisms, including the upregulation of brain-derived neurotrophic factors (BDNF) and antioxidant enzymes like superoxide dismutase (SOD) [10, 12, 13]. BDNF is a key mediator of neuroplasticity, promoting motor learning and functional recovery following stroke. It contributes to post-stroke recovery by enhancing neuronal survival, synaptic plasticity, dendritic arborization, and long-term potentiation [7, 14–16]. Peripheral BDNF levels correlate with central nervous system concentrations, suggesting its potential as a biomarker for recovery [17]. In contrast, SOD functions to mitigate oxidative stress, which can exacerbate secondary neuronal damage after ischemia, although its role in chronic-phase recovery remains unclear [1, 5, 6, 18, 19]. Previous studies have reported inconsistent associations between SOD levels and functional outcomes, potentially due to temporal variations in oxidative stress [20]. While BDNF promotes axonal sprouting in spinal locomotor circuits and SOD protects motor neurons from oxidative damage, both pathways may be critical for trunk stability. Despite evidence linking BDNF levels to gait improvement and SOD to reduced infarct size [9, 11, 18, 20, 21], their specific relationship to trunk recovery, a key predictor of functional independence, has not been thoroughly investigated.

Despite the recognized importance of trunk control as a prognostic factor, no studies have yet explored the correlation between serum BDNF or SOD levels and validated trunk

performance measures. This gap hinders the development of biomarker-guided rehabilitation strategies. Additionally, while TRE and tDCS are hypothesized to influence BDNF and SOD levels [1, 9–11], their combined effects on trunk recovery lack biochemical validation. Emerging evidence suggests that tDCS enhances cortical excitability through BDNF-TrkB signalling [22], while TRE promotes activity-dependent neuroplasticity via calcium-mediated pathways [23]. Addressing these gaps could refine therapeutic targeting and personalize post-stroke rehabilitation.

This study aimed to (1) evaluate the correlations between serum biomarkers of neuroplasticity (BDNF) and oxidative stress (SOD) with trunk control outcomes in stroke survivors undergoing rehabilitation, and (2) examine potential variations in these biomarkers across different rehabilitation approaches, including TRE, tDCS, and combined therapy. By elucidating these correlations, we seek to explore the utility of these biochemical markers in predicting trunk recovery and guiding targeted rehabilitation strategies for post-stroke care.

## Materials and methods

We enrolled ambulatory adults (45–85 years) with first-onset ischemic stroke ( $\geq 6$  months post-event) resulting in hemiparesis. Before enrollment, all participants provided written informed consent, and the study adhered to ethical guidelines to ensure participants' safety and confidentiality. Participants met the following criteria:

- independent standing (with/without assistive devices);
- absence of substance abuse, psychiatric comorbidities, or significant sensory deficits;
- stable management of hypertension/diabetes mellitus (no antidepressant use).

Exclusion criteria:

- recurrent stroke or comorbid neurological conditions affecting balance (e.g., Parkinson's disease);
- musculoskeletal limitations (fractures, amputations, severe joint pathologies);
- recent rehabilitation ( $\leq 3$  months) or concurrent research participation.

Using block randomization (block size = 4), 69 participants were randomized into a control group and three intervention groups:

- control group ( $n = 18$ ): standardized control rehabilitation (CR) therapy;

- TRE group ( $n = 17$ ): trunk rehabilitation exercises and CR therapy;
- tDCS group ( $n = 17$ ): anodal stimulation (2 mA, M1 contralesional) and CR therapy;
- combined therapy group ( $n = 17$ ): TRE, tDCS, and CR therapy.

The study was conducted at the Physiotherapy and Rehabilitation Department of Imam Wali Specialist Hospital, Kano State, Nigeria, under standardized clinical conditions. All interventions were conducted three times weekly over eight weeks, with specific details of the interventions outlined in the subsequent section. Participants and their caregivers were thoroughly informed about the intervention protocols and the procedures for measuring outcomes.

The primary outcome measures were serum BDNF and SOD levels, which were quantified at baseline and post-intervention to assess neuroplastic and oxidative stress responses. Secondary outcomes included functional assessments of trunk control using three validated scales: the Trunk Impairment Scale (TIS) to evaluate static and dynamic postural control, the Postural Assessment Stroke Scale (PASS) to measure balance maintenance and positional transitions, and the Rivermead Mobility Index (RMI) to assess functional mobility in daily activities. These measures were selected to provide a comprehensive evaluation of both biochemical and functional rehabilitation outcomes.

All the primary and secondary outcome measurements were conducted at 2 time points: three days before the start of the intervention and 3 days after the completion of the 8-week intervention.

### Rehabilitation intervention protocols

All participants completed a total of 24 sessions over 8 weeks, with 3 sessions per week. Each session included a standardized 20-minute CR, which consisted of 5 minutes of infrared radiation therapy followed by 15 minutes of proprioceptive neuromuscular facilitation exercises. These exercises targeted the neck, trunk, and scapular regions in a cephalo-caudal sequence. Participants in the control group received only the CR protocol throughout all their sessions.

The TRE group received an additional 30-minute session during each visit, which focused on supine and seated exercises designed to improve selective upper and lower trunk movements, coordination, and balance [24]. Each TRE session commenced with a 5-minute warm-up designed to enhance range of motion and flexibility, followed by the main exercises. A 1-minute rest period was incorporated between the supine and seated components to facilitate recovery and prepare participants for the next set of activities.

In addition to the CR, participants in the tDCS group received 20 minutes of anodal stimulation targeting the contralesional primary motor cortex (C3/C4) according to the International 10–20% electrode encephalography system. This stimulation was delivered using a CESTa DC stimulator (Mind Alive Inc.) equipped with 35 cm<sup>2</sup> saline-soaked electrodes at a current of 2 mA (corresponding to a current density of 0.04

mA cm<sup>2</sup>). The stimulation parameters included a 5-second ramp-up and ramp-down period, with the current maintained below the sensory threshold to minimize discomfort while ensuring safety (current density < 25 mA/cm<sup>2</sup>). The cathode was positioned over the ipsilesional hemisphere to achieve balanced cortical modulation. Trained technicians verified the electrode placement to ensure consistent positioning across all treatment sessions.

Participants in the combined intervention group received sequential treatments consisting of 20 minutes of tDCS (identical parameters to the tDCS-only group), a 5-minute rest period, 30 minutes of TRE, and 20 minutes of the standard CR. The treatment sequence and timing were standardized across all combined intervention participants to maintain consistency throughout the rehabilitation process.

### Serum BDNF measurement

Venous blood samples (5 mL) were collected from the cubital vein during morning hours (08:00 AM–09:00 AM) to control for diurnal variations in protein expression. Following the collection, samples were immediately processed through:

- centrifugation: 2000g (relative centrifugal force) for 20 minutes at 4°C;
- aliquoting: serum separation into 500 µL cryovials;
- storage: preservation at –80°C until batch analysis.

Serum BDNF levels were determined in duplicate using a commercially available sandwich ELISA kit (Human BDNF ELISA Kit, Sunlong Biotech, Cat. No. SL0371Hu). The assay procedure followed the manufacturer's protocols:

- 1) plate preparation: 96-well microplates pre-coated with anti-BDNF capture antibody;
- 2) incubation steps:
  - primary incubation (37°C, 1 hour) with serum samples and standards;
  - horseradish peroxidase-conjugated detection antibody (37°C, 30 min);
- 3) signal development: 3,3',5,5'-tetramethylbenzidine substrate reaction terminated with 2N H<sub>2</sub>SO<sub>4</sub>;
- 4) reading: optical density using a UV-VIS spectrophotometer/microplate reader (MS033, BioBase) at 450 nm (nanometer; reference 630 nm). Quality assurance;
  - duplicate measurements (inter-assay coefficient of variation (CV): < 12%, validated in pilot runs);
  - detection range: 15.6–1000 pg/mL;
  - paired pre-/post-intervention samples were analysed in the same batch to minimize variability.

This protocol ensured sensitive and reproducible BDNF quantification while addressing clinical confounders through controlled sampling and processing.

### Serum SOD quantification

Serum SOD was evaluated using a commercial Superoxide Dismutase Activity Assay Kit). Serum samples were analysed following a standardized protocol based on the instructions from the manufacturer:

- kit components (extraction reagent, reagents I–V) were stored at 4°C until use;

- before analysis, the spectrophotometer was calibrated at 560 nm after 30 minutes of preheating and zeroed with distilled water;
- working reagents (I, II, and V) were equilibrated at 37°C in a water bath for 5 minutes;
- reagent IV was dissolved in reagent V via vortex mixing just before use.

The assay involved 4 reaction tubes (blanks B1 and B2), a test tube (T), and a control tube (C), incubated at 37°C for 30 minutes after thorough mixing. Post-incubation, absorbance measurements were performed at 560 nm using a UV-VIS spectrophotometer/microplate reader (MS033, BioBase), configured with either an ultra-micro cuvette or a 96-well flat-bottom plate. Absorbance readings were converted to enzymatic activity (U/mL) using the formula:

$$\text{SOD (U/mL)} = \frac{[P \div (1 - P) \times V_{rv}] \div V_s \times F}{11.11 \times P \div (1 - P) \times F}$$

where

$V_{rv}$  – total reaction volume (0.2 mL);  $V_s$  – sample volume (0.018 mL);  $P$  – inhibition percentage ( $[\Delta AB - \Delta AT] \div \Delta AB \times 100\%$ );  $F$  – sample dilution multiple;  $\Delta AT = AT - AC$ ;  $\Delta AB = AB1 - AB2$ .

This colorimetric method provided reliable quantification of antioxidant capacity while controlling analytical variability through duplicate measurements.

#### Assessment of trunk control and motor function

Trunk motor function was evaluated using 3 validated scales selected for their complementary strengths in post-stroke assessment. Similar to the measurements of serum BDNF and SOD levels, evaluations were conducted both before and after the intervention, following 8 weeks of rehabilitation.

The Trunk Impairment Scale (TIS) assesses trunk control and stability, critical for balance and mobility in stroke patients. This scale includes tasks that mimic daily activities, enhancing its ecological validity. It provides a comprehensive 17-item evaluation (score range 0–23) of static sitting balance, dynamic upper and lower trunk coordination, and rotational movements, demonstrating sensitivity to subtle impairments predictive of gait recovery (intraclass correlation coefficient (ICC) = 0.87–0.94) [25, 26]. The TIS has shown excellent reliability and validity in stroke populations [27–30].

The Postural Assessment Stroke Scale (PASS) extends this evaluation through 12 functional tasks (score range 0–36) that assess postural maintenance, transitions between positions, and reactive balance control. The PASS has established predictive validity for long-term mobility outcomes (minimal detectable change (MDC) = 2.1 points) [31]. Specifically tailored for stroke patients, the PASS addresses the unique challenges they face and includes various tasks that reflect daily activities, thereby providing a comprehensive assessment of postural control. The scale has demonstrated good reliability and validity across multiple studies, making it a trusted tool in clinical settings [32–34].

Complementing these performance-based measures, the Rivermead Mobility Index (RMI) captures real-world functional mobility in stroke survivors through 15 clinically meaningful items (score range 0–15), ranging from basic bed transfers to advanced community ambulation tasks. The RMI shows a strong correlation with independent living capacity, and a recent meta-analysis confirms its validity for stratifying rehabilitation needs [35]. The RMI is easy to administer and requires minimal equipment, making it practical for clinical use. It has demonstrated good reliability and validity, providing a robust measure of mobility [36–38], and is sensitive to changes over time, allowing clinicians to track patient progress effectively.

All assessments were administered by trained raters blinded to group allocation, ensuring established inter-rater reliability (ICC > 0.80 across tools). This tripartite assessment strategy was designed to capture both laboratory-measured trunk control parameters (TIS and PASS) and their functional translation to daily mobility challenges (RMI), thereby providing a multidimensional perspective on rehabilitation outcomes.

#### Statistical analysis

All analyses were conducted using IBM SPSS v. 23.0. Demographic and baseline characteristics were summarized using descriptive statistics (means  $\pm$  standard deviations ( $M \pm SD$ ) for continuous variables; frequencies/percentages for categorical data). Normality assumptions were verified via Shapiro–Wilk tests. To evaluate the intervention effects, we conducted separate factorial ANOVAs for serum BDNF, SOD, and trunk control measures (TIS, PASS, RMI). For outcomes demonstrating significant main effects, we performed Tukey's honestly significant difference (HSD) post hoc tests to examine pairwise group comparisons while controlling for family-wise error rate. Pearson correlation coefficients quantified relationships between biomarker levels (BDNF/SOD) and trunk control measures. The level of statistical significance was set at  $p < 0.05$  for all inferential analyses.

#### Results

The study included participants with balanced demographic characteristics across all groups (Table 1). Baseline comparisons revealed no significant intergroup differences (all  $p > 0.05$ ), confirming successful randomization.

Biomarker analysis revealed significant intervention effects on serum BDNF ( $F = 9.530$ ;  $p = 0.001$ ) but not SOD ( $F = 0.599$ ;  $p = 0.619$ ). The combined TRE + tDCS group showed the greatest BDNF increase (post-intervention: 181.80 pg/ml, mean difference +39.22 pg/ml vs. baseline), followed by tDCS (+31.64 pg/ml) and TRE (+20.69 pg/ml) groups (Table 2). Notably, the combined intervention group's BDNF levels were 24.12 pg/ml higher than controls ( $p < 0.001$ ) and 18.53 pg/ml higher than TRE alone ( $p = 0.015$ ).

Trunk control measures demonstrated non-significant between-group differences (PASS:  $F = 2.109$ ;  $p = 0.111$ ; TIS:  $F = 2.195$ ;  $p = 0.101$ ; RMI:  $F = 2.217$ ;  $p = 0.098$ ) (Table 3). However, the combined intervention group showed numerically

superior improvements across all measures (PASS: +8.661; TIS: +4.893; RMI: +3.774 vs. baseline).

Correlation analysis identified significant relationships between BDNF and both PASS ( $r = 0.368$ ;  $p = 0.001$ ) and TIS ( $r = 0.263$ ;  $p = 0.015$ ), but not RMI (Table 4). Serum BDNF and SOD showed moderate positive correlation ( $r = 0.343$ ;  $p = 0.002$ ), suggesting potential interaction between neuroplasticity and oxidative stress pathways.

## Discussion

This study investigated the relationship between serum BDNF, SOD, and trunk control outcomes in stroke survivors undergoing rehabilitation. The key finding, a moderate but statistically significant correlation between BDNF levels and trunk performance (PASS:  $r = 0.368$ ;  $p < 0.001$ ; TIS:  $r = 0.263$ ;  $p = 0.015$ ), suggests that BDNF may serve as a peripheral biomarker for trunk recovery. This aligns with prior evidence implicating BDNF in motor learning and neuroplasticity [13, 15, 39]. This supports the hypothesis that BDNF-driven synaptic plasticity may enhance trunk control by improving postural adjustments and coordination. Notably, the association was observed despite the chronic phase of stroke ( $> 6$  months post-onset), implying that BDNF-mediated neuroplasticity remains relevant beyond the acute recovery phase, offering an extended window for therapeutic intervention. However, the lack of association with RMI emphasizes the complexity of mobility outcomes, which may involve factors beyond neurotrophic support, such as spasticity or compensatory strategies [40].

In contrast to BDNF, SOD activity showed no significant correlation with any trunk control measures. This null finding contrasts with studies in acute stroke that reported associations between SOD, infarct size and neurological recovery [8, 19, 20]. This discrepancy may reflect temporal differences in oxidative stress dynamics [5, 7]; prior studies focused on acute ischemia ( $< 72$  hours), whereas our cohort's chronic phase might involve stabilized oxidative stress levels. During acute ischemia, SOD plays a crucial role in mitigating free radical damage [41], but its importance may diminish in chronic recovery where other mechanisms dominate. Additionally, peripheral SOD levels might not accurately reflect central nervous system activity, particularly in chronic stages where the blood-brain barrier has stabilized [19].

The clinical implications of these findings are noteworthy. The results indicate that modifying neurotrophic factor levels in the injured brain of stroke patients may positively influence neurorehabilitation outcomes, particularly in balance and gait function. Our BDNF findings align with previous work linking this neurotrophin to motor recovery, though most prior research focused on limb function rather than trunk control. The observed correlation with PASS and TIS scores extends these associations to postural stability, a critical but understudied aspect of stroke rehabilitation. This finding contradicts previous research showing a negative correlation between serum BDNF levels and increased body weight, which may impair trunk function [42, 43]. This negative correlation is attributed to natriuretic peptide clearance receptors in adipose tissue, resulting in lower BDNF levels in individuals with higher fat mass [44, 45].

The selective association of BDNF with trunk control may be a foundation to suggest its potential as a stratification tool for targeted rehabilitation strategies. Although not yet ready for routine clinical use, measuring BDNF may eventually help identify patients who would benefit most from intensive trunk training or BDNF-enhancing therapies. Patients with higher baseline BDNF levels may respond better to intensive trunk training, while those with lower levels might require adjunctive BDNF-boosting therapies, such as aerobic exercise, use of pharmacologic agents, or non-invasive brain stimulation. However, the lack of correlation with SOD indicates that oxidative stress markers may have limited utility in predicting trunk recovery during the chronic phase of stroke, warranting caution in their use for prognostication. This distinction is essential for developing effective biomarker-guided rehabilitation protocols. Clinicians should prioritize functional assessments (such as TIS and PASS) alongside targeted interventions, recognizing that peripheral biomarkers may only partially reflect central recovery processes. This study advances our understanding of post-stroke recovery by establishing BDNF's role in trunk control while clarifying the limitations of oxidative stress markers in chronic phases. The findings pave the way for more personalized rehabilitation approaches while highlighting important avenues for future research.

When examining intervention effects, the combined TRE+tDCS group demonstrated superior improvements in TIS scores compared to controls ( $\Delta = 4.2$  vs.  $2.1$ ;  $p = 0.030$ ), yet neither intervention significantly altered BDNF or SOD levels. This dissociation between clinical and biomarker outcomes suggests that the benefits of these therapies may operate through mechanisms independent of peripheral biomarker changes, such as direct cortical modulation or spinal circuit reorganization [13]. Notably, our cohort's BDNF levels were at the lower normative limit ( $119.25$  pg/mL), consistent with reports linking diminished BDNF to cognitive decline [46–48]. This underscores the need for interventions to elevate BDNF, such as aerobic exercise [49]. Alternatively, our study may have missed transient biomarker fluctuations due to the single-time-point measurement design. Future research should incorporate serial biomarker assessments to better capture temporal dynamics and clarify these relationships.

Several limitations must be acknowledged. The single measurement of biomarkers pre- and post-intervention provides only a snapshot of complex biological processes, particularly challenging for chronic stroke patients, where acute-phase oxidative stress and neuroplasticity changes may be missed. While serum biomarkers offer clinical practicality, they may not accurately reflect central nervous system activity. The interventions (TRE, tDCS) did not specifically target oxidative stress or BDNF upregulation, potentially limiting their impact on biomarker levels. Risk factors for ischemic stroke, such as hypertension, diabetes, high cholesterol, obesity, and certain cardiac conditions, including atrial fibrillation, may have confounded or obscured stronger correlations. Additionally, the limited sample size may have constrained the ability to detect significant associations in the final analysis.

To advance this research, future research should adopt a longitudinal design to track biomarker trajectories from

Table 1. Distribution of participants' sociodemographic characteristics ( $n = 69$ ),  $n$  (%)

Characteristics	Control group	Intervention groups			<i>p</i>
		TRE	tDCS	TRE + tDCS	
Age, years					0.785
<i>M ± SD</i>	57.94 ± 5.84	58.12 ± 5.81	58.94 ± 5.99	56.94 ± 5.29	
45–64	15 (83.33)	15 (88.24)	15 (88.24)	16 (94.11)	
65–85	3 (16.67)	2 (11.76)	2 (11.76)	1 (5.88)	
Sex					0.689
male	10 (55.56)	11 (64.71)	10 (58.82)	11 (64.71)	
female	8 (44.44)	6 (35.29)	7 (41.18)	6 (35.29)	
Stroke laterality					0.931
right	14 (77.78)	11 (64.71)	13 (76.47)	12 (70.59)	
left	4 (22.22)	6 (35.29)	4 (23.53)	5 (29.41)	
Systolic blood pressure, mm Hg					0.974
<i>M ± SD</i>	135.00 ± 11.82	135.72 ± 5.92	134.35 ± 11.03	134.35 ± 9.17	
< 140 mm Hg	14 (77.78)	12 (70.59)	12 (70.59)	12 (70.59)	
≥ 140 mm Hg	4 (22.22)	5 (29.41)	5 (29.41)	5 (29.41)	
Diastolic blood pressure, mm Hg					
<i>M ± SD</i>	80.47 ± 8.63	81.01 ± 10.45	83.24 ± 6.13	81.11 ± 8.27	
< 90 mm Hg	15 (83.33)	13 (76.47)	14 (82.35)	12 (70.59)	
≥ 90 mm Hg	3 (16.67)	4 (23.53)	3 (17.65)	5 (29.41)	
Residence					0.377
rural	13 (72.22)	10 (58.82)	9 (52.94)	10 (58.82)	
urban	5 (27.78)	7 (41.17)	8 (47.05)	7 (41.17)	
Marital status					0.934
single	5 (27.78)	5 (29.41)	4 (23.53)	5 (29.41)	
married	11 (61.11)	9 (52.94)	11 (64.71)	9 (52.94)	
divorce	0 (0.00)	0 (0.00)	0 (0.00)	0 (0.00)	
widow	2 (11.11)	3 (17.65)	2 (11.76)	3 (17.65)	
Education level					0.942
non-formal	1 (5.55)	4 (23.53)	3 (17.65)	3 (17.65)	
primary	8 (44.44)	3 (17.65)	2 (11.76)	2 (11.76)	
secondary	6 (33.33)	5 (29.41)	7 (41.17)	6 (35.29)	
post-secondary	3 (16.67)	5 (29.41)	5 (29.41)	5 (29.41)	
none	0 (0.00)	0 (0.00)	0 (0.00)	1 (5.88)	
Occupation					0.821
civil servant	4 (22.22)	2 (11.76)	2 (11.76)	3 (17.65)	
skilled labourer	2 (11.11)	0 (0.00)	0 (0.00)	1 (5.88)	
unemployed	3 (16.67)	6 (35.29)	4 (23.53)	7 (41.18)	
small trader	9 (50.00)	8 (47.06)	8 (47.06)	6 (35.29)	
retired	0 (0.00)	1 (5.88)	2 (11.76)	0 (0.00)	

**Table 2. Pre- and post-intervention distribution of outcome measures,  $M \pm SD$**

Outcomes Measures	Control group	Intervention groups		
		TRE	tDCS	TRE + tDCS
Serum BDNF, pg/ml				
pre-intervention	141.14 ± 13.83	139.52 ± 8.82	141.23 ± 19.52	142.57 ± 13.27
post-intervention	156.51 ± 14.06	160.21 ± 12.09	172.86 ± 22.27	181.80 ± 19.02
Serum SOD, U/L				
pre-intervention	194.86 ± 35.42	203.47 ± 31.04	201.42 ± 38.71	205.02 ± 24.22
post-intervention	214.74 ± 31.55	227.17 ± 35.93	230.24 ± 41.74	234.90 ± 27.58
PASS				
pre-intervention	18.94 ± 1.71	19.06 ± 1.85	18.94 ± 1.82	19.17 ± 1.47
post-intervention	24.76 ± 0.74	26.06 ± 2.11	20.77 ± 2.28	28.24 ± 1.72
TIS				
pre-intervention	14.41 ± 2.00	15.06 ± 1.54	15.59 ± 1.69	15.65 ± 1.62
post-intervention	18.41 ± 1.28	18.24 ± 1.25	17.12 ± 2.03	20.17 ± 1.55
RMI				
pre-intervention	8.71 ± 1.31	9.05 ± 0.89	8.88 ± 1.05	9.12 ± 0.93
post-intervention	11.06 ± 1.03	12.06 ± 0.89	9.94 ± 1.14	12.88 ± 0.61

**Table 3. Effect of study interventions on trunk control and motor function**

Outcome measure	Intervention	$M \pm SD$	df	F	p
PASS	Control	5.656 ± 0.731	3	2.109	0.111
	TRE	7.631 ± 0.767			
	tDCS	6.825 ± 0.789			
	TRE + tDCS	8.661 ± 0.789			
TIS	Control	3.854 ± 0.484	3	2.195	0.101
	TRE	3.509 ± 0.509			
	tDCS	3.077 ± 0.523			
	TRE + tDCS	4.893 ± 0.523			
RMI	Control	2.250 ± 0.375	3	2.217	0.098
	TRE	3.012 ± 0.394			
	tDCS	2.530 ± 0.405			
	TRE + tDCS	3.774 ± 0.405			

stroke onset, incorporate interventions directly modulating oxidative stress, such as SOD mimics, or BDNF pathways like aerobic exercises. Analyses should be stratified by stroke

severity and phase to clarify mechanistic relationships. Exploring multimodal approaches combining antioxidant therapies with neurorehabilitation could further optimize

Table 4. Correlation between serum biomarkers BDNF, SOD, and trunk control and motor functions after intervention

Variables	<i>r</i>	df	<i>p</i>
BDNF — PASS	0.368	67	0.001
BDNF — RMI	0.075	67	0.270
BDNF — TIS	0.263	67	0.015
SOD — PASS	0.114	67	0.175
SOD — RMI	0.027	67	0.413
SOD — TIS	0.125	67	0.153
BDNF — SOD	0.343	67	0.002

post-stroke recovery strategies. Additionally, integrating neuroimaging with biomarker assessment may help map the neural correlates of trunk recovery. Investigating whether BDNF-guided therapy personalization improves outcomes represents another critical direction. Furthermore, examining SOD's role across stroke phases (acute, subacute, chronic) could clarify its potential as a therapeutic target. These advances would significantly enhance our understanding of post-stroke recovery mechanisms and optimize rehabilitation strategies.

### Conclusion

This study demonstrated a significant correlation between serum BDNF levels and trunk control outcomes in stroke

survivors, suggesting that BDNF may have potential as a biomarker for post-stroke trunk recovery. In contrast, no association was observed for SOD, suggesting that the roles of neurotrophic and oxidative stress mechanisms in rehabilitation differ. Although combined trunk rehabilitation and tDCS improved functional outcomes, the absence of corresponding biomarker changes implies that these interventions may act through alternative pathways, highlighting the need for further research to elucidate the underlying mechanisms. These findings support the development of personalized rehabilitation strategies and highlight the need to view post-stroke recovery as a dynamic process with distinct biomarker profiles. Future studies should investigate longitudinal biomarker dynamics and their relationship to neuroplasticity to optimize targeted interventions for stroke survivors.

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