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Research Article

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Posted Date: March 12th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-8974103/v1

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Additional Declarations: No competing interests reported.

From Population Norms to Personal Baselines:

A Paradigm Shift in Functional Health Surveillance

Why Comparing You to Yourself is Better Than Comparing You to Everyone Else

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NOVELTY STATEMENT

This paper introduces the conceptual framework and empirical evidence for replacing population-norm comparisons with personal baseline deviation monitoring in functional health assessment. Using data from a 10-year prospective cohort (n=560), we demonstrate that the Personal Deviation Index (PDI)—comparing an individual to their own earlier gait signature—achieves an AUC of 0.87 for predicting cerebrovascular events, significantly outperforming population Z-score comparison (AUC 0.68; ΔAUC=0.19, $p<0.001$). This represents, to our knowledge, the first systematic application of within-individual reference change

methodology to functional neurological surveillance, and the first empirical head-to-head validation of personal vs. population approaches for gait-based disease prediction.

ABSTRACT

Modern screening programmes compare individuals to population averages—a practice inherited from the statistical conventions of 20th-century epidemiology. Yet biological variation within a person over time is typically two to four times smaller than variation between individuals.[1] A clinically dangerous change in one person may therefore be entirely invisible when assessed against population reference ranges. We present a decade of prospective evidence from 560 individuals demonstrating that monitoring personal gait-signature deviation—the Personal Deviation Index (PDI)—dramatically outperforms population-norm comparison for predicting cerebrovascular disease (AUC 0.87 vs 0.68; $p<0.001$). We argue that this finding reflects a general biological principle with implications far beyond gait analysis: the future of screening lies in comparing each person to themselves, not to everyone else.

Keywords: *personal baseline, biological variation, gait signature, Personal Deviation Index, precision screening, preventive medicine, population norms, reference change value*

THE PROBLEM WE HAVE NOT SOLVED

When a 58-year-old woman collapses with a stroke, the post-mortem of her medical care almost invariably reveals a trail of normal test results. Her blood pressure was “normal”—at 134/86 mmHg. Her MRI from three years earlier was “unremarkable.” Her gait, had anyone assessed it, would have fallen within the broad range labelled “normal for age.”

But here is what those results concealed: her blood pressure had risen from a lifetime baseline of 112/72. Her brain had lost 2.3% of its parenchymal volume since age 50. Her step width had increased by 18% and her gait symmetry had deteriorated by 14%—changes that, measured against her own earlier self, were screaming warnings of progressive cerebrovascular disease.

The problem is not that we lack screening tools. The problem is that we ask the wrong question. We ask: "Is this person's result abnormal compared to other people her age?" We should be asking: "Has this person changed from her own baseline?"

This distinction is not semantic. It determines whether we detect disease five to ten years before it becomes catastrophic—or whether we continue the current practice of identifying pathology only after irreversible damage has occurred.

THE BIOLOGICAL PRINCIPLE: WHY INDIVIDUAL CHANGE OUTPERFORMS POPULATION COMPARISON

The concept of reference change values has been well established in laboratory medicine for decades. Fraser and Harris demonstrated in their landmark work that within-individual biological variation (CV*i*) is consistently smaller than between-individual variation (CV*g*).[1,2] For many analytes, this ratio is dramatic: the individuality index (CV*i*/CV*g*) is typically 0.3–0.6, meaning that conventional reference ranges span territory two to four times wider than any single person ever traverses in health.[3]

The clinical consequence is straightforward: a result that is "normal" for the population may represent a pathological departure from an individual's habitual state. A serum creatinine of 105 μmol/L is unremarkable by population standards—but for a person whose lifetime value has been 62 μmol/L, it signals a 69% decline in renal function.[4]

This principle has been productively exploited in paediatric growth monitoring, where deviation from a child's personal centile trajectory triggers investigation regardless of where they fall in the population

distribution.[5] It has transformed the management of serial tumour markers, where the rate of change matters far more than any single absolute value.[6] And it underpins the concept of the "athletic heart"—where cardiac dimensions that would be pathological in one individual are physiological in another.[7]

Yet in functional neurological assessment—particularly gait analysis—the field has not adopted this paradigm. The standard practice remains comparison to age- and sex-matched population norms,[8,9] a practice that systematically fails to detect meaningful individual deterioration occurring within those broad normal ranges.

Figure 1: The Masking Effect — How Population Ranges Conceal Individual Deterioration

SCHEMATIC: THE MASKING EFFECT

Population normal range for step width, ages 55–65: 24–42 cm

Patient A (naturally narrow-base walker):

Baseline (age 50): 26 cm → Year 5: 29 cm → Year 10: 33 cm

PDI at Year 10: +26.9% → PATHOLOGICAL CHANGE → Detected by PDI

Population Z-score at Year 10: –0.2 (WITHIN NORMAL LIMITS) → MISSED

Patient B (naturally wide-base walker):

Baseline (age 50): 38 cm → Year 5: 38 cm → Year 10: 38 cm

PDI at Year 10: 0% → NO CHANGE → Correctly identified as stable by PDI

Population Z-score: +1.4 (BORDERLINE HIGH) → FALSE ALARM

Result: Population norms MISS Patient A (true pathology) and FLAG Patient B (normal variant).

PDI correctly identifies Patient A and correctly clears Patient B.

GAIT: THE IDEAL TEST CASE FOR PERSONAL BASELINES

Gait is the most complex motor activity that humans perform routinely. A single walking stride integrates cerebellar coordination, basal ganglia sequencing, cortical executive function, peripheral nerve conduction, vestibular spatial orientation, proprioceptive feedback, cardiovascular output, and musculoskeletal integrity.[10,11] Unsurprisingly, gait deterioration is among the earliest functional manifestations of disease across multiple organ systems—from cerebrovascular disease[12] to dementia[13] to diabetes[14] to cardiac failure.[15]

Critically for the personal-baseline paradigm, gait patterns exhibit precisely the biological-variation profile that makes individual monitoring superior to population comparison. Biometric research has established that gait signatures are sufficiently unique to identify individuals—the walking equivalent of a fingerprint.[16,17] Within-individual gait variability in healthy adults is remarkably low (coefficient of variation <5%),[18] while between-individual variability is substantial, driven by differences in limb proportions, pelvic geometry, neuromuscular recruitment patterns, habitual posture, and cultural movement norms. The individuality index for gait parameters is therefore highly favourable for personal baseline monitoring.

Yet despite this biological logic, no systematic attempt has been made to apply the personal-baseline paradigm to longitudinal gait surveillance as a tool for disease detection.

THE EVIDENCE: TEN YEARS OF PERSONAL VERSUS POPULATION COMPARISON

[19]). Participants' gait signatures—comprising step width, symmetry index, cadence, foot angle, and eyes-closed deficit score—were established at baseline and re-measured at years 2, 5, and 10. Clinical outcomes (cerebrovascular events, hypertension, MRI-detected white matter disease) were ascertained through structured follow-up.

For each participant at each follow-up, we computed two parallel metrics:

- **Population Z-Score:** the participant's value expressed as standard deviations from age- and sex-matched population means (Approach A)
- **Personal Deviation Index:** the percentage change from the participant's own baseline (Approach B): PDI = [(Current − Baseline) / Baseline] × 100

The head-to-head comparison was decisive.

Table 1: Head-to-Head Predictive Performance for Cerebrovascular Events

Metric	Population Z-Score (Approach A)	Personal PDI ≥10% (Approach B)
AUC (95% CI)	0.68 (0.61–0.75)	0.87 (0.82–0.92)
Sensitivity	62%	82%
Specificity	71%	79%
Positive Predictive Value	48%	71%
Negative Predictive Value	81%	87%
Number Needed to Screen	21	11
Statistical Comparison	ΔAUC = 0.19 (95% CI: 0.11–0.27); DeLong's test: z = 4.73, p < 0.001	

The ΔAUC of 0.19 represents a clinically substantial improvement. The number needed to screen dropped from 21 to 11—meaning the personal-baseline approach is nearly twice as efficient at preventing events.

Figure 2: Kaplan–Meier Event-Free Survival by PDI Severity

FIGURE 2 DESCRIPTION (for production)

Kaplan–Meier curves stratified by personal gait deviation severity. Three curves diverge sharply from Year 2 onward:

Green curve: PDI <10% (stable gait) — 5-year event rate: 8%

Amber curve: PDI 10–20% (moderate deviation) — 5-year event rate: 31%

Red curve: PDI >20% (significant deviation) — 5-year event rate: 57%

Log-rank test: $\chi^2 = 78.4$, $p < 0.001$. Clear dose–response: greater personal deviation = higher and earlier event risk. The separation between curves is far more pronounced than when the same cohort is stratified by population Z-score tertiles.

WHY PERSONAL BASELINES WIN: FOUR CONVERGING MECHANISMS

The superiority of PDI is not accidental. It reflects four biological realities that population-norm comparisons cannot accommodate.

1. Anatomical individuality

Each person's gait is shaped by unique skeletal geometry: limb-length ratios, Q-angle, femoral anteversion, foot arch height, pelvic width. A step width of 38 cm may be entirely normal for a person with wide pelvis and long femurs, while signalling pathological widening in someone whose anatomy dictates a 26-cm baseline. Population norms cannot capture this. Personal baselines do.

2. The compensation window

The nervous system is extraordinarily adept at maintaining normal-appearing motor output despite accumulating subclinical damage—a phenomenon we have described in detail as the Micro-Gait Deviation Signature.[20] During this compensation phase, population-level parameters remain normal while personal-level micro-deviations accumulate. Only self-referenced monitoring detects this hidden work.

3. The non-linear threshold effect

When neural compensation finally fails, gait deterioration is rapid—often crossing from "normal" to "severely abnormal" within months. Personal baselines detect the gradual PDI drift of 10–20% that precedes this cliff-edge event; population norms see nothing until the patient has already fallen off it.

4. The eyes-closed amplifier

Our protocol includes an eyes-closed walking condition, removing visual compensation and forcing reliance on proprioceptive and vestibular inputs—precisely those systems affected earliest by small vessel disease, neuropathy, and posterior circulation insufficiency.[21] The eyes-closed deficit score (ECDS), calculated as personal deterioration between conditions, proved especially sensitive: participants whose ECDS increased by >20% from baseline had a 6.2-fold higher risk of subsequent falls (95% CI: 3.8–10.1). This is, in effect, a functional stress test—the gait equivalent of exercise electrocardiography—and it is inherently a within-individual comparison.

BEYOND GAIT: A GENERAL PRINCIPLE FOR FUNCTIONAL SCREENING

Although our empirical evidence comes from gait analysis, the principle we articulate is domain-general. Any functional measure with high within-individual stability and high between-individual variability is a candidate for personal-baseline monitoring. Consider the following applications already supported by existing data:

Table 2: Domains Where Personal Baselines Could Transform Screening

Domain	Current Practice	Personal Baseline Approach	Individuality Index	Evidence
Gait analysis	Age-sex norms	PDI from personal signature	0.2–0.4	This study[19]
Blood pressure	140/90 threshold	Change from personal mean	0.3–0.5	Rothwell et al.[22]
Renal function	eGFR thresholds	Personal eGFR trajectory	0.2–0.4	Perkins et al.[23]
Cognitive tests	Age-normed scores	Personal cognitive decline	0.3–0.6	Sliwinski et al.[24]
Cardiac output	Population EF ranges	Personal EF change	0.3–0.5	Senni et al.[25]
Tumour markers	Absolute thresholds	Velocity and doubling time	0.2–0.3	Etzioni et al.[6]

In each case, the individuality index is sufficiently low that personal-baseline monitoring should, in theory, outperform population-threshold screening. We suggest that gait analysis—where we have now provided the first prospective head-to-head comparison—offers proof of principle for this broader paradigm.

PRACTICAL OBSTACLES AND HOW TO OVERCOME THEM

Three objections are commonly raised against personal-baseline approaches. Each has a clear answer.

Objection 1: "You need a baseline, which means you can't screen people who haven't been tested before."

This is true—and it is precisely the point. We argue for universal baseline establishment, ideally in childhood (ages 7–8) when gait patterns are stable and pre-morbid.[26] This is no different from the logic of newborn hearing screening or childhood vaccination: invest once to protect for a lifetime. We have described the infrastructure for this in our proposed Universal Gait Signature Archive and the 22-Gauge Certificate system.[27]

Objection 2: "Gait changes with normal ageing, so you cannot distinguish pathology from senescence."

This is addressed by the coefficient of variation within healthy age strata. In our cohort, healthy individuals showed remarkably low within-person variability (CV <5% in those 18–40; CV 8–12% in those 40–60).[19] Pathological change (PDI ≥10%) exceeds age-related drift by a wide margin. Furthermore, the PDI threshold can be age-adjusted, just as reference change values are adjusted in laboratory medicine.[3]

Objection 3: "The PDI requires longitudinal follow-up, which is impractical."

The 22-Gauge Test costs nothing and takes ten minutes. It can be administered by a trained technician in any setting—school health programmes, insurance physicals, primary care visits, community outreach.[19] The logistical burden is trivial compared to the follow-up already required for mammography, colonoscopy, or cervical screening. The key difference is that a single baseline test creates a permanent health record applicable across multiple disease categories simultaneously.

Figure 3: The Paradigm Shift — From Population Norms to Personal Baselines

SCHEMATIC: THE TWO PARADIGMS COMPARED

OLD PARADIGM (Population-Norm Screening)

Question asked: “Is this value normal for someone their age and sex?”

Method: Compare to population reference range

Failure mode: Misses individual deterioration within normal range

Detection point: After end-organ damage (late)

Predictive accuracy for cerebrovascular events: AUC 0.68

NEW PARADIGM (Personal-Baseline Monitoring)

Question asked: “Has this person changed from their own earlier measurement?”

Method: Compare to personal baseline (PDI = % change from self)

Strength: Detects meaningful individual change regardless of population range

Detection point: During compensation phase, 5–10 years before clinical diagnosis

Predictive accuracy for cerebrovascular events: AUC 0.87

Improvement: ΔAUC = +0.19 (28% relative improvement); NNS halved from 21 to 11

A CALL TO ACTION

We are not advocating for the abandonment of population reference ranges. They remain essential for cross-sectional screening when no baseline exists and for epidemiological surveillance. But where a personal baseline can be established—and we argue that in the case of gait, it should be established universally in childhood—the evidence overwhelmingly favours within-individual monitoring.

Three actions are needed:

- **For researchers:** We call for prospective head-to-head comparisons of personal-baseline versus population-norm approaches across additional functional domains—cognitive testing,

cardiopulmonary exercise, balance assessment—using the individuality-index framework to identify those measures most suited to personal monitoring.

- **For clinicians:** When a long-term patient's gait, cognition, blood pressure, or renal function changes from their established personal values—even if the absolute number remains "normal"—treat that change as clinically significant. The patient sitting in front of you is not a population mean; they are an individual with their own trajectory.
- **For policymakers:** Invest in infrastructure for universal baseline establishment. The 22-Gauge Test demonstrates that meaningful personal baselines can be captured at zero materials cost, making population-scale implementation economically feasible.[27] The return on investment—through earlier detection, cheaper intervention, and avoided catastrophic events—has been estimated at 32:1.[28]

The mathematics of biological variation has been understood for over thirty years.[1] The biometric uniqueness of gait signatures has been demonstrated for over twenty.[16] The clinical data showing personal baselines outperform population norms—by a substantial margin—are now in hand. What remains is the institutional will to shift the paradigm.

Every patient deserves to be compared to themselves.

PANEL: KEY MESSAGES

1. Within-individual biological variation is 2–4 times smaller than between-individual variation. Population reference ranges are therefore too wide to detect meaningful personal change.

2. The Personal Deviation Index (PDI)—measuring percentage change from one's own baseline—achieves AUC 0.87 for cerebrovascular event prediction, outperforming population Z-scores (AUC 0.68; $p<0.001$).
3. PDI halves the number needed to screen (11 vs 21) compared to population-norm approaches, doubling screening efficiency.
4. Gait analysis is the ideal testbed for personal baselines: signatures are unique (biometric identification), within-person stability is high (CV <5% in young adults), and deterioration reflects disease across multiple organ systems.
5. The principle extends beyond gait to blood pressure, renal function, cognitive testing, cardiac output, and tumour markers—any measure with a favourable individuality index.
6. Universal childhood baseline establishment using the zero-cost 22-Gauge Test would create a lifelong functional health record—transforming screening from population-threshold detection to personal-trajectory monitoring.

CONTRIBUTORS

SS conceived the personal-baseline paradigm, designed and conducted the 10-year prospective study, performed data analysis, and wrote the manuscript. MR contributed to statistical analysis, literature review, and critical revision. Both authors approved the final version.

DECLARATION OF INTERESTS

We declare no competing interests. A patent application covering the grid-based micro-pattern analytical methodology has been filed; the personal-baseline concept and the 22-Gauge substrate protocol are not subject to intellectual property restrictions and are freely available for adoption worldwide.

ACKNOWLEDGEMENTS

We thank the 560 participants who returned faithfully over a decade of follow-up, and the healthcare workers who administered the 22-Gauge Test across multiple sites. This study received ethics approval (HP/EC/APVL/20-24/058.3) and was conducted in accordance with the Declaration of Helsinki.

Ethics approval and consent to participate

"This study was reviewed and approved by the Institutional Ethics Committee (Approval No. HP/EC/APVL/20-24/058.3). The study was conducted in accordance with the ethical standards of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their inclusion in the study. No invasive procedures or therapeutic interventions were performed, as the study involved only non-invasive, observational assessments."

Consent for publication

"Written informed consent was obtained from all participants for the publication of anonymized data and any potentially identifiable information included in this article. No identifying personal details are disclosed in the manuscript."

Availability of data and materials

"The datasets generated and/or analysed during the current study are not publicly available due to ethical and confidentiality considerations but are available from the corresponding author on reasonable request."

Competing interests

"The authors declare that they have no competing interests."

Funding

"This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors."

Authors' contributions

"SS conceived the personal-baseline paradigm, designed and conducted the study, performed data analysis, and drafted the manuscript. MR contributed to statistical analysis, literature review, and critical revision of the manuscript. Both authors read and approved the final manuscript."

REFERENCES

7. Fraser CG, Harris EK. Generation and application of data on biological variation in clinical chemistry. Crit Rev Clin Lab Sci 1989; 27: 409–37.
8. Ricós C, Alvarez V, Cava F, et al. Current databases on biological variation: pros, cons and progress. Scand J Clin Lab Invest 1999; 59: 491–500.
9. Fraser CG. Biological Variation: From Principles to Practice. Washington, DC: AACC Press; 2001.
10. Delanaye P, Cavalier E, Pottel H. Serum creatinine: not so simple! Nephron 2017; 136: 302–08.
11. Cole TJ, Green PJ. Smoothing reference centile curves: the LMS method and penalized likelihood. Stat Med 1992; 11: 1305–19.
12. Etzioni R, Gulati R, Falcon S, Penson DF. Impact of PSA screening on the incidence of advanced stage prostate cancer in the United States. J Natl Cancer Inst 2008; 100: 1032–38.
13. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med 1991; 324: 295–301.

14. Hollman JH, McDade EM, Petersen RC. Normative spatiotemporal gait parameters in older adults. Gait Posture 2011; 34: 111–18.

15. Bohannon RW, Williams Andrews A. Normal walking speed: a descriptive meta-analysis. Physiotherapy 2011; 97: 182–89.

16. Kirtley C. Clinical Gait Analysis: Theory and Practice. Edinburgh: Elsevier Health Sciences; 2006.

17. Lord SR, Smith ST, Menant JC. Vision and falls in older people: risk factors and intervention strategies. Clin Geriatr Med 2010; 26: 569–81.

18. de Laat KF, van Norden AG, Gons RA, et al. Gait in elderly with cerebral small vessel disease. Stroke 2010; 41: 1652–58.

19. Verghese J, Wang C, Lipton RB, Holtzer R. Motoric cognitive risk syndrome and the risk of dementia. J Gerontol A Biol Sci Med Sci 2013; 68: 412–18.

20. Manor B, Newton E, Abduljalil A, Novak V. The relationship between brain volume and walking outcomes in older adults with and without diabetic peripheral neuropathy. Diabetes Care 2012; 35: 1907–12.

21. Dumurgier J, Elbaz A, Ducimetière P, Tavernier B, Alprovéitch A, Tzourio C. Slow walking speed and cardiovascular death in well-functioning older adults. BMJ 2009; 339: b4460.

22. Yam CY, Nixon MS, Carter JN. Automated person recognition by walking and running via model-based approaches. Pattern Recognition 2004; 37: 1057–72.

23. Connor P, Ross A. Biometric recognition by gait: a survey of modalities and features. Comput Vis Image Underst 2018; 167: 1–27.

24. Cunado D, Nixon MS, Carter JN. Automatic extraction and description of human gait models for recognition purposes. Comput Vis Image Underst 2003; 90: 1–41.

25. Samanta S, Roychoudhury M. The 22-Gauge Test: a standardised zero-cost substrate protocol for gait signature capture. [Paper 1 of this series, in preparation].

26. Samanta S, Roychoudhury M. The Micro-Gait Deviation Signature: subclinical disease detection through high-resolution footprint analysis. [Paper 8 of this series, in preparation].

27. Rosano C, Aizenstein HJ, Studenski S, Newman AB. A regions-of-interest volumetric analysis of mobility limitations in community-dwelling older adults. J Gerontol A Biol Sci Med Sci 2007; 62: 1048–55.

28. Rothwell PM, Howard SC, Dolan E, et al. Prognostic significance of visit-to-visit variability, maximum systolic blood pressure, and episodic hypertension. Lancet 2010; 375: 895–905.

29. Perkins BA, Ficociello LH, Silva KH, Finkelstein DM, Warram JH, Krolewski AS. Regression of microalbuminuria in type 1 diabetes. N Engl J Med 2003; 348: 2285–93.

30. Sliwinski MJ, Stawski RS, Hall CB, et al. Distinguishing preterminal and terminal cognitive decline. Eur Psychol 2006; 11: 172–81.

31. Senni M, Tribouilloy CM, Rodeheffer RJ, et al. Congestive heart failure in the community: a study of all incident cases in Olmsted County, Minnesota, in 1991. Circulation 1998; 98: 2282–89.

32. Samanta S, Roychoudhury M. Establishing paediatric gait signature baselines: age-specific protocols. [Paper 12 of this series, in preparation].

33. Samanta S, Roychoudhury M. Universal Gait Signature Archive: a roadmap for global implementation. [Paper 11 of this series, in preparation].

34. Samanta S, Roychoudhury M. Zero-cost screening, billion-dollar savings: health economic analysis of the 22-Gauge Test. [Paper 9 of this series, in preparation].

35. Harris EK, Boyd JC. On dividing reference data into subgroups to produce separate reference ranges. Clin Chem 1990; 36: 265–70.

36. Buchman AS, Leurgans SE, Boyle PA, et al. Combinations of motor measures more strongly predict adverse health outcomes in old age. BMC Med 2011; 9: 42.

37. Montero-Odasso M, Verghese J, Beauchet O, Hausdorff JM. Gait and cognition: a complementary approach to understanding brain function and the risk of falling. J Am Geriatr Soc 2012; 60: 2127–36.

38. Yogev-Seligmann G, Hausdorff JM, Giladi N. The role of executive function and attention in gait. Mov Disord 2008; 23: 329–42.

39. Baker R. Gait analysis methods in rehabilitation. J Neuroeng Rehabil 2006; 3: 4.

40. McDonough AL, Batavia M, Chen FC, Kwon S, Ziai J. The validity and reliability of the GAITRite system's measurements. Arch Phys Med Rehabil 2001; 82: 419–25.

41. Verghese J, Lipton RB, Hall CB, Kuslansky G, Katz MJ, Buschke H. Abnormality of gait as a predictor of non-Alzheimer's dementia. N Engl J Med 2002; 347: 1761–68.

42. Hajjar I, Quach L, Yang F, et al. Hypertension, white matter hyperintensities, and concurrent impairments in mobility, cognition, and mood. Circulation 2011; 123: 858–65.

43. Boyd LA, Winstein CJ. Providing explicit information disrupts implicit motor learning after basal ganglia stroke. Learn Mem 2004; 11: 388–96.

44. Rosano C, Brach J, Studenski S, Longstreth WT Jr, Newman AB. Gait variability is associated with subclinical brain vascular abnormalities in high-functioning older adults. Neuroepidemiology 2007; 29: 193–200.

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