Physical Activity for Stroke Prevention: Mechanisms, Evidence, and Implementation Strategies

Xuefeng Guo*
School of Physical Education, Southwest Petroleum University, Chengdu 610500, China
*Corresponding email: 202231140104@stu.swpu.edu.cn

Abstract

Stroke is a leading cause of mortality and long-term disability worldwide, largely driven by modifiable vascular and metabolic risk factors. Physical Activity (PA) is a low-cost, accessible intervention that reduces stroke risk, particularly ischemic stroke, and may mitigate stroke severity. In survivors, structured exercise improves cardiorespiratory fitness, blood pressure, mobility, and quality of life, though evidence for preventing recurrent stroke is less robust. Mechanistically, activity benefits hypertension, dyslipidemia, insulin resistance, obesity, endothelial function, inflammation, thrombosis, and cerebral perfusion, and may enhance ischemic tolerance and neuroplasticity. Despite clear benefits, implementation is limited by disability, fatigue, fear, motivation, follow-up, and access. Individualized, multidisciplinary strategies are essential to integrate PA into stroke prevention.

Keywords

Physical Activity, Stroke prevention, Primary prevention, Secondary prevention, Exercise prescription, Neuroprotection

Introduction

Stroke is a heterogeneous cerebrovascular disorder encompassing ischemic stroke, intracerebral hemorrhage, and subarachnoid hemorrhage, with ischemic stroke accounting for the majority of cases. Despite advances in acute reperfusion, neurocritical care, and risk-factor management, stroke remains a leading cause of mortality, long-term disability, and substantial socioeconomic burden globally. Beyond survival, stroke frequently results in cognitive impairment, mobility limitations, mood disturbances, recurrent vascular events, and prolonged healthcare utilization, underscoring the critical importance of effective preventive strategies at both population and individual levels [1].

Stroke pathogenesis is multifactorial. Conventional vascular risk factors - such as hypertension, diabetes, dyslipidemia, obesity, smoking, atrial fibrillation, and atherosclerotic disease - contribute substantially to stroke incidence. However, emerging evidence highlights additional mechanisms, including endothelial dysfunction, chronic low-grade inflammation, autonomic imbalance, thrombotic activation, impaired cerebrovascular reserve, and diminished tissue resilience. These factors collectively influence stroke development, severity, and recovery [2,3]. This complexity provides a mechanistic rationale for lifestyle interventions, particularly PA, to impact multiple pathways simultaneously rather than a single risk factor.

Physical inactivity is increasingly recognized as an independent, modifiable determinant of stroke risk. International guidelines consistently recommend regular exercise as an essential component of primary and secondary stroke prevention, owing to its effects on blood pressure, metabolic control, vascular health, inflammatory modulation, and overall physical reserve. Observational studies and meta-analyses demonstrate an inverse, dose-responsive relationship between habitual PA - especially total and leisure-time activity, and incident stroke, with clinically meaningful benefits achievable at moderate, attainable activity levels [4]. In stroke survivors and individuals with Transient Ischemic Attack, structured exercise interventions improve cardiovascular risk profiles, functional capacity, mobility, and quality of life. Yet barriers such as residual disability, fatigue, cognitive or mood impairments, fear of falling, and environmental

limitations limit long-term adherence.

This review provides a comprehensive, evidence-based synthesis of the role of PA in stroke prevention. It integrates epidemiological data, mechanistic insights, and clinical implementation strategies, highlighting the importance of individualized, multidisciplinary approaches to maximize preventive benefit.

Epidemiological evidence for PA in stroke prevention

Primary prevention: Population-level evidence and dose-response relationships

The epidemiological association between PA and stroke prevention has been demonstrated consistently across large prospective cohort studies, pooled analyses, and meta-analyses. Although early studies varied in their definitions of activity exposure and stroke outcome, the overall direction of effect has been remarkably consistent: individuals who engage in regular PA tend to have a lower risk of developing stroke than sedentary individuals. This relationship appears particularly robust for ischemic strokes, which is biologically plausible given the close connections between PA and atherosclerosis, blood pressure regulation, metabolic health, and thrombosis [5].

Recent meta-analytic work has strengthened this evidence base by examining dose-response patterns rather than simple active-versus-inactive comparisons. A large dose-response meta-analysis reported that higher total and moderate-to-vigorous PA were associated with progressively lower stroke risk across a very large, pooled population. Importantly, the greatest relative gains appeared when individuals moved from low or inactive levels toward moderate activity, while additional benefits at higher exposure levels tended to plateau [6]. This nonlinear pattern has major implications for public health because it suggests that the largest marginal risk reductions may occur in the least active segments of the population. In other words, encouraging sedentary adults to begin consistent moderate activity may have greater preventive yield than focusing exclusively on high-volume training.

A similar pattern has been observed for leisure-time PA. Meta-analyses indicate that total and leisure-time activity is inversely associated with both overall stroke and ischemic stroke, though associations with hemorrhagic stroke are generally weaker and less consistent. Leisure-time activity may be especially relevant because it is often more modifiable than occupational activity and may better reflect sustained cardiovascular conditioning. Broader cardiovascular outcome analyses also show that non-occupational PA is associated with lower risks of cardiovascular disease, cancer, and all-cause mortality, reinforcing the idea that stroke prevention through PA is part of a larger spectrum of cardiometabolic benefit [7]. An additional point of interest is the relationship between PA and stroke severity at the time of presentation. Systematic review evidence suggests that individuals who were physically active prior to stroke onset may present with less severe deficits at admission. Although residual confounding cannot be excluded, this observation raises the possibility that regular activity does more than simply reduce incident stroke risk. It may also improve cerebrovascular resilience, collateral recruitment, cardiorespiratory reserve, or systemic stress response during acute ischemia. This concept aligns with growing interest in preconditioning and exercise-induced ischemic tolerance as mechanisms linking habitual activity with more favorable stroke phenotypes.

Nevertheless, the evidence for primary prevention remains predominantly observational. Randomized controlled trials directly designed to test incident stroke as the primary endpoint are limited, largely because such trials require very large sample sizes and long follow-up periods. As a result, much of the interventional evidence must be inferred from improvements in intermediate risk markers such as blood pressure, insulin sensitivity, body weight, lipid profile, and inflammatory status. From a methodological standpoint, this is an important limitation. However, in the context of stroke prevention, these intermediates are not trivial surrogates: They are well-established drivers of vascular risk and constitute biologically credible pathways through which PA may reduce stroke incidence. A further consideration is measurement heterogeneity. Self-reported PA may be affected by recall bias and social desirability, whereas device-based measures such as accelerometry provide more objective estimates. Studies using accelerometer derived activity profiles have strengthened confidence in the association between movement and vascular outcomes, especially among high-risk populations such

as adults with hypertension [8]. This supports the view that protective relationships are not merely an artifact of self-report methodology.

Overall, the epidemiological evidence strongly supports PA as a major component of primary stroke prevention. The most persuasive conclusions are that regular activity reduces overall stroke risk. The association is strongest for ischemic stroke. The benefit follows a dose-response pattern with diminishing returns at higher levels. Clinically meaningful protection is achievable at moderate volumes of activity that are realistic for the general population.

Secondary prevention: Stroke survivors, Transient Ischemic Attack populations, and long-term outcomes

The relevance of PA in secondary prevention is both intuitive and clinically compelling. Patients with prior stroke or Transient Ischemic Attack represent a high-risk population in whom risk-factor modification is urgent, recurrent events are common, and disability may interact with sedentary behavior to accelerate further decline. In these individuals, PA may serve a dual purpose: reducing vascular risk while simultaneously improving mobility, participation, mood, and functional independence.

Observational evidence indicates that physically active stroke survivors have lower all-cause mortality than inactive survivors [9]. This association has been reported in community-dwelling populations and appears to persist after adjustment for several demographic and clinical variables. Although observational design limits causal inference, the consistency of these findings supports the clinical importance of maintaining activity after stroke. Importantly, PA in this context may reflect both behavioral exposure and a marker of preserved functional reserve. Even so, from the standpoint of clinical care, both interpretations support efforts to promote movement and prevent deconditioning.

The gap between recommendation and practice remains substantial. National data from the United States indicate that a relatively small proportion of stroke survivors meet recommended activity targets [10]. This low adherence is concerning because inactivity after stroke may amplify recurrent vascular risk, worsen physical frailty, and contribute to social isolation and reduced quality of life. Longitudinal studies also suggest that adherence to both PA and cardiovascular prevention recommendations decline over time after rehabilitation discharge, underscoring that the transition from supervised care to community living is a vulnerable period [11,12]. Interventional studies in stroke survivors provide encouraging but still incomplete evidence. In the “PREVENT trial”, a 12-week program combining exercise and education in patients with non-disabling stroke or Transient Ischemic Attack led to improvements in selected vascular risk indicators, including diastolic blood pressure and low-density lipoprotein cholesterol [13]. Although the study was not powered to detect differences in recurrent stroke, it demonstrated that structured programs can be implemented safely and may favorably influence mechanisms relevant to secondary prevention. Similarly, a telehealth-delivered pilot trial showed that remotely supported PA and diet interventions are feasible and can produce meaningful behavior change in selected stroke populations [14]. These findings are particularly relevant in healthcare systems where access to in-person long-term rehabilitation is limited.

However, the literature also shows that general lifestyle advice alone is often insufficient. Systematic review evidence suggests that non-specific lifestyle interventions without structured support, individualized planning, or behavioral reinforcement rarely produce durable increases in PA after stroke or Transient Ischemic Attack (TIA) [15]. This is a critical translational insight. Simply advising patients to “exercise more” does not adequately address the complex barriers they face. These barriers may include weakness, spasticity, imbalance, fatigue, pain, transportation difficulties, low self-efficacy, caregiver dependence, cognitive deficits, and fear of recurrent stroke or falling [16-18]. Accordingly, effective secondary prevention programs should not be conceived as information delivery alone. They should function as supported behavior-change interventions embedded within a broader rehabilitation and cardiovascular prevention framework.

The secondary prevention literature also highlights the broader outcomes that matter after stroke. Even when recurrent stroke is not measured directly, exercise interventions may improve cardiorespiratory fitness, gait speed, walking distance, balance, cognition, and health-related quality of life. These gains are clinically

meaningful because they influence independence and the ability to sustain preventive behaviors. A patient who walks more efficiently, feels less fatigued, and gains confidence in mobility is more likely to remain active over time. Thus, functional improvement may be viewed not only as a rehabilitation outcome but also as a mediator of long-term prevention. An additional point of emerging interest is the role of digital health tools.

Remote coaching, tele-rehabilitation platforms, mobile applications, and wearable activity monitors may help extend the benefits of rehabilitation into the chronic phase. Such tools can provide feedback, self-monitoring, goal reinforcement, and contact with clinicians or therapists, all of which may help bridge the gap between short-term supervised exercise and long-term autonomous activity. Nevertheless, their effectiveness likely depends on usability, patient cognition, caregiver support, technological access, and integration into ongoing care pathways.

Taking together, current evidence supports the inclusion of PA as a core component of secondary stroke prevention. The strongest evidence concerns improvements in vascular risk markers, physical function, and survival-related outcomes, whereas evidence for direct reduction of recurrent stroke remains less definitive than desired. This should not be interpreted as a reason for therapeutic hesitation. Rather, it reflects the practical difficulty of conducting long-duration recurrence trials in a heterogeneous and disabled population. From a clinical perspective, the cumulative evidence is sufficiently strong to justify systematic implementation of individualized PA strategies after stroke and TIA.

Biological mechanisms underlying the protective effects of PA

Modification of traditional vascular and metabolic risk factors

A central mechanism through which PA reduces stroke risk is its favorable effect on conventional vascular risk factors. Hypertension is the single most important modifiable determinant of both ischemic and hemorrhagic stroke, and regular PA has well-established antihypertensive effects. Aerobic exercise can lower resting blood pressure, reduce sympathetic overactivity, improve endothelial-dependent vasodilation, and enhance vascular compliance. Resistance training, when appropriately prescribed, may further contribute to overall blood pressure control and cardiometabolic health. Because blood pressure plays a dominant role across stroke subtypes, this mechanism alone gives PA major preventive relevance.

PA also improves glucose metabolism and insulin sensitivity. In patients with diabetes or prediabetes, chronic hyperglycemia promotes endothelial injury, oxidative stress, inflammation, and accelerated atherosclerosis. Lifestyle patterns that include regular PA are associated with lower stroke incidence in diabetes populations, highlighting the contribution of metabolic control to cerebrovascular protection [19]. Exercise enhances skeletal muscle glucose uptake, improves insulin signaling, reduces central adiposity, and may lower glycated hemoglobin over time. These changes reduce the vascular toxicity associated with dysglycemia and help mitigate the progression of small- and large vessel diseases. Lipid metabolism is another important pathway. Regular PA is associated with improved lipid profiles, including increased high-density lipoprotein cholesterol and reductions in triglyceride-rich lipoproteins. Although the absolute magnitude of lipid change with exercise alone may vary, these effects occur in parallel with improved weight regulation, reduced visceral adiposity, and attenuated chronic low-grade inflammation. Because adipose tissue is metabolically active and contributes to inflammatory cytokine production, reduction of excess adiposity may lessen vascular injury beyond its effect on body mass alone.

These risk-factor improvements should not be viewed in isolation. Hypertension, insulin resistance, dyslipidemia, obesity, and inflammation interact biologically and often cluster within the same patient. PA addresses this clustering effect by exerting coordinated benefits across several pathways simultaneously. This multidimensional action distinguishes exercise from many single-target interventions and helps explain why it remains valuable even when patients are already receiving pharmacological therapy for individual risk factors.

Direct effects on endothelial function, cerebrovascular reserve, and ischemic tolerance

Beyond systemic risk-factor modification, PA appears to influence vascular biology directly. Exercise enhances

endothelial function through increased nitric oxide bioavailability, improved shear-stress signaling, and reduced expression of vasoconstrictive and pro-inflammatory mediators. Healthier endothelium promotes vasodilation, inhibits platelet aggregation, reduces leukocyte adhesion, and improves overall vascular homeostasis. These properties are directly relevant to ischemic stroke prevention because endothelial dysfunction contributes to atherosclerotic progression, plaque instability, and impaired cerebral perfusion [20].

Another important concept is cerebrovascular adaptability. Habitual exercise may improve vascular remodeling, collateral circulation, and cerebral blood flow reserve. In ischemic stroke, the quality of collateral circulation strongly influences infarct growth and tissue survival. Evidence linking pre-stroke PA with better cerebral collateral status supports the hypothesis that regular exercise enhances the brain's capacity to tolerate arterial occlusion [21]. This may help explain why physically active individuals sometimes present with less severe neurological deficits despite suffering stroke. The idea of exercise-related conditioning also deserves attention. Repeated physiological stress from PA may trigger adaptive responses resembling ischemic preconditioning, including enhanced antioxidant defense, improved mitochondrial function, more efficient energy utilization, and altered cellular stress signaling [22]. Although the exact clinical significance of these mechanisms remains difficult to quantify in humans, they provide a biologically plausible explanation for how habitual activity might reduce tissue vulnerability during acute cerebrovascular events.

Inflammation, hemostatic balance, and neurobiological adaptation

Stroke pathophysiology is strongly influenced by inflammation and thrombosis. PA can modulate both of these domains. Regular exercise is associated with lower systemic inflammatory burden, including favorable effects on inflammatory cytokines and endothelial activation. Because chronic inflammation contributes to plaque development, vascular stiffness, and thrombogenic potential, attenuation of inflammation may reduce both the initiation and progression of cerebrovascular disease. Exercise also affects hemostatic balance. Moderate habitual PA is associated with improved fibrinolytic activity and reduced prothrombotic tendency, while extreme unaccustomed exertion may transiently increase thrombogenic stress in susceptible individuals. In preventive medicine, this distinction is important: The beneficial effects of regular, appropriately dosed activity differ from the physiological stress of sporadic excessive exertion. Over the long term, exercise appears to shift vascular biology toward a less thrombosis-prone state, thereby contributing to protection against ischemic events.

Neurobiological adaptations may further support stroke prevention and post-stroke recovery. Exercise promotes the release of neurotrophic and muscle-derived signaling molecules that influence synaptic plasticity, neurogenesis, cognitive function, and neuronal survival. Irisin has attracted particular attention as a possible mediator linking skeletal muscle activity to brain health, although its translational role remains under active investigation [23]. Similarly, interest has grown in whether high-intensity interval training may stimulate neuroplastic processes more efficiently than conventional continuous training, especially during rehabilitation [24]. Current evidence suggests promise, but patient selection and safety remain critical, particularly in those with frailty, uncontrolled cardiovascular disease, or significant residual neurological deficits.

Thus, the mechanistic literature supports a broad model in which PA reduces stroke risk through coordinated action on vascular risk factors, endothelial function, perfusion reserve, inflammation, thrombosis, and neurobiological resilience. This breadth of effect is one of the strongest scientific arguments for integrating PA into stroke prevention strategies.

Stroke subtypes and clinical heterogeneity

The protective effect of PA is not uniform across all forms of stroke. In general, evidence is more consistent and stronger for ischemic stroke than for hemorrhagic stroke. This is likely to reflect the fact that PA acts directly on pathways central to ischemic stroke pathogenesis, including atherosclerosis, endothelial dysfunction, dyslipidemia, metabolic syndrome, and thrombotic risk. By contrast, hemorrhagic stroke is more strongly driven by chronic hypertension, vascular fragility, and structural vessel abnormalities. PA still has

preventive value in this setting, particularly through blood pressure control, but the magnitude and consistency of association are less clear.

This subtype difference should not lead to a simplistic conclusion that exercise is only relevant for ischemic events. Rather, it suggests that the dominant preventive mechanism varies significantly according to distinct pathophysiology. In ischemic stroke, PA may reduce incident events and possibly attenuate severity through favorable effects on vascular structure and tissue tolerance. In hemorrhagic stroke, benefit is more likely mediated through control of hypertension, vascular health maintenance, and reduction of metabolic burden. At the same time, clinicians must recognize that excessively vigorous or poorly supervised exercise may provoke large transient blood pressure elevations in individuals with uncontrolled hypertension. Therefore, intensity prescription should be carefully individualized in patients at high hemorrhagic risk.

Patient-level heterogeneity is equally important in clinical application. Age modifies both physiology and practical implementation. In older adults, PA remains highly beneficial, but prescription must account for frailty, sarcopenia, balance impairment, osteoarthritis, and increased fall risk. Women may derive at least similar, and in some studies greater, stroke-risk reduction from PA than men, although biological explanations remain uncertain and social or behavioral confounding may contribute [25]. In stroke survivors with residual disability, cognitive impairment, aphasia, or depression, the challenge is less about proving benefit and more about translating evidence into feasible daily practice that supports long-term adherence.

Longitudinal studies show that maintenance of PA often becomes more difficult after the early rehabilitation phase. Cognitive status may influence this further, as lower activity before and after stroke has been associated with less favorable early cognitive performance [26]. For these reasons, PA counseling in stroke prevention should not rely on generic recommendations alone. It must be carefully adapted to individual neurological status, mobility, cognition, cardiovascular comorbidity, medication burden, psychosocial support, and environmental access to ensure safe and sustainable participation.

Clinical recommendations and practical exercise prescription

Guideline framework and the clinical meaning of "recommended activity"

International guidance provides a clear baseline for exercise counseling. The World Health Organization recommends that adults perform 150 to 300 minutes of moderate-intensity aerobic PA per week, or 75 to 150 minutes of vigorous-intensity activity, or an equivalent combination, together with muscle-strengthening activity on at least 2 days per week [27]. Stroke-specific prevention guidelines similarly support lifestyle modification, including regular PA, as a key component of comprehensive secondary prevention after stroke or Transient Ischemic Attack [28]. For clinicians, however, the key issue is not merely reciting guideline thresholds. It is interpreting them in a way that supports sustainable behavior change. Many high-risk patients are sedentary, deconditioned, multimorbid, or functionally limited, for them, an immediate goal of 150 minutes per week may feel unrealistic. The evidence that substantial benefit occurs when individuals move from inactivity to modest regular activity is therefore highly valuable. It supports the practical principle that "some activity is better than none", and that progression toward guideline targets may be more important than immediate achievement of them.

Sedentary behavior should also be addressed explicitly. A patient who meets weekly exercise targets but spends most of the remainder of the day sitting may still carry elevated vascular risk. Interrupting prolonged sedentary time with brief walking, standing, or light functional activity may be particularly relevant in older adults and stroke survivors, for whom total daily movement may matter as much as formal exercise sessions.

Exercise prescription using the FITT principle

The frequency, intensity, time, and type (FITT) framework provides a practical structure for individualized prescription. For many patients at elevated stroke risk, aerobic activity on 3 to 5 days per week is a reasonable starting point, gradually progressing toward guideline-concordant weekly volume. Moderate intensity is often the most appropriate initial target because it is safer, more sustainable, and sufficiently effective for many cardiometabolic outcomes. Walking, stationary cycling,

treadmill exercise, aquatic exercise, and other rhythmic aerobic modalities are commonly suitable.

Resistance training should be included at least twice weekly when feasible. This is particularly important for older adults and stroke survivors because muscular weakness contributes to poor mobility, insulin resistance, reduced walking efficiency, and loss of independence. Functional strengthening of the lower limbs and trunk may also improve gait, transfer ability, and balance, thereby indirectly supporting greater daily activity. Flexibility and balance training can be added for individuals with fall risk, reduced range of motion, or postural instability.

Intensity should be individualized according to cardiovascular status, functional capacity, and neurological limitation. Moderate intensity can be operationalized through perceived exertion, the talk test, or percentage of heart-rate reserve where appropriate. In stroke survivors, clinicians should be mindful of autonomic dysfunction, beta-blocker use, gait asymmetry, exertional fatigue, and impaired motor control, all of which may make simple heart-rate targets less reliable. Supervised initiation may therefore be preferable in selected patients. Session duration should also be individualized. Some patients can tolerate continuous sessions of 30 to 45 minutes, whereas others may require multiple shorter bouts accumulated across the day. This is especially relevant after stroke, when fatigue, attention limitations, or mobility constraints may reduce tolerance for prolonged exercise. Accumulated activity is still meaningful if it contributes to total weekly volume and encourages a transition away from sustained sedentariness.

Safety assessment, adherence, and long-term integration into stroke prevention care

Successful exercise prescription depends not only on the planned dose but also on screening, education, support, and follow-up. Before prescribing activity in high-risk individuals or stroke survivors, clinicians should consider cardiovascular stability, blood pressure control, arrhythmia burden, fall risk, mobility limitation, and cognitive ability to follow instructions. In some patients, referrals for supervised rehabilitation or exercise testing may be appropriate.

Adherence remains the central challenge. Qualitative studies show that barriers to activity after stroke often include fear of falling, fatigue, transportation problems, embarrassment about disability, low confidence, uncertainty about safe intensity, and lack of tailored instruction [29]. Advances in deep learning-based motion analysis technology have further optimized the individualization and scientific of exercise prescription for stroke patients and high-risk groups; video analysis of key motion positions can accurately identify abnormal movement patterns, quantify exercise intensity and posture standardization, and provide real-time corrective feedback for unsupervised home exercise [30]. These findings suggest that successful implementation requires more than knowledge transfer. Patients often benefit from goal setting, symptom-based progression, motivational reinforcement, family involvement, and programs that clearly connect exercise with personally meaningful outcomes such as walking independently, returning to work, or reducing future stroke risk. Behaviorally informed and technology-assisted models may improve long-term uptake. Telehealth delivery, wearable devices, step-count feedback, app-based reminders, and remote coaching can help extend support beyond formal rehabilitation. These strategies may be especially useful for geographically isolated patients or those with limited access to specialist care. However, technology should complement rather than replace individualized clinical judgment. Digital tools are most effective when embedded in multidisciplinary pathways that include physicians, rehabilitation professionals, nurses, and caregivers.

Ultimately, the most effective exercise prescription is one that is personalized, safe, and sustainable. For stroke prevention, the goal is not simply to generate short-term increases in activity, but to incorporate movement into the patient's long-term lifestyle and risk-reduction strategy.

Conclusion

PA should be regarded as a foundational element of contemporary stroke prevention. The available evidence indicates that regular PA is associated with lower risk of first-ever stroke, especially ischemic stroke, and with better long-term outcomes among stroke survivors. Its preventive effects are biologically plausible and multifactorial. They encompass improvement of blood pressure, metabolic health, endothelial function,

inflammatory burden, thrombotic balance, cerebrovascular reserve, and possibly ischemic tolerance and neuroplasticity. Although direct trial evidence for reduction of recurrent stroke remains less extensive than ideal, the cumulative epidemiological, mechanistic, and clinical data strongly support routine incorporation of PA into both primary and secondary prevention.

The principal challenge is no longer whether PA matters, but how to implement it effectively. Generic advice is insufficient for many patients, especially those with prior stroke, disability, multimorbidity, or low self-efficacy. A clinically meaningful approach requires individualized prescription, safety assessment, behavioral support, and long-term follow-up that extends beyond the formal rehabilitation period. Telehealth platforms, wearable monitoring, and multidisciplinary care models may strengthen adherence and allow more patients to benefit from exercise-based prevention.

In summary, PA offers a rare combination of accessibility, low cost, systemic biological benefit, and relevance across the entire stroke continuum. It should not be framed as an optional adjunct to medical care, but as a core component of comprehensive stroke prevention policy and practice.

Funding

This work was supported by Sichuan University Students Innovation Training Program (Grant No. S202410615269).

Acknowledgements

The author would like to show sincere thanks to those techniques who have contributed to this research.

Conflict of Interest

The author declares no conflict of interest.

References

[1] Tian, G., Ji, Z., Lin, Z., Pan, S., Yin, J. (2021) Cerebral autoregulation is heterogeneous in different stroke mechanism of ischemic stroke caused by intracranial atherosclerotic stenosis. *Brain and Behavior*, 11(1), e01907.

[2] Johansson, A., Drake, I., Engström, G., Acosta, S. (2021) Modifiable and non-modifiable risk factors for atherothrombotic ischemic stroke among subjects in the Malmö Diet and Cancer Study. *Nutrients*, 13(6), 1952.

[3] Alsbrook, D. L., Di Napoli, M., Bhatia, K., Biller, J., Andalib, S., Hinduja, A., Rodrigues, R., Rodriguez, M., Sabbagh, S, Y., Selim, M., Farahabadi, M, H., Jafarli, A., Divani, A. A. (2023) Neuroinflammation in acute ischemic and hemorrhagic stroke. *Current Neurology and Neuroscience Reports*, 23(8), 407-431.

[4] Hung, S. H., Ebaid, D., Kramer, S., Werden, E., Baxter, H., Campbell, B. C., Brodtmann, A. (2021) Pre-stroke Physical Activity and admission stroke severity: a systematic review. *International Journal of Stroke*, 16(9), 1009-1018.

[5] Liu, Y., Ya, X., Zhou, L., Chen, T., Shen, F., Chen, Y., Wang, X. (2025) Dose-response association of total and leisure-time Physical Activity with the risk of different subtypes of stroke: a systematic-review and meta-analysis. *Journal of Public Health*, 47(3), 567-583.

[6] Bakker, E. A., Lee, D. C., Hopman, M. T., Oymans, E. J., Watson, P. M., Thompson, P. D., Eijsvogels, T. M. (2021) Dose-response association between moderate to vigorous Physical Activity and incident morbidity and mortality for individuals with a different cardiovascular health status: a cohort study among 142,493 adults from the Netherlands. *PLoS Medicine*, 18(12), e1003845.

[7] Garcia, L., Pearce, M., Abbas, A., Mok, A., Strain, T., Ali, S., Brage, S. (2023) Non-occupational Physical Activity and risk of cardiovascular disease, cancer and mortality outcomes: a dose-response meta-analysis of large prospective studies. *British Journal of Sports Medicine*, 57(15), 979-989.

[8] del Pozo Cruz, B., Ahmadi, M., Inan-Eroglu, E., Huang, B. H., Stamatakis, E. (2022) Prospective associations of accelerometer - assessed Physical Activity with mortality and incidence of cardiovascular disease among adults with hypertension: the UK biobank study. *Journal of the American Heart Association*, 11(6), e023290.

[9] Joundi, R. A., Patten, S. B., Lukmanji, A., Williams, J. V., Smith, E. E. (2021) Association between Physical Activity and mortality among community-dwelling stroke survivors. *Neurology*,

97(12), e1182-e1191.

[10] Vemuri, A. K., Hejazian, S. S., Sadr, A. V., Zhou, S., Decker, K., Hakun, J., Zand, R. (2025) Adherence to Physical Activity recommendations among stroke survivors in the United States. *Stroke*, 56(1), 113-121.

[11] Fini, N. A., Bernhardt, J., Churilov, L., Clark, R., Holland, A. E. (2021) Adherence to Physical Activity and cardiovascular recommendations during the 2 years after stroke rehabilitation discharge. *Annals of Physical and Rehabilitation Medicine*, 64(2), 101455.

[12] Fini, N. A., Bernhardt, J., Churilov, L., Clark, R., Holland, A. E. (2021) A 2-year longitudinal study of Physical Activity and cardiovascular risk in survivors of stroke. *Physical Therapy*, 101(2), pzaa205.

[13] MacKay-Lyons, M., Gubitz, G., Phillips, S., Giacomantonio, N., Firth, W., Thompson, K., Dewolfe, J. (2022) Program of rehabilitative exercise and education to avert vascular events after non-disabling stroke or Transient Ischemic Attack (PREVENT trial): a randomized controlled trial. *Neurorehabilitation and Neural Repair*, 36(2), 119-130.

[14] English, C., Ramage, E. R., Attia, J., Bernhardt, J., Bonevski, B., Burke, M., Patterson, A. (2024) Secondary prevention of stroke. A telehealth-delivered Physical Activity and diet pilot randomized trial (ENAbLE-pilot). *International Journal of Stroke*, 19(2), 199-208.

[15] Hendrickx, W., Vlietstra, L., Valkenet, K., Wondergem, R., Veenhof, C., English, C., Pisters, M. F. (2020) General lifestyle interventions on their own seem insufficient to improve the level of Physical Activity after stroke or TIA: a systematic review. *BMC Neurology*, 20(1), 168.

[16] Karadag-Saygi, E., Giray, E., Eren, N., Yolcu, G., Coskun, O. K., Cifcili, S. (2024) Barriers and facilitators to Physical Activity participation among community-dwelling physically inactive individuals after stroke: a qualitative exploratory study. *International Journal of Rehabilitation Research*, 47(1), 34-40.

[17] Krawcyk, R. S., Christoffersen, L. C., Danielsen, A. K., Kruuse, C. (2023) Motivators for Physical Activity in patients with minor stroke: a qualitative study. *Disability and Rehabilitation*, 45(2), 277-285.

[18] Fini, N. A., Bernhardt, J., Said, C. M., Billinger, S. A. (2021) How to address Physical Activity participation after stroke in research and clinical practice. *Stroke*, 52(6), e274-e277.

[19] Iwase, M., Komorita, Y., Ohkuma, T., Fujii, H., Ide, H., Yoshinari, M., Kitazono, T. (2021) Incidence of stroke and its association with glycemic control and lifestyle in Japanese patients with type 2 diabetes mellitus: the Fukuoka diabetes registry. *Diabetes Research and Clinical Practice*, 172, 108518.

[20] Wang, C., Redgrave, J., Shafizadeh, M., Majid, A., Kilner, K., Ali, A. N. (2019) Aerobic exercise interventions reduce blood pressure in patients after stroke or transient ischaemic attack: a systematic review and meta-analysis. *British Journal of Sports Medicine*, 53(24), 1515-1525.

[21] Hung, S. H., Kramer, S., Werden, E., Campbell, B. C., Brodtmann, A. (2022) Pre-stroke Physical Activity and cerebral collateral circulation in ischemic stroke: a potential therapeutic relationship? *Frontiers in Neurology*, 13, 804187.

[22] Hess, D. C., Khan, M. B., Kamat, P., Vaibhav, K., Dhandapani, K. M., Baban, B., Andersen, G. (2021) Conditioning medicine for ischemic and hemorrhagic strokes. *Conditioning Medicine*, 4(3), 124.

[23] Zhang, Y., Zhang, X., Lin, S. (2022) Irisin: a bridge between exercise and neurological diseases. *Heliyon*, *8*(12), e12352.

[24] Hugues, N., Pellegrino, C., Rivera, C., Berton, E., Pin-Barre, C., Laurin, J. (2021) Is high-intensity interval training suitable to promote neuroplasticity and cognitive functions after stroke? *International Journal of Molecular Sciences*, 22(6), 3003.

[25] Madsen, T. E., Samaei, M., Pikula, A., Yu, A. Y., Carcel, C., Millsaps, E., Bushnell, C. (2022) Sex differences in Physical Activity and incident stroke: a systematic review. *Clinical Therapeutics*, 44(4), 586-611.

[26] Viktorisson, A., Andersson, E. M., Lundström, E., Sunnerhagen, K. S. (2021) Levels of Physical

Activity before and after stroke in relation to early cognitive function. *Scientific Reports*, 11(1), 9078.

[27] Piercy, K. L., Troiano, R. P., Ballard, R. M., Carlson, S. A., Fulton, J. E., Galuska, D. A., George, S, M., Olson, R. D. (2018) The Physical Activity guidelines for Americans. *Jama*, 320(19), 2020-2028.

[28] Kleindorfer, D. O., Towfighi, A., Chaturvedi, S., Cockroft, K. M., Gutierrez, J., Lombardi-Hill, D., Williams, L. S. (2021) 2021 guideline for the prevention of stroke in patients with stroke and Transient Ischemic Attack: a guideline from the American Heart Association/American Stroke Association. *Stroke*, 52(7), e364-e467.

[29] Walsh, M., Galvin, R., Horgan, N. F. (2017) Fall-related experiences of stroke survivors: a meta-ethnography. *Disability and Rehabilitation*, 39(7), 631-640.

[30] Wang, Z., Cheng, H., Qin, Q. (2025) The research on video analysis of key motion positions based on deep learning technology. *International Journal of e-Collaboration (IJeC)*, 21(1), 1-16.