Research Connections, 2026, 1, vmag073
https://doi.org/10.1093/rescon/vmag073
Published: 16 May 2026
Commentary

research connections OXFORD

Neurovascular ageing as an upstream determinant of injury risk: a resilience-based framework from Japan

Kaechang Park*

Department of Decoded Neurofeedback, Computational Neuroscience Laboratory Group, Advanced Telecommunications Research (ATR) Institute International, Kyoto 619-0288, Japan
*Corresponding author: E-mail: kpark@atr.jp.

Editor: Jinghua Li

Abstract

Mobility-related injury remains a major cause of morbidity, disability, and loss of independence in older adults. Conventional prevention frameworks predominantly emphasize environmental exposure while underrepresenting biological heterogeneity. In later life, injury risk is not simply a function of exposure but emerges from a dynamic mismatch between environmental demand and neurobiological capacity. White matter hyperintensities (WMH) and brain atrophy (BA), reflecting cumulative neurovascular burden, may reduce the neural reserve required for safe mobility. We propose an exposure–resilience interaction framework in which injury vulnerability arises when environmental demands exceed compensatory capacity. Evidence from neuroimaging and functional studies indicates that structural brain burden is associated with impaired hazard perception and reduced real-world performance, even among cognitively normal individuals. Japan provides a natural translational setting, where advanced population ageing and existing brain health screening systems intersect. Integrating neurovascular resilience into injury epidemiology reframes prevention from external hazard control toward preservation of intrinsic capacity.

Keywords magnetic resonance imaging, injury prevention, white matter hyperintensities, brain atrophy, neurovascular ageing

Background and public health context

Population ageing is reshaping patterns of morbidity worldwide, with mobility-related injury emerging as a major contributor to disability and loss of independence in later life. In Japan, where more than 28% of the population is aged ≥65 years, older adults account for a disproportionate share of serious injury and fatality outcomes despite reduced exposure compared with younger populations [1, 2]. Falls, trauma, traffic accidents, and other injury mechanisms represent leading causes of functional decline and long-term care dependency in this group [3].

Traditional prevention strategies have focused on environmental safety, infrastructure design, and behavioral regulation. While these approaches have yielded measurable benefits, they implicitly assume relatively uniform responses to comparable exposures. This assumption becomes increasingly untenable in ageing populations, where biological heterogeneity strongly influences functional capacity.

In ageing populations, injury risk should be understood not as exposure alone, but as a failure of alignment between environmental demands and the brain's capacity to sustain safe behavior.

Neurovascular ageing and functional reserve

WMH, historically termed leukoaraiosis, and BA are common manifestations of neurovascular ageing, reflecting cumulative vascular burden and cerebral small vessel disease [4, 5]. WMH progression and BA trajectories are strongly associated with hypertension, diabetes, smoking, and related cardiometabolic exposures, and may be attenuated through effective risk optimization [6, 7]. These structural changes disrupt large-scale brain networks, particularly fronto–subcortical and parietal systems involved in attention, processing speed, and visuospatial integration.

Importantly, these alterations are frequently subclinical. The clinically relevant concept is not overt impairment but reduced neural reserve—the capacity to maintain performance under increasing cognitive or environmental demand. Individuals with greater structural burden may function adequately under low-demand conditions yet exhibit vulnerability when exposed to complex or high-load environments.

Received: March 9, 2026. **Revised:** May 9, 2026. **Accepted:** May 12, 2026

Exposure–resilience interaction framework

Classical injury epidemiology has long been guided by Haddon's matrix, which emphasizes the interaction between host, agent, and environment in the occurrence of injury [8]. In ageing populations, heterogeneity within the "host" dimension becomes increasingly important.

Structural brain ageing may therefore represent an additional layer of vulnerability that modifies how individuals respond to environmental exposure. This perspective extends classical injury models by incorporating neurovascular resilience as a biological determinant of injury vulnerability in later life.

An exposure–resilience interaction model may help to conceptualize this relationship (Fig. 1). We propose an exposure–resilience interaction framework in which injury risk emerges when environmental demands exceed neurovascular compensatory capacity. This framework suggests that injury vulnerability is threshold-based rather than age-determined.

Specifically, three implications arise. First, similar exposures may yield different outcomes depending on biological reserve. Second, vulnerability may remain latent until environmental demand increases. Third, functional failure may occur in individuals who are otherwise cognitively normal under baseline conditions.

This model provides a biologically grounded explanation for variability in real-world safety performance among older adults.

Empirical evidence linking brain structure and functional safety

Accumulating evidence supports the association between structural brain changes and mobility-related function. WMH burden has been linked to impaired hazard perception and reduced processing speed [9–11]. Region-specific lesions, particularly in parietal and occipital regions, are associated with decreased driving safety performance [11]. Cortical atrophy and WMH have also been associated with behavioral adaptations, including self-regulation of driving exposure [12].

In addition, large-scale screening data from Japan suggest that higher WMH burden is associated with high-risk driving behaviors, such as wrong-way entry and near-miss events on highways [13].

Taken together, these findings indicate that subclinical neurovascular changes may have measurable real-world consequences even in the absence of diagnosed cognitive impairment.

Literature identification approach

This article is based on a narrative synthesis of existing literature. Relevant studies were identified through searches of PubMed and related databases using combinations of keywords including "white matter hyperintensities," "brain atrophy," "driving safety," "injury risk," and "ageing." Priority was given to longitudinal studies, neuroimaging research, and population-based analyses. Additional references were identified through citation tracking.

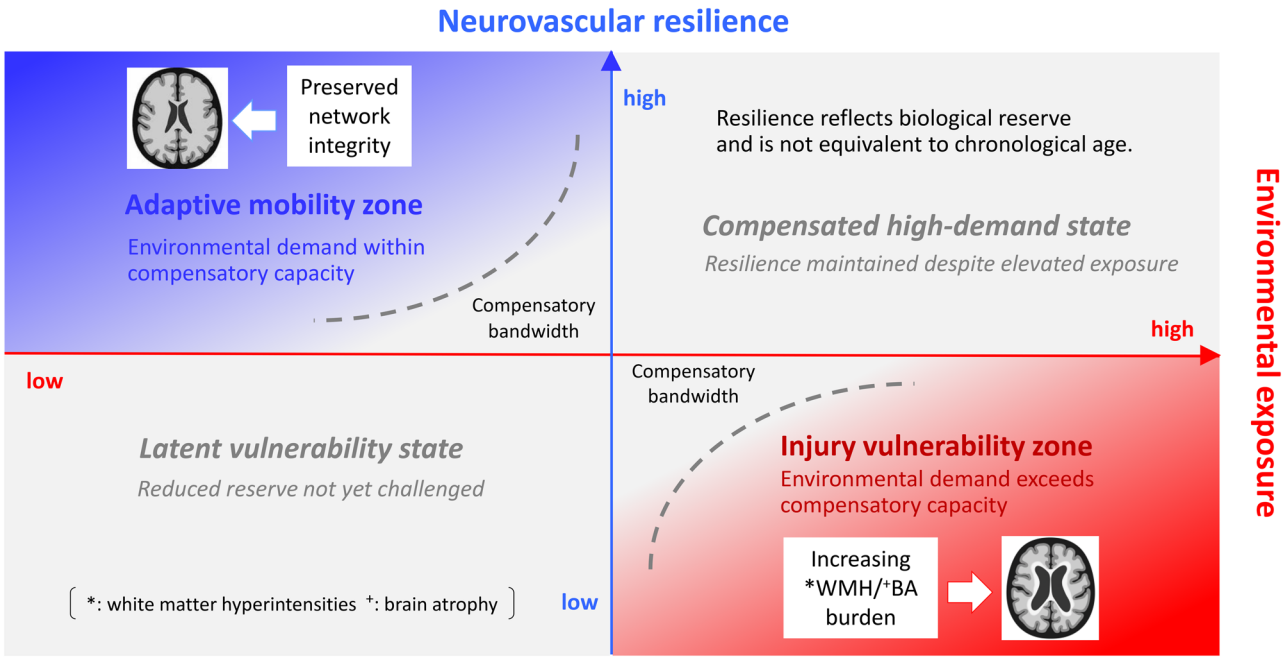

Figure 1 Exposure–resilience interaction model of mobility-related injury in later life. Mobility-related injury is conceptualized as an interaction between environmental exposure and neurovascular resilience. Environmental complexity and cognitive load increase along the horizontal axis, while biological resilience—partly determined by structural brain changes such as white matter hyperintensities (WMH) and brain atrophy (BA)—declines along the vertical axis. Injury vulnerability emerges when environmental demands exceed compensatory neural capacity, representing a threshold phenomenon rather than a direct consequence of chronological ageing. Individuals with preserved resilience may maintain safe mobility even under relatively high-demand conditions, whereas those with reduced reserve may remain asymptomatic until exposed to increased environmental stress. This framework extends classical injury epidemiology by incorporating neurobiological heterogeneity within the host dimension. Structural imaging markers are therefore interpreted as probabilistic indicators of vulnerability rather than deterministic predictors of harm.

The aim was to integrate evidence across disciplines to support a conceptual framework rather than to conduct a formal systematic review.

Implications for screening and prevention: the case of Japan

Japan provides a unique context through its long-standing brain screening programmes ("Brain Dock"), originally developed for stroke prevention. These systems demonstrate the feasibility of identifying structural abnormalities in asymptomatic individuals at scale.

Within a resilience-oriented framework, such infrastructures could support longitudinal monitoring of neurovascular health, integration of vascular risk optimization strategies, and personalized risk communication. Importantly, structural markers should be interpreted as probabilistic indicators within a broader clinical context and should complement, rather than replace, functional assessment.

This approach does not advocate indiscriminate screening or deterministic use of imaging findings, but rather a calibrated integration of biological and functional risk indicators.

Limitations and implementation considerations

Several limitations should be acknowledged. First, the proposed framework is conceptual and does not establish causality. Longitudinal studies linking neuroimaging markers to incident injury outcomes are required.

Second, screening-based approaches carry risks, including overdiagnosis, medicalization, and unintended behavioral restriction such as unjustified driving cessation. These risks necessitate careful ethical governance.

Third, cost and resource implications are substantial, and the cost-effectiveness of large-scale neuroimaging for injury prevention remains uncertain.

Fourth, implementation feasibility varies across healthcare systems, and Japan's infrastructure may not be directly generalizable.

Finally, communication of subclinical risk is complex and may lead to stigma or reduced autonomy if not appropriately managed.

Future directions

Future research should prioritize longitudinal cohort studies linking WMH and BA trajectories with incident injury outcomes, integration of neuroimaging with functional and behavioral data, evaluation of vascular risk interventions, and ethical as well as economic analyses of screening implementation.

Conclusion

Mobility-related injury in later life may be best understood as an interaction between environmental exposure and neurovascular resilience.

Reframing injury risk as a capacity-limited phenomenon—arising from a mismatch between environmental demand and neurobiological reserve—provides a biologically grounded extension of existing models.

Japan's experience illustrates how existing screening infrastructures may support resilience-oriented prevention, provided that implementation prioritizes autonomy, equity, and rigorous evaluation.

As populations age, the challenge of injury prevention is shifting from controlling external hazards to preserving intrinsic capacity. Integrating neurovascular resilience into injury epidemiology represents a conceptual advance that aligns prevention with the biology of ageing. This shift—from exposure control to capacity preservation—may be essential for sustaining mobility, independence, and societal participation in ageing populations.

Author contributions

Kaechang Park (Conceptualization [lead], Data curation [lead], Formal analysis [lead], Funding acquisition [lead], Investigation [lead], Methodology [lead], Project administration [lead], Resources [lead], Software [equal], Supervision [lead], Validation [equal], Visualization [lead], Writing—original draft [lead])

Conflicts of interest

The author declares no conflict of interest.

Funding

This study was supported by JSPS KAKENHI (Grant Nos. 25H00762 and 23K17330).

Data availability

No new data were generated or analyzed in this study.

References

1. World Health Organization. *Global Status Report on Road Safety 2023*. Geneva: WHO, 2023. https://www.who.int/publications/i/item/9789240086517
2. National Police Agency, Japan *Traffic Accidents Situation in 2023*. Tokyo: NPA, 2024.
3. Ministry of Health, Labour and Welfare, Japan. *Vital Statistics of Japan 2022*. Tokyo: MHLW, 2023.
4. Debette S, Markus HS. The clinical importance of white matter hyperintensities on brain magnetic resonance imaging: systematic review and meta-analysis. *BMJ* 2010;**341**:c3666. https://doi.org/10.1136/bmj.c3666
5. Wardlaw JM, Smith C, Dichgans M. Small vessel disease: mechanisms and clinical implications. *Lancet Neurol* 2019;**18**:684–96. https://doi.org/10.1016/S1474-4422(19)30079-1
6. Livingston G, Huntley J, Sommerlad A *et al.* Dementia prevention, intervention, and care: 2020 report of the

lancet commission. *Lancet* 2020;**396**:413–46. https://doi.org/10.1016/S0140-6736(20)30367-6
7. Nasrallah IM, Pajewski NM, Auchus AP *et al.* Association of intensive vs standard blood pressure control with cerebral white matter lesions. *JAMA* 2019;**322**:524–34. https://doi.org/10.1001/jama.2019.10551
8. Haddon W.Jr., Advances in the epidemiology of injuries as a basis for public policy. *Public Health Rep* 1980;**95**:411–21. https://doi.org/7422807][Mismatch
9. Park K, Nakagawa Y, Kumagai Y *et al.* Leukoaraiosis, a common brain magnetic resonance imaging finding, as a predictor of traffic crashes. *PLoS One* 2013;**8**:e57255. https://doi.org/10.1371/journal.pone.0057255
10. Park K, Renge K, Nakagawa Y *et al.* Aging brains degrade driving safety performances of the healthy elderly. *Front Aging Neurosci* 2021;**13**:783717. https://doi.org/10.3389/fnagi.2021.783717
11. Oba H, Park K, Yamashita F *et al.* Parietal and occipital leukoaraiosis due to cerebral ischaemic lesions decrease the driving safety performance of healthy older adults. *Sci Rep* 2022;**12**:21436. https://doi.org/10.1038/s41598-022-25899-4
12. Babulal GM, Chen L, Carr DB *et al.* Cortical atrophy and leukoaraiosis, imaging markers of cerebrovascular small vessel disease, are associated with driving behavior changes among cognitively normal older adults. *J Neurol Sci* 2023;**448**:120616. https://doi.org/10.1016/j.jns.2023.120616
13. Park K, Nakagawa Y. Leukoaraiosis predicts wrong-way entry and near one on highways for healthy drivers. *J Neurol Disord* 2023;**11**:537. https://doi.org/10.4172/2329-6895.11.2.537

 Research Connections 2026 1(2) 1–4
https://doi.org/10.1093/rescon/vmag073
Commentary Health Systems and Models of Care Delivery