CCN 2026 · APS COLLEGE OF CLINICAL NEUROPSYCHOLOGISTS

Cerebral small vessel disease and cognitive performance

A community-based cohort study

PRESENTED BY MICHELLE J. FRANCIS

michelle.francis1@monash.edu

M. J. Francis , E. Rowsthorn, B. Sinclair, R. Whiriskey, L. Vivash, K. Franks, T. Chong, I. H. Harding, T. J. O'Brien, M. Law, M. P. Pase & R. J. Stolwyk

Monash University · Monash–Epworth Rehabilitation Research Centre · Turner Institute for Brain & Mental Health

MONASH University

A FAMILIAR SCENARIO

A patient is referred to you for cognitive assessment .

You take a history, review their brain MRI — and there, unexpected, is white matter damage.

But it goes by many names…

- white matter hyperintensities
- small vessel disease
- leukoaraiosis
- white matter lesions
- age-related white matter changes
- white matter disease

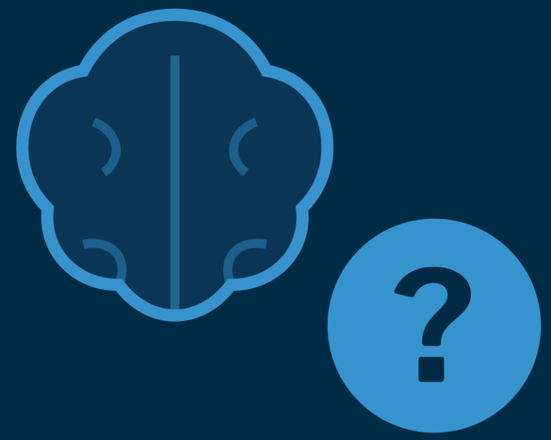
the finding that prompts the question

Does it explain anything about their cognition?

01

SECTION

Background & rationale



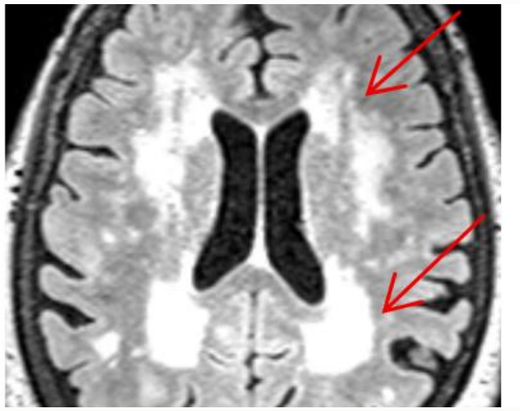
One umbrella term, four markers

“Small vessel disease” is a broad umbrella — many names, several distinct pathologies. We narrowed to the **four markers** sheltering under it that we could measure and examine.

SVD

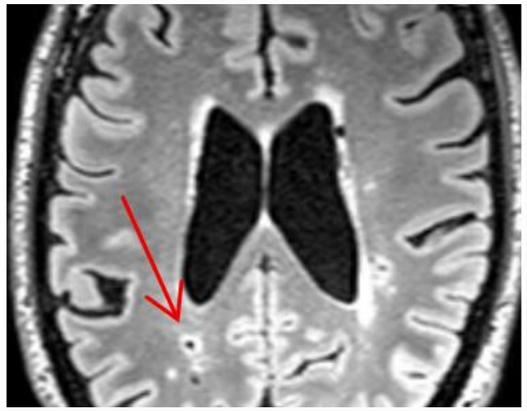
ALSO UNDER THE UMBRELLA (STRIVE): Recent small subcortical infarct · Brain atrophy

OUR FOCUS — FOUR MEASURABLE MARKERS



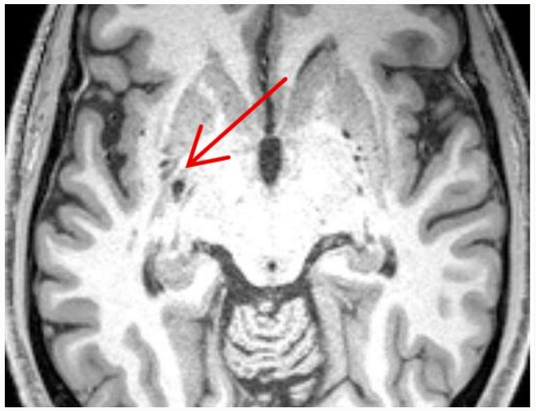
White matter hyperintensities

Diffuse ischaemic injury — periventricular & deep.



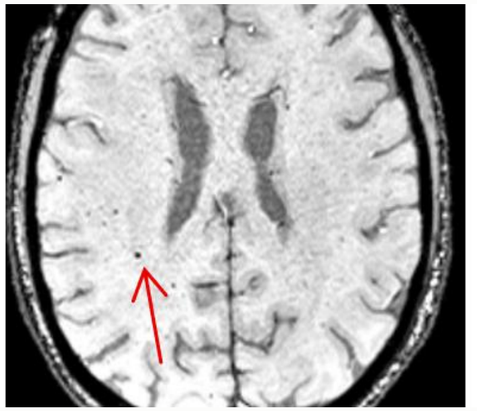
Lacunes

Small subcortical infarcts.



Perivascular spaces

Impaired fluid drainage — basal ganglia.



Cerebral microbleeds

Focal leakage from fragile vessels.

BACKGROUND / 01

Incidental small vessel disease: a clinical dilemma

Increasingly common

Mild WMH are routinely encountered as incidental findings.

No longer benign

Markers of poorer vascular health — raised stroke and dementia risk.

No guidance

No standardised interpretation — reassure or investigate? Uncertainty itself provokes anxiety.

Does mild, incidentally detected SVD actually carry a measurable cognitive cost?

Most evidence comes from clinical samples with moderate–severe disease — **mild SVD in community-dwelling adults remains strikingly under-researched.**

THE WIDER EVIDENCE

Systematic review & meta-analysis

01 What we did

02 What we found

First — what does the wider evidence say?

Aim - To ascertain the cognitive profile of SVD, across multiple cognitive domains at varying levels of severity.

24

studies included

systematic search, PRISMA

16

pooled quantitatively

Only sufficient WMH group data

3-level

random-effects models

multiple effects per study

0–3

Fazekas kept graded

severity not collapsed

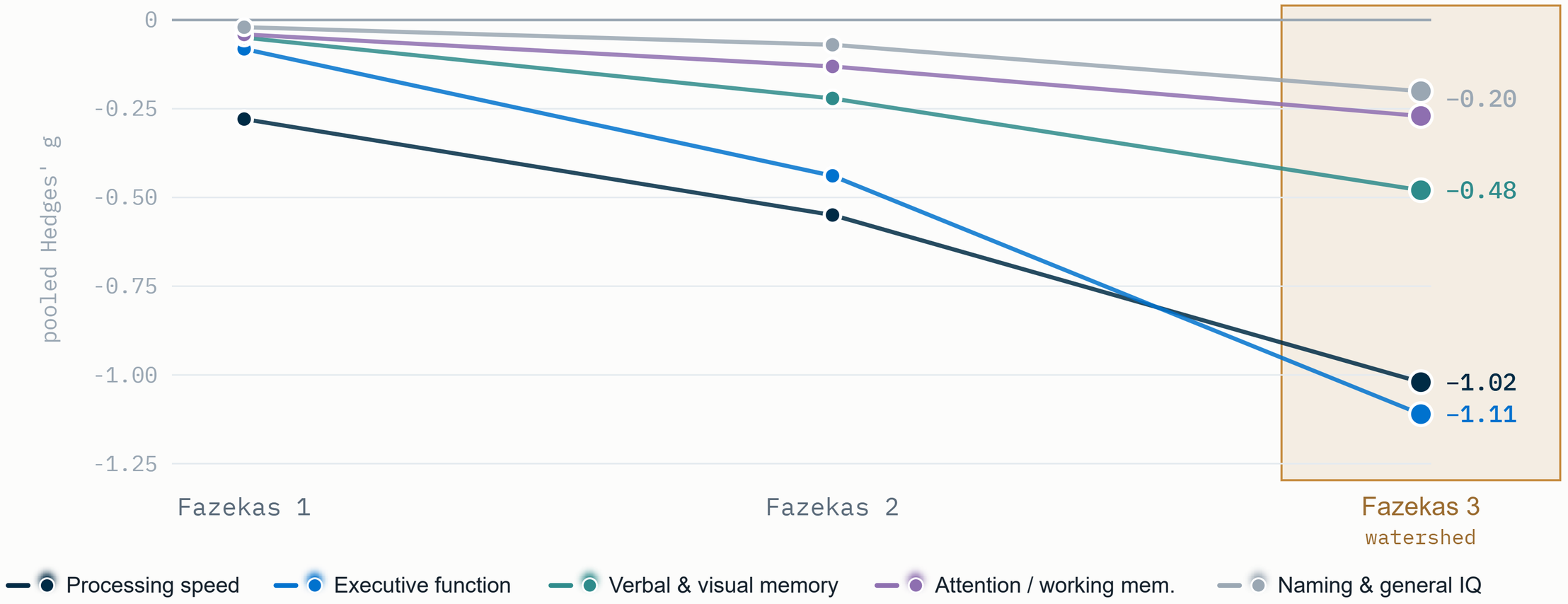
THE QUESTION WE ASKED

Not simply *does SVD affect cognition* **— but which domains , and at what point on the severity spectrum?**

Francis et al (Under review).

✓ A gradient, not a switch

Deficits emerge along the severity spectrum



Gaps in prior work & our rationale

One central gap — and two methodological ones. Each shaped a design choice.

KEY GAP

Mild SVD in community cohorts is under-researched — and the evidence is equivocal

Most data come from clinical samples with advanced disease.

→ **Study a community cohort at the mild end.**

01 **Within-study scores limit interpretability** → Benchmark against **population norms**.

02 **Cognition & SVD rarely anchored to clinical benchmarks** → Anchor **both** to standards (AACN, STRIVE/Fazekas).

Study aims

In a community cohort at the mild end of the spectrum, we set out to:

01

Characterise

cognition across **7 domains** , each of the **4 SVD markers** separately, and combined burden.

02

Interpret

with two complementary metrics — **normative** (vs population) and **ipsative** (vs own premorbid ability).

03

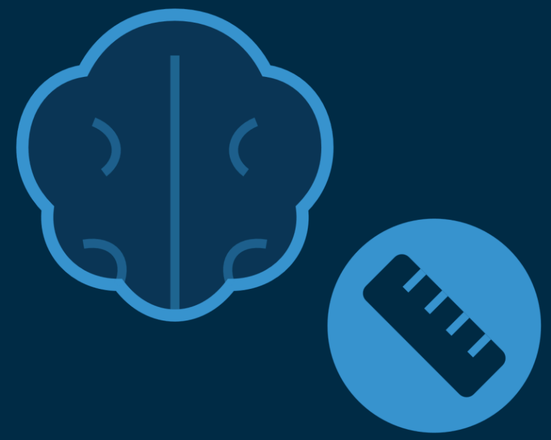
Anchor to benchmarks

Both cognition and SVD mapped to clinical standards — AACN descriptors & STRIVE/Fazekas.

02

SECTION

Methods



METHODS / 01

The BACH community cohort

Community-dwelling adults recruited via advertisement across Australia; cross-sectional cognitive and MRI data, 2022–2024.

241
participants

67.1 yr
mean age (55–80)

17 yr
mean education

51 %
women

Excluded: dementia, major psychiatric or neurological conditions — a relatively healthy, highly educated sample.

SEVEN COGNITIVE DOMAINS ASSESSED

- Visual memory
- Executive function
- Processing speed
- Language
- Social cognition
- Visuospatial
- General cognition

METHODS / 02

How we rated the scans

All markers rated to **STRIVE consensus criteria** (Duering et al., 2023) on a 3T Siemens Prisma (FLAIR · T1-MPRAGE · T2* MEGRE) — **scans manually rated**, each by a method suited to it.

SVD MARKER	SEQUENCE	RATING METHOD
White matter hyperintensities	FLAIR	**Manually rated** on the Fazekas scale 0–3 — PV & deep separately
Lacunes	T1 + FLAIR	**Manually counted** & segmented in MRIcron
Cerebral microbleeds	T2* MEGRE (8th echo)	**Manually counted** & segmented in MRIcron
Perivascular spaces	T1	Doubal grade — automated via validated **PINGU** deep-learning model

ICC > .85

Manually rated and double-rated by trained raters — high inter-rater reliability (> .80 for lacunes & microbleeds).

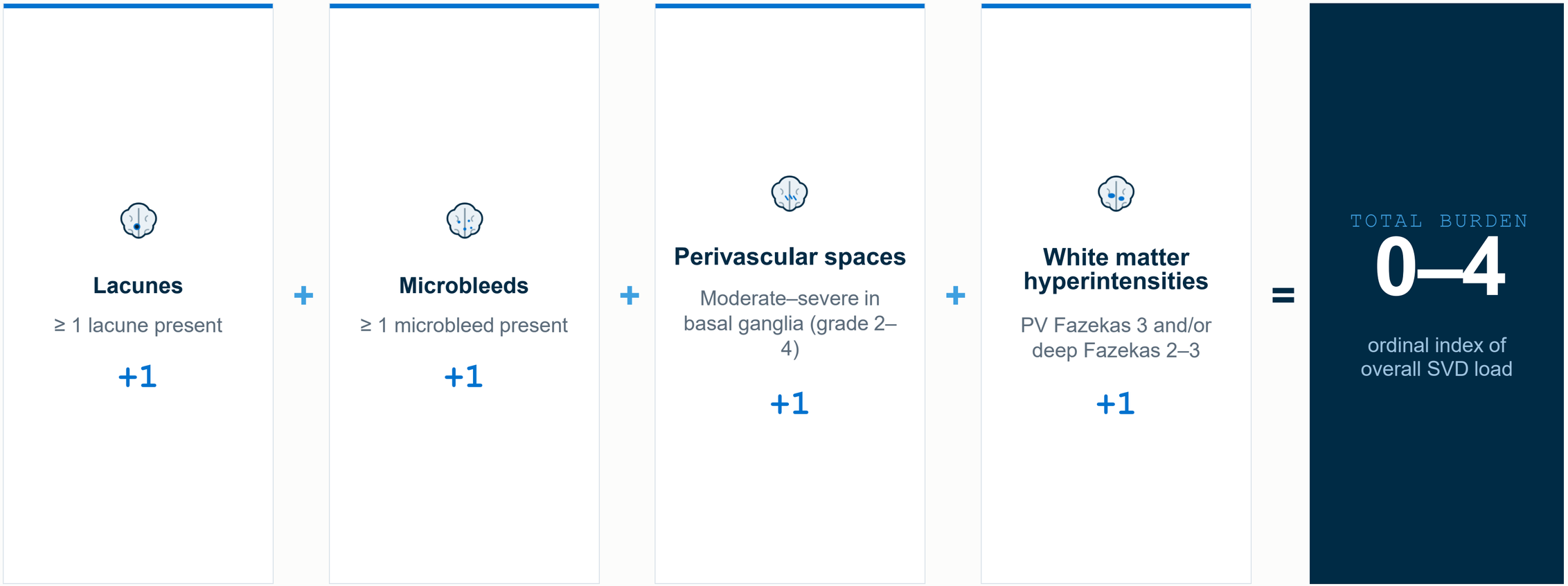
WITH THANKS TO OUR MRI RATERS

Humza Tufail · Reece Nowell · Katie Peterson · Fiona Ellis

METHODS · COMPOSITE MEASURE

Building the composite SVD burden score

One point per marker present at a clinically meaningful threshold — summed to a single 0–4 index (Staals et al., 2015).



Framework: Staals et al. (2015) · STRIVE: Duering et al. (2023) · Fazekas (1987) · Doubal et al. (2010)

METHODS / 03 · KEY APPROACH

Two complementary scoring metrics

NORMATIVE

vs. the population

Referenced to age- and education-adjusted population norms.

benchmark

AACN descriptive labels

IPSATIVE

vs. one's own premorbid self

Current minus estimated premorbid (TOPF) — catches decline norms can mask.

benchmark

Deviation-severity labels

METHODS / 04

Anchored to clinical benchmarks

NORMATIVE — AACN DESCRIPTORS

High Average	z 0.65 – 1.31
Average	z −0.69 – 0.64
Low Average	z −1.37 – −0.70
Below Average & under	z < −1.38

IPSATIVE — DEVIATION SEVERITY

Normal	0 – −0.66
Mild	−0.67 – −1.32
Moderate	−1.33 – −1.99
Severe	≤ −2.00

AACN descriptors: Guilmette et al. (2020)
Deviation severity: Crawford (1992); Lezak et al. (2012)

METHODS / 05

Analytic strategy

Each marker modelled separately

Periventricular WMH (PVWMH), deep WMH (DWMH), microbleeds (CMB) and total burden each regressed on cognition independently — distinct pathologies may act through different mechanisms.

Multiple linear regression

SVD severity → cognition, adjusted for sex. Norms already age/education-adjusted.

56 regression models (4 predictors × 7 domains × 2 metrics), with **Benjamini–Hochberg FDR** correction across the family.

$n \geq 30$ retention criterion per group. Lacunes & PVS (perivascular spaces) met it only at no-pathology — reported **descriptively**.

FDR: Benjamini & Hochberg (1995)

03

SECTION

Results

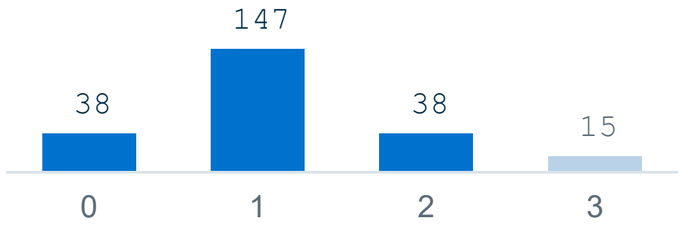
RESULTS / 01 · FIGURE 1 ✓ Overwhelmingly none-to-mild

Distribution of SVD pathology, by marker

Entered regression (n ≥ 30)
Descriptive only / n < 30

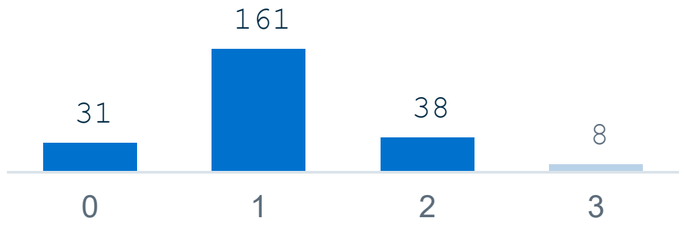
Periventricular WMH

Fazekas grade



Deep WMH

Fazekas grade



DESCRIPTIVE

MRI-visible PVS

Doubal grade (basal ganglia)

Cerebral microbleeds

count

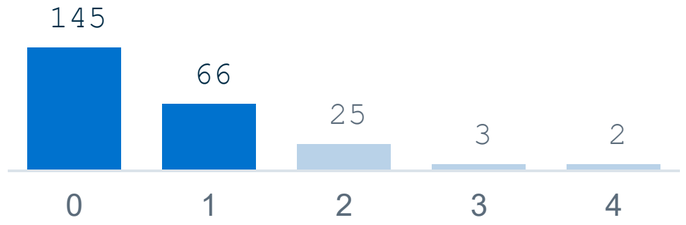
DESCRIPTIVE

Lacunes

count

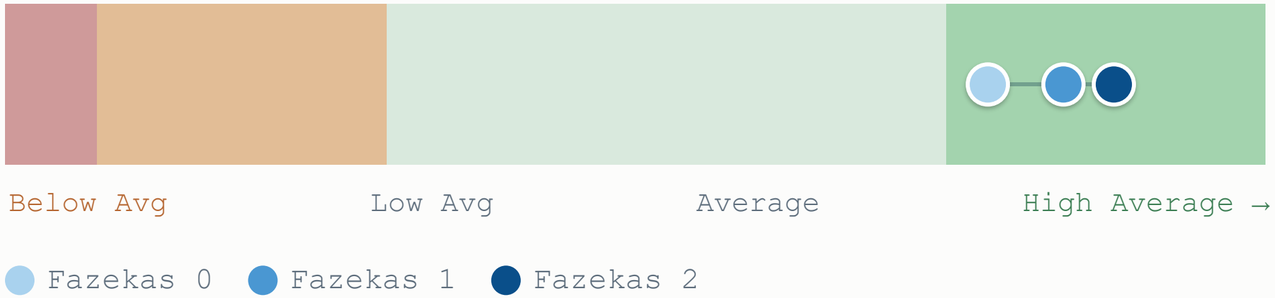
Overall SVD burden

composite score



One cell, up close

Example: **PVWMH × Visual memory** — one dot per severity level on the AACN performance bands.



Each dot = a severity level

light → dark = increasing Fazekas grade.

Read left ↔ right

position = performance band. Further right is better.

Dots clustered & high = no cost

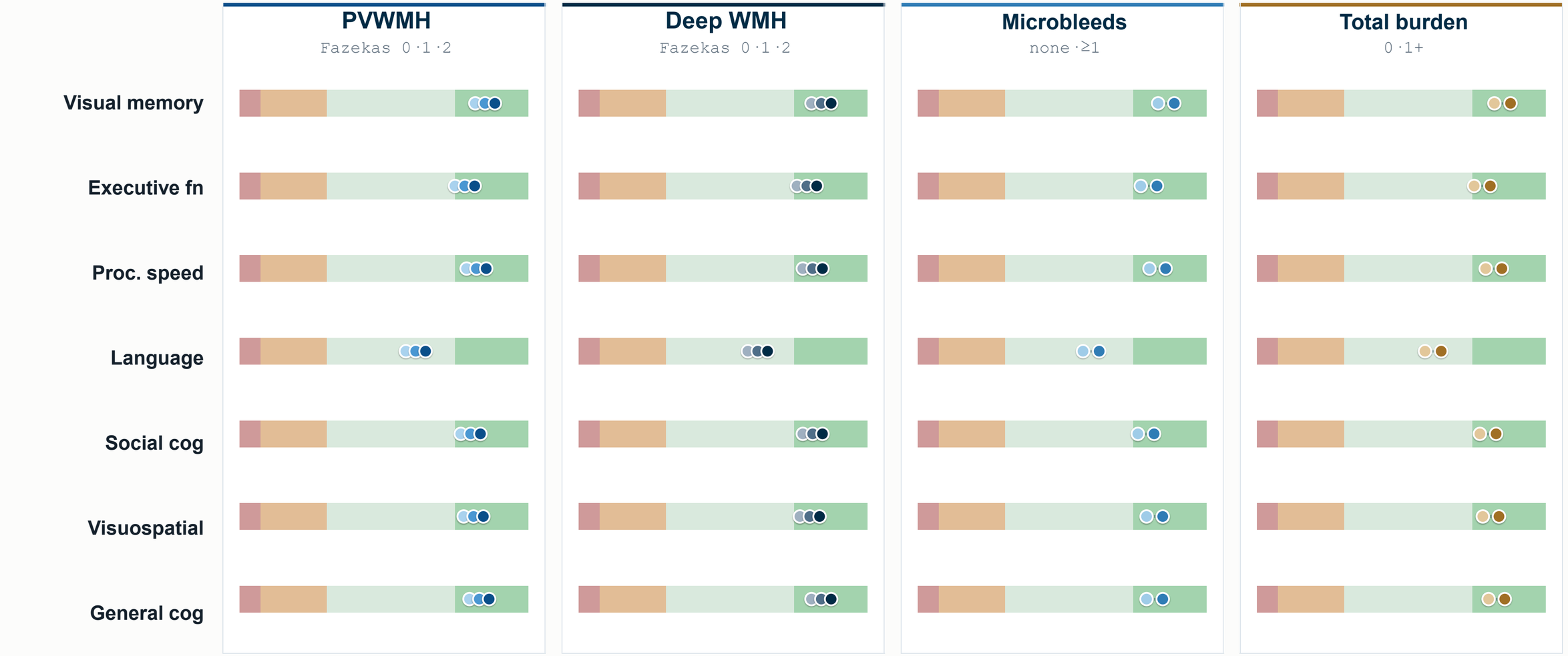
flat across severity → SVD carries no measurable cost here.

Next → the same cell tiled across every domain (rows) × every marker (columns).

✓ Average or better in every cell

Normative: seven domains, by pathology & severity

dots = severity levels (light→dark) per domain on the AACN bands

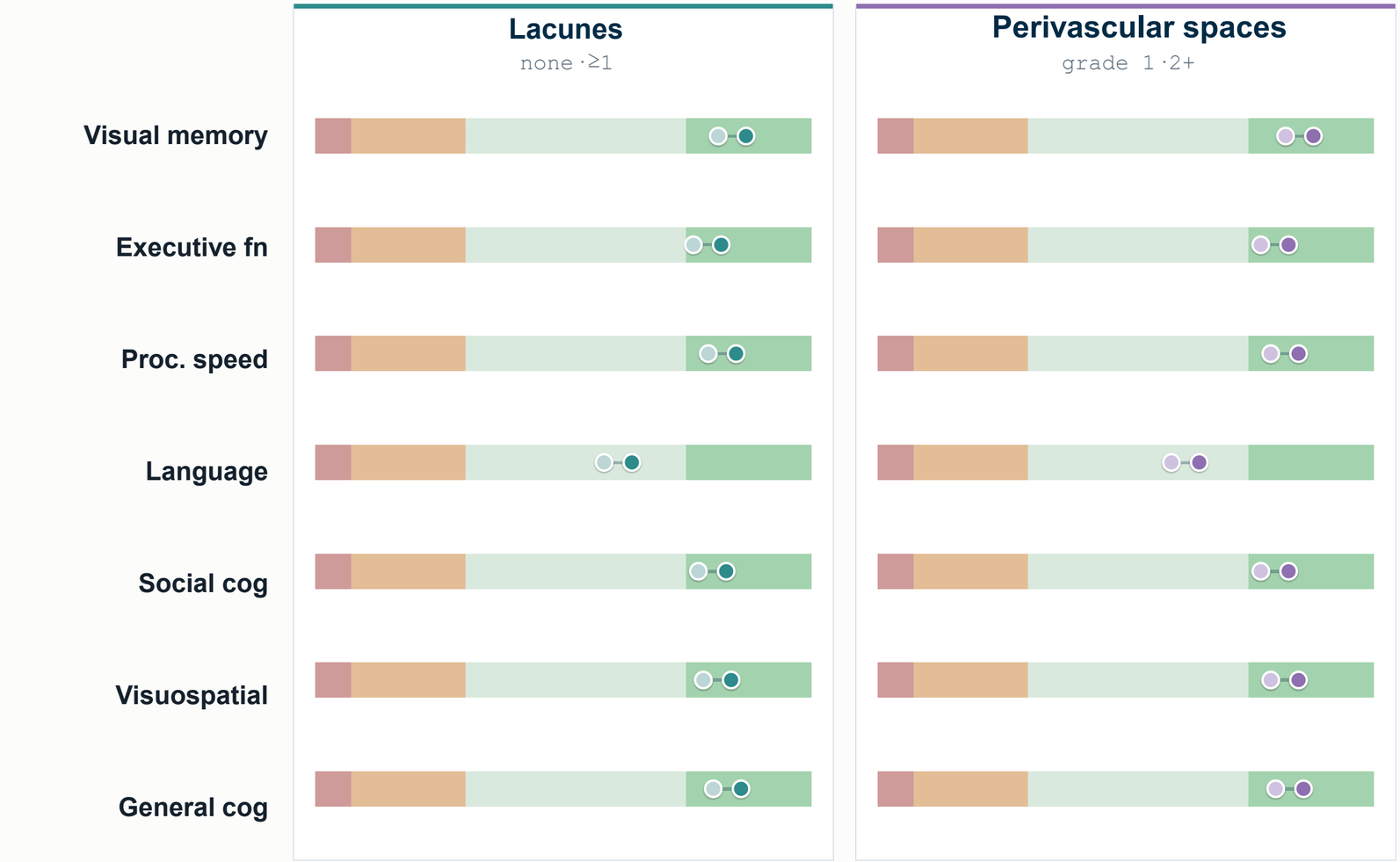


KEY TAKEAWAY No cognitive cost — every marker & domain Average or better

✓ Descriptive markers agree

Normative descriptive markers, by domain & severity

dots = severity levels (light→dark) per domain on the AACN bands

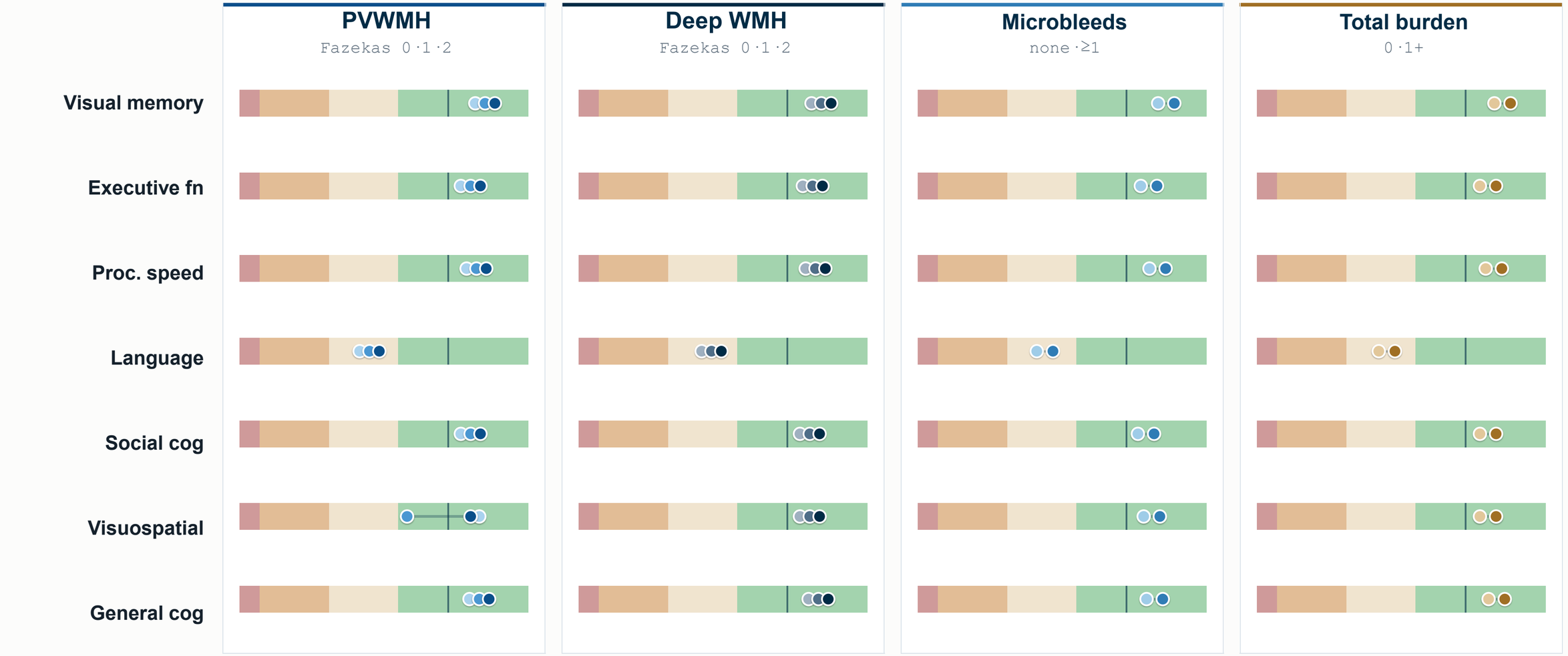


KEY TAKEAWAY Lacunes & PVS agree — no domain below Average

✓ No decline below premorbid

Ipsative: seven domains, by pathology & severity

dots = severity levels (light→dark) per domain | premorbid

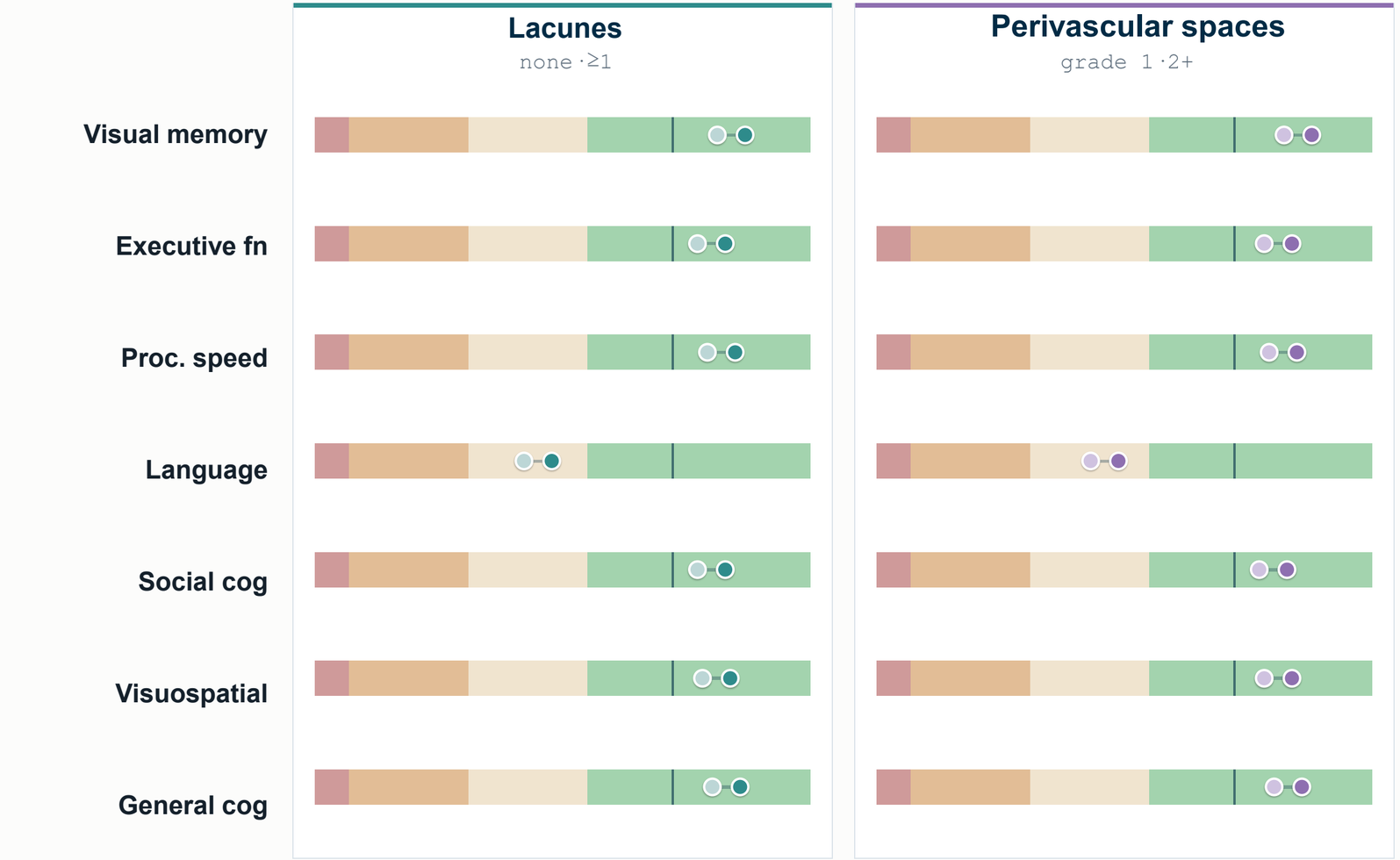


KEY TAKEAWAY No decline below premorbid — bar one tentative PVWMH signal

✓ Descriptive markers agree

Ipsative descriptive markers, by domain & severity

dots = severity levels (light→dark) per domain | premorbid

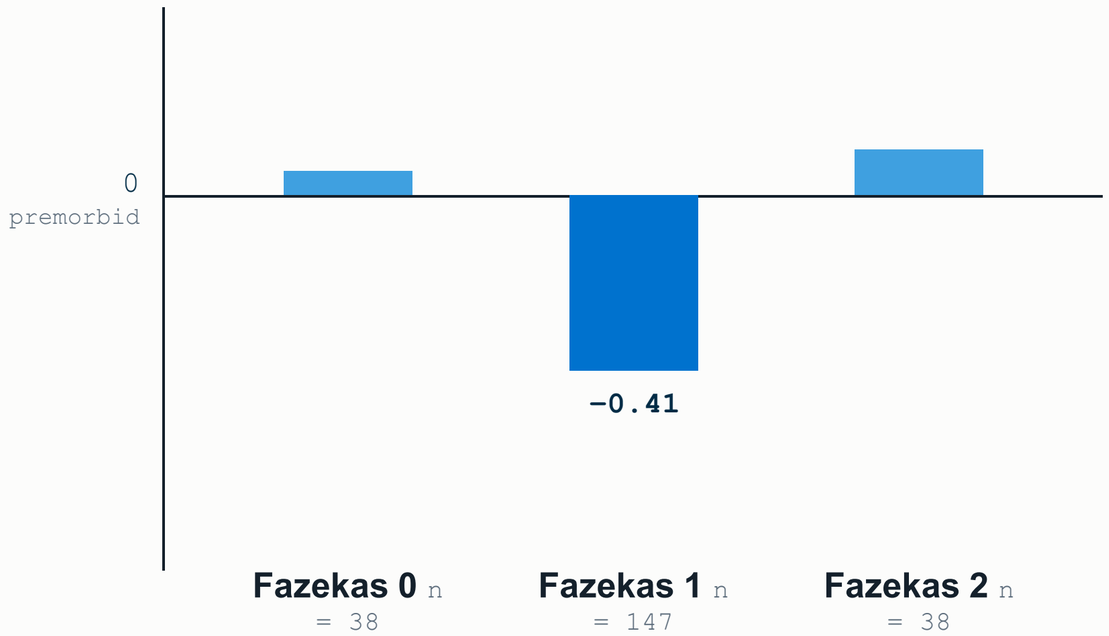


KEY TAKEAWAY No decline below premorbid — for any marker or domain

RESULTS / 04

A single surviving association

Periventricular WMH × visuospatial function (ipsative) — the only result to survive FDR correction.



Mean ipsative visuospatial deviation by PVWMH severity

β **−0.14** p_{FDR} **.042**

The pattern is **non-monotonic** — deviation concentrates in the mild (Fazekas 1) group and returns toward premorbid at moderate. Not a simple dose-response gradient.

Detected only under the ipsative approach, and consistent with prior links between periventricular change and visuospatial difficulty — best regarded as a **tentative signal requiring replication**.

04

SECTION

Discussion & implications

DISCUSSION / 01

Interpreting our results

NORMATIVE

Many means in **High Average** — likely reflecting the sample's above-average education.

→

IPSATIVE

Means shift into the **Normal** deviation range — preserved relative to each person's own baseline.

→

Convergent evidence: mild SVD was not accompanied by meaningful cognitive deficits.

This helps resolve the equivocal prior literature and fits with our systematic review: mixed findings at "mild" SVD likely reflect clinical samples & single-method scoring — deficits emerge reliably only at moderate-to-severe stages, not the mild end.

DISCUSSION / 02

Implications for clinical practice

Don't assume impairment

In a well older adult without complaints, incidental mild SVD ≠ deficit.

Look further if impaired

Where impairment is present, mild SVD alone is unlikely to explain it.

Reassure, but manage risk

Measured reassurance — while still managing vascular risk.

Anchoring to routine benchmarks (Fazekas, AACN) yields findings clinicians can readily interpret — and communicate without alarm.

DISCUSSION / 03

Strengths and limitations

STRENGTHS

- Comprehensive domain-specific battery — not a brief screener.
- Interpreted against established clinical benchmarks (AACN, STRIVE/Fazekas).
- Complementary normative + ipsative design reduces single-method bias.

LIMITATIONS & FUTURE WORK

- None-to-mild range by design — inference limited; higher-burden & longitudinal cohorts needed.
- Ordinal rating scales are coarse — no lesion size, location, or volume.
- High education may confer cognitive reserve that buffers SVD's impact.

DISCUSSION / 04

Future directions

01

Longitudinal follow-up

A single time point speaks to cognition now, not prognosis — track the same people over time.

02

Higher-burden cohorts

Sample moderate-to-severe disease to find where a cognitive cost actually emerges.

03

Replicate the one signal

Test the non-monotonic PVWMH × visuospatial association before reading into it.

04

Extend the framework

Apply this dual-method, benchmark-anchored approach to other incidental findings.

WHERE THIS GOES NEXT

From cognition to everyday function

Francis et al., *to be submitted* — the same BACH cohort, asking what this means for everyday function

FRANCIS ET AL., TO BE SUBMITTED

Beyond cognition — does mild SVD affect everyday functioning?

WHO-ICF · OUTCOME 1

Activity limitations

Difficulties *doing* tasks — mobility, self-care, domestic life.

WHO-ICF · OUTCOME 2

Participation restrictions

Difficulties *being involved* — work, relationships, community life.

DESIGN STRENGTH

Participant & informant report

Self-report alone can miss change when awareness is reduced.

Functioning was also largely preserved at the mild end — converging with the cognitive findings.

Q & A

Thank you

Questions welcome

Cerebral small vessel disease and cognitive performance in a community-based cohort

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PRESENTER & CORRESPONDENCE

Michelle J. Francis

School of Psychological Sciences, Monash University **michelle.francis1@monash.edu**

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