

Ensuring anatomical specificity in lesion network mapping: a multicentre study of 2,950 stroke patients

Marvin Petersen¹, Kaustubh R. Patil^{2,3}, Simon B. Eickhoff^{2,3}, Geert Jan Biessels¹ on behalf of the Meta VCI Map Consortium

Abstract

As many neurobehavioural functions rely on distributed brain networks, anatomically diverse brain lesions can cause highly similar neurobehavioural deficits. Lesion network mapping (LNM) builds on this principle to identify symptom-specific brain networks by mapping focal lesions onto a normative functional connectome. Recent work, however, questioned the anatomical specificity of LNM, showing that commonly used LNM procedures converge on nonspecific properties of the normative connectome rather than symptom-specific networks. Here, we investigated whether anatomically specific LNM can be achieved through appropriate study design and statistical inference. Using a multicenter dataset of 2,950 stroke patients across 12 cohorts, we compared lesion-derived connectivity maps between patients with and without impairment across six cognitive domains. We evaluated group differences using both permutation of symptom labels and parametric two-sample inference, and additionally examined a one-sample approach similar to what was used in the previous critique. Both two-sample approaches yielded distinct, biologically plausible lesion network maps with evident domain-specific features, and limited correspondence to nonspecific connectome properties. Simulation analyses across 10,000 null studies confirmed valid type I error control of the permutation framework. In contrast, the one-sample analyses, which aggregated lesion connectivity maps across affected patients without a control comparison, reproduced the previously described convergence toward nonspecific connectome properties and showed marked similarity across cognitive domains. These findings demonstrate that connectome-driven convergence is not an inherent limitation of LNM, but depends critically on study design: case-control comparisons with appropriate statistical inference ensure anatomical specificity and provide a robust framework for identifying brain networks associated with distinct neurobehavioural deficits.

Author affiliations:

1 University Medical Center Utrecht Brain Center, Utrecht, The Netherlands

2 Institute for Systems Neuroscience, Medical Faculty, Heinrich-Heine University Düsseldorf, Düsseldorf, Germany

3 Institute of Neuroscience and Medicine, Brain and Behaviour (INM-7), Research Center Jülich, Germany

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Correspondence to: m.petersen-28@umcutrecht.nl; g.j.biessels@umcutrecht.nl

Running title: Anatomical specificity in lesion network mapping

Keywords: lesion network mapping; permutation; connectome; post-stroke cognitive impairment; vascular cognitive impairment; neuroimaging

Introduction

That anatomically distinct lesions can cause highly similar neurobehavioural symptoms has been a longstanding enigma in neuroscience. One explanation is that distinct lesions may disrupt brain areas belonging to a common brain network: a lesion anywhere in a network fundamental to a given function will impair that function, regardless of the exact lesion site. Lesion network mapping (LNM) operationalizes this principle, providing an analytical framework that leverages the many-to-one relationship between lesion location and symptom to offer insight into the brain's functional anatomy from a network perspective.¹ By projecting lesion locations onto a normative functional connectome, LNM generates lesion network maps that help to identify the distributed neural circuits underlying clinical deficits.² This premise has contributed to its rapid adoption across neurology and psychiatry.

However, recent work has raised concerns about the anatomical specificity of LNM.³ Across multiple analyses, lesion network maps derived from clinically distinct symptoms were observed to exhibit substantial similarity, suggesting that commonly used LNM procedures may preferentially recover general properties of the underlying normative connectome rather than symptom-specific brain networks. The proposed explanation was that lesion-derived connectivity maps are inherently shaped by the fixed connectivity architecture onto which all lesions are projected, introducing a connectome-driven bias whereby features such as regional hubness or degree dominate statistical maps. These concerns have been raised across different implementations of LNM, including approaches that aggregate lesion-derived connectivity maps across affected patients (often referred to as *sensitivity analyses*) as well as symptom-anchored analyses relating patient-level connectivity profiles to clinical measures or comparing them between cases and controls (*specificity analyses*).³ While these observations have prompted important questions regarding the specificity and interpretability of LNM, the extent to which they generalize across different analytical frameworks remains an active topic of debate.

These considerations prompted us to revisit the methodological approach we had used in earlier work, in which we applied LNM to white matter hyperintensity data of memory clinic patients.⁴ We found that in contrast to the critique,³ our statistical approach already accounts for this bias through its use of permutation of symptom labels. Label permutation preserves the structured dependence induced by the normative connectome while breaking the relationship between lesion-derived connectivity maps and clinical phenotype. Consequently, connectome-driven convergence is incorporated into the null distribution, allowing only signals exceeding this shared background signal to be identified as significant. We originally adopted this approach because permutation-based inference is well established in neuroimaging and has already been

implemented in many lesion network mapping studies.^{5–8} However, the extent to which this inferential framework – or, more generally, symptom-anchored case-control analyses – mitigates the recently described convergence has not been evaluated systematically.

Hence, the central unresolved question we address here is whether appropriate study design and statistical inference can distinguish nonspecific connectome properties from symptom-specific network effects.⁹ We therefore tested whether anatomically specific lesion network maps can be achieved within a symptom-anchored case-control framework. To do so, we used a multicenter dataset of 2,950 patients with acute ischemic stroke from the Meta VCI Map Consortium. Acute ischemic stroke provides a prototypical model of focal lesions which underpin much of the lesion network mapping literature. We compared three analytical strategies, which have been previously used in this context: 1) label permutation and 2) parametric two-sample inference, both comparing cognitively impaired and unimpaired patients in a case-control design, and 3) one-sample aggregation of lesion-derived connectivity maps only taking impaired patients into account. We further evaluated the validity of the label permutation framework through simulation analyses and assessed whether the resulting lesion network maps capture information beyond focal lesion location by comparison with voxel-based lesion-symptom mapping. By systematically comparing these approaches, we show that symptom-anchored case-control analyses yield anatomically specific and biologically plausible lesion network maps with both label permutation-based and parametric inference, whereas one-sample analyses reproduce the connectome-driven convergence described previously.³ These findings demonstrate that connectome-driven convergence is not an inherent limitation of lesion network mapping but depends critically on study design and statistical inference.

Materials and methods

We analyzed data from 2,950 stroke patients across 12 post-stroke cognitive impairment cohorts of the Meta VCI Map Consortium, all with acute ischemic stroke lesion segmentations and harmonized cognitive assessment.¹⁰

Cognitive status was classified as impaired or unimpaired for six domains – attention/executive function, processing speed, language, verbal memory, visuospatial functions, and visuospatial memory. Each domain aggregates performance across several standardized neuropsychological tests, grouped according to the harmonization procedure established within the Meta VCI Map Consortium, with impairment defined as performance below the fifth percentile of local normative data within each contributing cohort. The domains are therefore multi-test composites rather than single isolated cognitive processes; the full per-test operationalization is given in the source description of the dataset.¹⁰ Cognitive impairment labels provided the basis for all statistical comparisons.

Acute lesions were segmented within each cohort and normalized to MNI152Nlin6Asym space. These normalized lesion masks were used to compute lesion-derived functional connectivity maps, which estimate the functional networks connected to each patient's lesion. The connectivity maps were computed with Lead-DBS using the GSP1000 normative functional connectome.^{11,12}

Statistical analyses were conducted with Permutation Analysis of Linear Models (PALM) using model specifications matched as closely as possible across inference approaches.¹³ For each cognitive domain, inference was performed using three approaches: (i) symptom-label permutation testing contrasting cognitively impaired and unimpaired patients, (ii) a parametric two-sample t-test contrasting cognitively impaired and unimpaired patients, and (iii) a one-sample t-test restricted to cognitively impaired patients. Each voxelwise statistical map was corrected for multiple comparisons using the false discovery rate (FDR). Label permutation-based analyses employed 1,000 permutations with threshold-free cluster enhancement (TFCE) and p-values were calculated as the proportion of permuted test statistics equalling or exceeding the observed statistic (two-sided).¹³

Because patients were pooled across the 12 cohorts, cohort was modeled as a fixed effect, and label permutation-based inference was performed within cohort using exchangeability blocks – that is, impairment labels were permuted only among patients from the same cohort. This restricts the permutation null to respect the multi-cohort structure of the data. Primary label permutation and parametric group analyses were performed without further covariates beyond cohort. Models additionally adjusting for lesion volume and NIHSS are reported in the supplementary materials. Missing NIHSS values (n = 296) were median-imputed. The one-sample t-tests were not adjusted for covariates.

To quantify pairwise similarity across cognitive domains, we computed spatial correlations of t-statistic maps and Dice overlap of thresholded significance maps. Pairwise similarity with the degree map of the GSP1000 connectome was also quantified.

Domain-specific effects were anatomically localized by identifying the five strongest positive local maxima in each t-statistic map, constrained to be at least 20 mm apart. Peaks were assigned to atlas parcels (Schaefer1000 cortex, Tian54 subcortex, and Buckner cerebellum), with peaks located in white matter assigned to the nearest grey matter parcel.^{14–16}

To assess type I error control of the label permutation framework under the null hypothesis, we conducted simulation analyses across 10,000 null studies using both synthetic lesions and real lesion masks from the Meta VCI Map cohort. Under a valid null, the proportion of significant ROIs ($p < 0.05$) should approximate the nominal false positive rate ($\approx 5\%$). We quantified the empirical false positive rate and characterized its spatial distribution and associations with nonspecific connectome properties (degree and PC1). Details are presented in the supplementary materials.

To determine whether label permutation-based LNM captures information beyond focal lesion location, we further compared the resulting networks with voxel-based lesion-symptom mapping (VLSM) performed on the same dataset taking binary lesion masks as input. VLSM was performed within the same statistical framework as LNM: a voxelwise general linear model in PALM contrasting impaired and unimpaired patients for each cognitive domain, with symptom-label permutation (1,000 permutations, two-sided), TFCE, and FDR correction. Matching previous studies, VLSM was adjusted for lesion volume.¹⁰

Each contributing cohort obtained local ethics approval and written informed consent, as previously described.^{10,17}

Results

Lesion coverage in the dataset was high, with 86.9% of brain voxels affected in at least five patients (for a lesion distribution heatmap see **Supplementary figure S1**). Average pairwise co-occurrence of cognitive domain impairment was low (mean $\phi = 0.29 \pm 0.10$, mean Jaccard index = 0.26 ± 0.08 ; **Supplementary figure S2**), showing that patients had diverse deficits.

Label permutation yielded statistically significant lesion network maps for five of the six domains (**Figure 1**; for unthresholded maps see **Supplementary figures S3** and **S4**): attention/executive function, processing speed, language, verbal memory, and visuospatial functions; visuospatial memory showed no significant group differences under the permutation null. Applying parametric two-sample t-tests to the identical data produced highly comparable results, recovering the same per domain lesion network maps as the label permutation-based analysis (**Supplementary figures S5–S7**, mean Dice = 0.85 ± 0.05). Additionally adjusting for lesion volume and NIHSS also resulted in highly comparable lesion network maps, although visuospatial functions no longer reached significance (**Supplementary figure S8**). For parsimony, we report the label permutation-based model without lesion volume and NIHSS as the primary analysis in the following paragraphs.

Across domains, the label permutation-based maps overlapped only weakly with the connectome's degree map (mean Dice = 0.20 ± 0.07 , mean $r = 0.28 \pm 0.24$) and were only

moderately similar to one another (mean Dice = 0.50 ± 0.11 , mean $r = 0.55 \pm 0.28$) (**Figure 2**), showing that the identified networks did not converge onto a shared, domain-general pattern driven by nonspecific connectome structure. This anatomical specificity was most readily apparent for language. The identified language network highlighted left-lateralized perisylvian regions, including left inferior frontal and temporal cortex, aligning with a priori expectations for language circuitry.

Peaks of the t-statistics underlying both label permutation and parametric inference differed across cognitive domains, underscoring map specificity (**Figure 3**). The attention/executive function, processing speed, language, and verbal memory maps were left-lateralized, with all top peaks confined to the left hemisphere, whereas visuospatial functions peaked exclusively in the right hemisphere; visuospatial memory peaks were bilaterally distributed. In every domain, the strongest peak fell in white matter proximal to a cortical or subcortical parcel. Attention/executive function peaked in white matter proximal to the left posterior parietal cortex (dorsal attention network, $t = +8.3$), the same area that led the language map ($t = +7.5$). Processing speed peaked in white matter proximal to the left lateral PFC (control network, $t = +4.3$), verbal memory in white matter proximal to the left caudate ($t = +7.7$), and visuospatial functions in white matter proximal to the right lateral PFC (control network, $t = +5.4$).

Simulation analyses confirmed valid type I error control under permutation testing across 10,000 null studies. Combining lesion masks with randomly assigned symptom labels, the observed mean false positive rate was close to the nominal 5% (5.0% for synthetic and 5.1% for real lesions; **Supplementary figure S9**). In these simulations, lesion network maps computed without label permutation aligned with connectome degree and PC1, reproducing the convergence described previously.³ Applying label permutation markedly reduced map alignment with connectome degree and PC1.

In contrast to case-control designs, in the same dataset the one-sample t-tests of cases generated maps that corresponded to a higher extent with the normative degree map (mean $r = 0.69 \pm 0.02$; mean Dice = 0.31 ± 0.006) and showed high similarity across the different cognitive domains (mean $r = 0.98 \pm 0.01$; mean Dice = 0.93 ± 0.03 ; **Supplementary figures S10 and S11**). For example, the one-sample t-test language map lacked the expected left lateralization and aligned with the normative degree map ($r = 0.70$). Overlap between label permutation-based and one-sample t-test significance maps was moderate (mean Dice = 0.56 ± 0.07 , **Supplementary figure S12**).

Label permutation-based lesion network maps overlapped only partially with voxel-based lesion-symptom mapping (VLSM) maps for the same cognitive outcomes (mean Dice = 0.37 ± 0.11) (**Supplementary figures S13 and S14**), indicating that label permutation-based LNM captures network-level information beyond what focal lesion location alone can explain. The VLSM maps were moderately similar across domains (**Supplementary figure S15**).

Discussion

In this multicenter study of 2,950 stroke patients, we demonstrated that lesion network mapping produces anatomically specific and biologically plausible network maps when performed within a symptom-anchored case-control framework. Lesion network maps derived from comparisons between cognitively impaired and unimpaired patients showed anatomically plausible patterns such as the expected left-lateralized language network, only moderate similarity across cognitive domains, and little correspondence with nonspecific connectome properties. Importantly, this specificity was achieved using non-parametric label permutation as well as with parametric two-sample inference. In contrast, one-sample analyses that aggregated lesion connectivity maps from impaired patients alone reproduced the strong convergence across symptoms and the alignment with connectome hubs described previously.³

Our findings help reconcile recent methodological concerns regarding lesion network mapping. It has recently been suggested that the reported convergence of lesion network maps primarily reflects analyses that summarize lesion connectivity across affected patients (often termed sensitivity analyses), rather than analyses that explicitly compare patients with and without a symptom (specificity analyses).¹⁸ Our results support this distinction and suggest that the recently described convergence is not an inherent property of lesion network mapping itself, but depends critically on study design and statistical inference. Lesion-derived connectivity maps share variance because all lesions are projected onto the same normative connectome. However, this shared variance is present in both patients with and without a given symptom. Consequently, comparing lesion connectivity between clinically defined groups allows this shared background signal to be distinguished from symptom-related effects. By contrast, one-sample analyses that simply aggregate lesion connectivity maps across affected patients lack such a reference and therefore converge toward highly connected regions. Consistent with this interpretation, one-sample analyses in our dataset produced highly similar maps across cognitive domains with correspondence to general connectome properties, whereas case-control analyses yielded more distinct and biologically plausible networks.

An important observation is that both permutation of symptom labels and parametric two-sample testing produced specific lesion network maps in the present dataset. The concordance between these approaches suggests that the primary determinant of anatomical specificity is the symptom-anchored case-control design itself rather than the inference approach. Although the resulting maps were highly similar, the two approaches differ in their underlying statistical assumptions. Parametric inference assumes independent observations, whereas lesion-derived connectivity maps are inherently correlated through the shared normative connectome. Whether this assumption is adequately met is not guaranteed for lesion-derived connectivity maps, and its adequacy in any given setting cannot be established from the present data alone. Permutation-based inference, which has been widely adopted in lesion network mapping studies, provides an alternative framework that does not require this independence assumption.¹³ The accompanying simulation analyses further support the validity of label permutation by demonstrating nominal type I error control across 10,000 null studies while reducing alignment with dominant connectome properties.

These findings also have important implications for interpreting the existing lesion network mapping literature. Symptom-anchored case-control analyses – whether analyzed with label permutation or parametric inference – are not a new methodological proposal but have already been implemented in many previous LNM studies.^{5–8,19–23} Our results therefore provide empirical support for this body of work, suggesting that these studies are unlikely to be explained solely by nonspecific connectome properties. More broadly, our findings complement recent methodological efforts to evaluate lesion network mapping across different study designs, including the development of dedicated null models for case-only analyses.⁹ Together, these developments indicate that connectome-driven convergence can be addressed using principled study design and statistical inference rather than representing an inherent limitation of the technique.^{9,18,24}

Our additional analyses further support the biological relevance of the identified networks. Label permutation-based lesion network maps overlapped only partially with voxel-based lesion-symptom mapping, indicating that the former capture network-level information beyond focal lesion location alone rather than simply reproducing conventional lesion-symptom associations. These results align with previous work showing that LNM significantly improves individual-level symptom prediction compared to pure lesion location – something that would not be possible if patient-level lesion connectivity maps carried only nonspecific or lesion location information.^{4,25,26} At the same time, the absence of significant findings for visuospatial memory illustrates the increased stringency of symptom-anchored inference. The analysis appropriately failed to identify a robust network once connectome-driven variance had been accounted for. Together, these observations suggest that greater anatomical specificity may come at the cost of reduced sensitivity for weaker or more heterogeneous clinical effects.

Several limitations should be considered. Our analyses focused on broad cognitive domains that comprise multiple cognitive processes and frequently co-occur after stroke. Residual similarity between lesion network maps may therefore reflect shared behavioural variance in addition to shared connectome structure. Relatedly, cognitive outcomes were based on post-hoc harmonized data and dichotomized into impaired and unimpaired groups rather than analyzed as continuous measures, with single-time-point assessment, differential recovery across domains, and domain-specific floor or missing data effects also affecting the anatomical specificity attainable.

In conclusion, our findings indicate that the convergence recently described in lesion network mapping, i.e., the tendency of maps derived from clinically distinct symptoms to resemble one another and to align with generic connectome properties, is not an inherent limitation of the technique but depends critically on study design and statistical inference. Symptom-anchored case-control analyses can preserve anatomical specificity, and label permutation provides a principled non-parametric framework that explicitly accounts for the dependence structure induced by the normative connectome. These results identify a practical methodological framework for obtaining biologically meaningful network maps and provide reassurance for the previous LNM studies that already adopted this approach.

Data availability

The domain-level lesion network maps are provided on Open Science Framework (<https://osf.io/puq36>). The individual-level data that support the findings of this study are available from the project leads on reasonable request (<https://metavcimap.org/group/become-a-member/>). Restrictions related to privacy and personal data sharing regulations and informed consent may apply. The analysis code is provided on GitHub (https://github.com/umcu-VCI-group/2026_petersen-revisiting_lesion_network_mapping).

Funding

This project is part of the Timely, Accurate, and Personalized Diagnosis of Dementia (TAP-dementia) program, which receives funding from ZonMw (#10510032120003) in the context of Onderzoeksprogramma Dementie, which is part of the Dutch National Dementia Strategy. Further funding comes from the German Research Foundation (Deutsche Forschungsgemeinschaft, DFG) – 579029650 (M.P.).

Competing interests

The authors report no competing interests.

Author contributions

Each author has made a significant contribution to the manuscript and all authors read and approved its final version. M.P.: Conceptualization, Funding, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Visualization, Writing—original draft, Writing – review & editing; K.P.: Investigation, Writing—review & editing; S.B.E.: Investigation, Writing – review & editing; G.J.B.: Conceptualization, Funding, Investigation, Resources, Supervision, Writing – review & editing. Contributors from the Meta VCI Map Consortium are listed in Appendix 1.

Supplementary material

Supplementary material is available at *Brain* online.

Appendix 1

The authors acknowledge the contributions of all researchers who provided resources to the Meta VCI Map post-stroke cognitive impairment initiative: Nick Weaver, PhD; J Matthijs Biesbroek, PhD; Hugo J Kuijf, PhD; Hugo P Aben, MD; Jill Abrigo, MD; Prof Hee-Joon Bae, PhD; Mélanie Barbay, MD; Jonathan G Best, MD; Prof Régis Bordet, PhD; Francesca M

Chappell, PhD; Christopher P L H Chen, MD; Prof Martin Dichgans, MD; Thibaut Dondaine, PhD; Jule Filler; Ruben S van der Giessen, PhD; Prof Olivier Godefroy, PhD; Bibek Gyanwali, MD; Olivia K L Hamilton, MSc; Saima Hilal, PhD; Irene M C Huenges Wajer, PhD; Prof Yeonwook Kang, PhD; Prof L Jaap Kappelle, PhD; Beom Joon Kim, PhD; Sebastian Köhler, PhD; Paul L M de Kort, PhD; Prof Peter J Koudstaal, PhD; Gregory Kuchcinski, MD; Bonnie Y K Lam, PhD; Prof Byung-Chul Lee, PhD; Keon-Joo Lee, MD; Jae-Sung Lim, MD; Renaud Lopes, PhD; Stephen D J Makin, PhD; Anne-Marie Mendyk, RN; Prof Vincent C T Mok, MD; Mi Sun Oh, MD; Prof Robert J van Oostenbrugge, PhD; Martine Roussel, PhD; Lin Shi, PhD; Julie Staals, PhD; Maria del C Valdés-Hernández, PhD; Narayanaswamy Venketasubramanian, MBBS; Prof Frans R J Verhey, PhD; Prof Joanna M Wardlaw, PhD; Prof David J Werring, PhD; Xu Xin, PhD; Prof Kyung-Ho Yu, PhD; Martine J E van Zandvoort, PhD; Lei Zhao, PhD.

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Figure legends

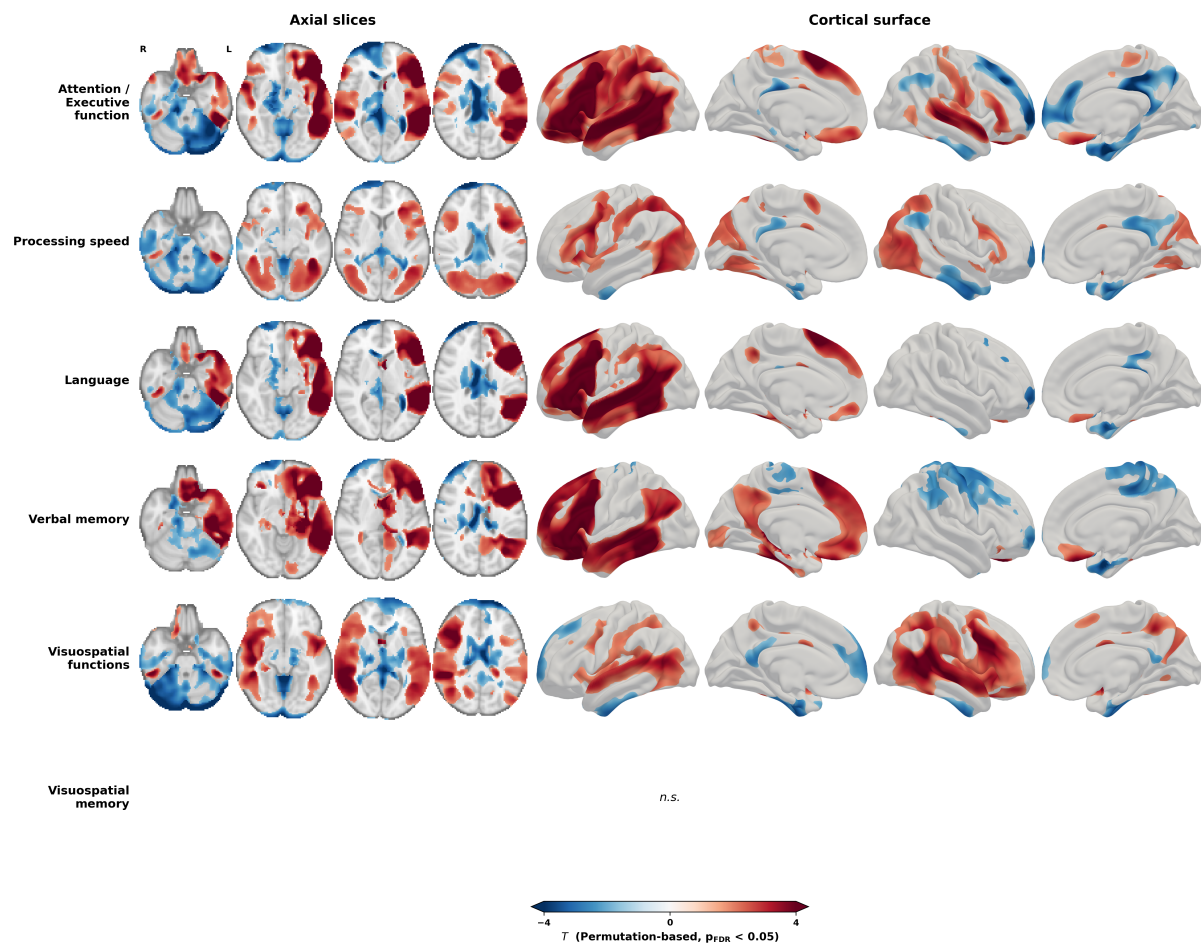


Figure 1 | Label permutation-based lesion network maps on axial slices. Lesion network maps across the six cognitive domains, thresholded at $p_{FDR} < 0.05$ and shown on four axial slices ($z = -26, -10, 6, 22$ mm; radiological orientation) as well as cortical surface renderings (Conte69 midthickness). Colours denote the direction of the group difference in lesion-derived functional connectivity: red, voxels more strongly connected to the lesions of impaired than unimpaired patients (impaired > unimpaired); blue, voxels more strongly connected to the lesions of unimpaired patients (impaired < unimpaired). Red thus marks the network whose disruption is associated with impairment in each domain. No voxels survived for visuospatial memory (n.s.). The colour scale is shared across panels. yabplot v0.5.1 was used for volume-to-surface projection and visualization.²⁷

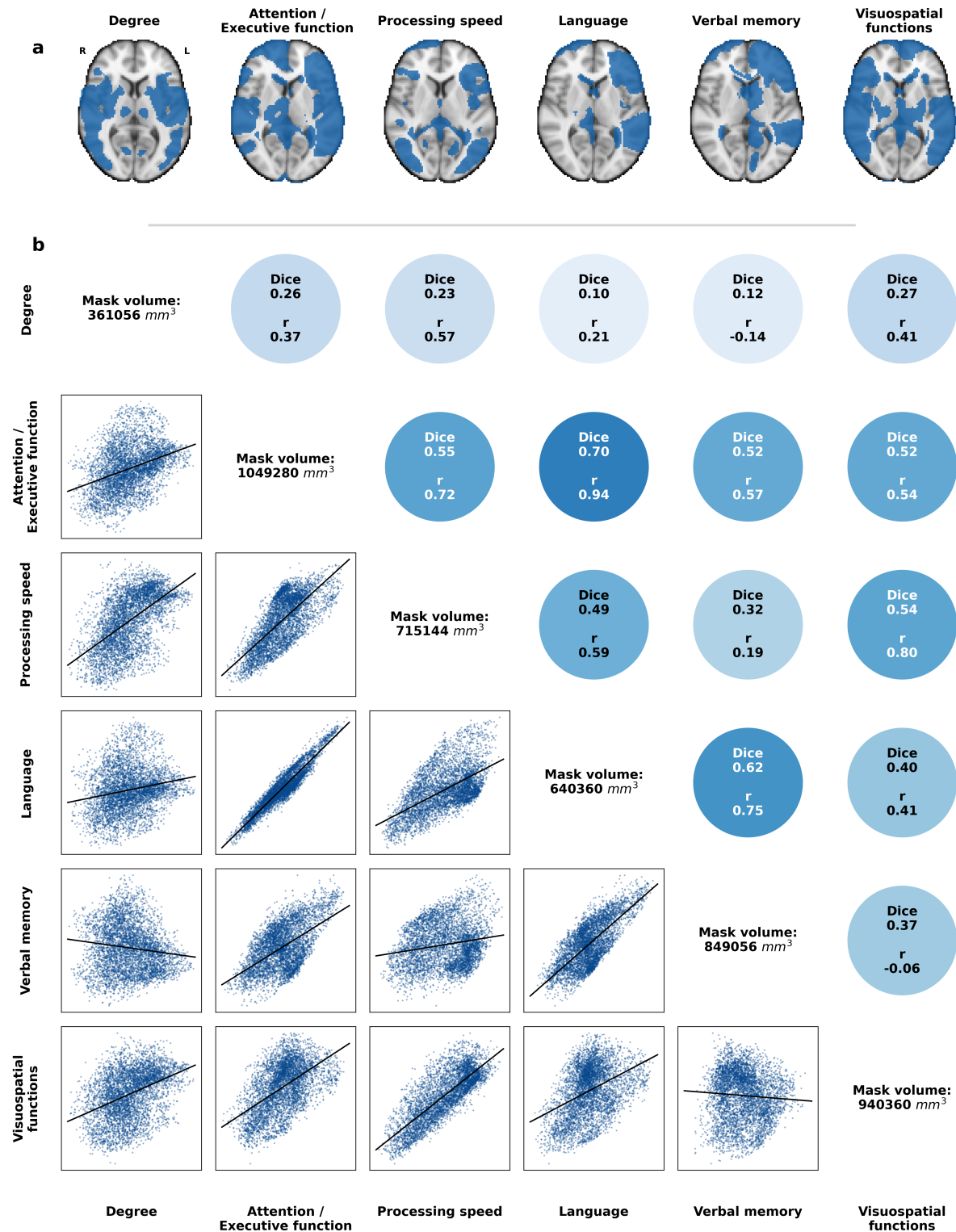
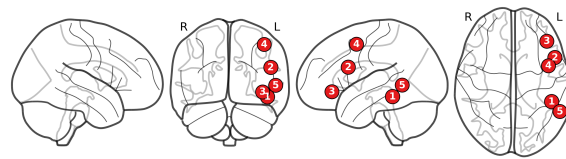


Figure 2 | Spatial correspondence of label permutation-based lesion network maps. (a) Symptom-linked lesion network maps derived using label permutation, thresholded at $p_{FDR} < 0.05$. (b) Pairwise comparisons of label permutation lesion network maps. Upper triangle: similarity quantified as Dice overlap of significance-thresholded maps. The first row/column compares each map with the normative connectome degree map (degree defined as the row-sum of the normative FC matrix; binary degree mask thresholded at the 95th percentile). Lower triangle: pairwise spatial correlation illustrated by regression plot of voxels from the

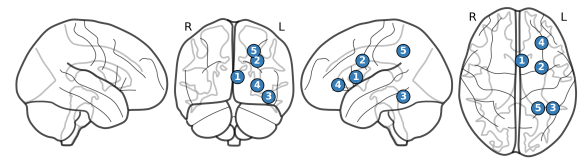
unthresholded t-statistic maps. Diagonal: volumes of thresholded masks of degree and significance maps (mm³).

Attention / Executive function



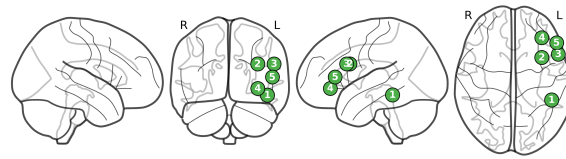
1. WM ~ LH DorsAttn Post 1 (T = +8.3)
2. LH Cont PFCL 10 (T = +7.7)
3. LH Default PFC 15 (T = +7.1)
4. LH Default PFC 42 (T = +7.0)
5. LH Default Temp 20 (T = +7.0)

Processing speed



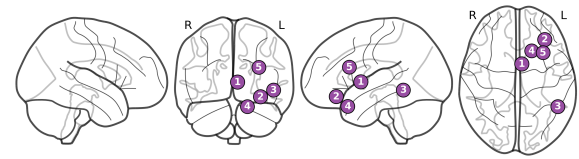
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2. WM ~ LH Cont PFCL 16 (T = +4.3)
3. LH DorsAttn Post 3 (T = +4.2)
4. WM ~ LH SalVentAttn FrOperIns 5 (T = +3.9)
5. WM ~ LH DorsAttn Post 32 (T = +3.6)

Language



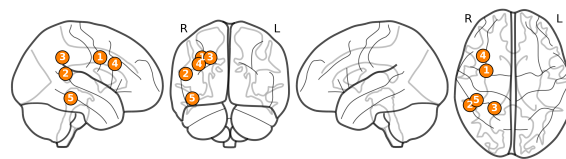
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2. LH Cont PFCL 16 (T = +7.1)
3. LH Cont PFCL 10 (T = +6.6)
4. WM ~ LH Default PFC 3 (T = +6.6)
5. LH Cont PFCL 4 (T = +6.4)

Verbal memory



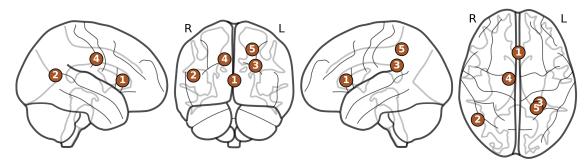
1. WM ~ CAU-VA-lh (T = +7.7)
2. LH Default PFC 3 (T = +7.5)
3. WM ~ LH DorsAttn Post 4 (T = +6.8)
4. LH Limbic OFC 3 (T = +6.3)
5. WM ~ LH Cont PFCL 14 (T = +6.3)

Visuospatial functions



1. WM ~ RH Cont PFCL 23 (T = +5.4)
2. RH SalVentAttn TempOccPar 5 (T = +4.8)
3. WM ~ RH Cont Par 17 (T = +4.8)
4. RH Cont PFCL 21 (T = +4.8)
5. WM ~ RH DorsAttn Post 3 (T = +4.7)

Visuospatial memory

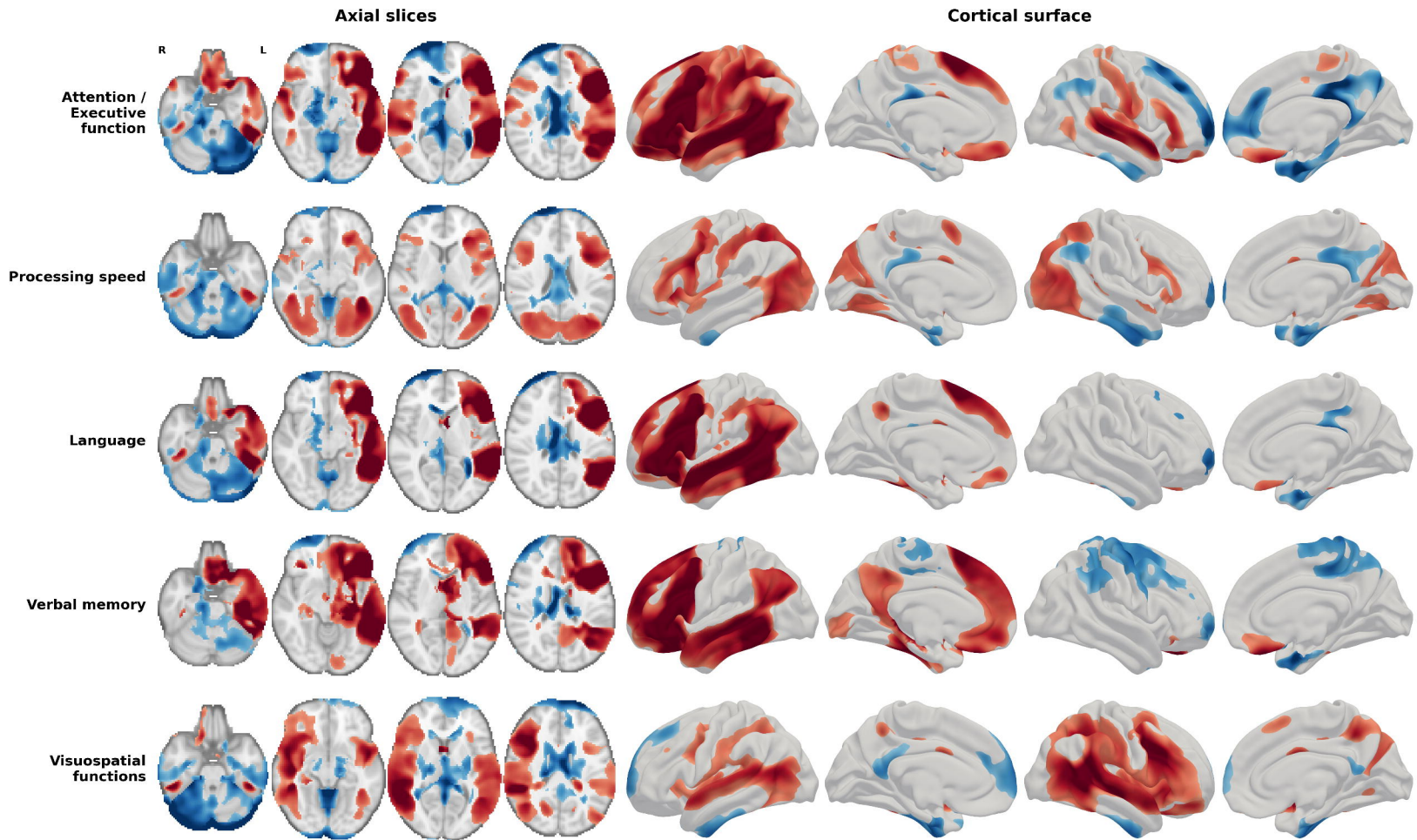


1. WM ~ LH Default PFC 25 (T = +3.6) (n.s.)
2. RH DorsAttn Post 9 (T = +3.5) (n.s.)
3. WM ~ LH Cont Par 14 (T = +3.4) (n.s.)
4. WM ~ RH SalVentAttn Med 7 (T = +3.4) (n.s.)
5. WM ~ LH SalVentAttn Med 9 (T = +3.1) (n.s.)

Figure 3 | Top five positive t-statistic peaks of lesion network maps per cognitive domain.

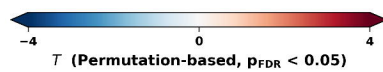
For each domain, the five strongest positive local maxima (≥ 20 mm apart as minimum separation constraint) of the unthresholded impaired-versus-unimpaired map are shown on a glass brain (left to right: sagittal right hemisphere, coronal, sagittal left hemisphere, axial), numbered by rank, and listed with their nearest anatomical parcel and t-value (R, right; L, left). Anatomical labels follow the Schaefer1000x7 cortical, Tian54 subcortical, and Buckner cerebellar parcellations; peaks falling in the white matter are labelled by their nearest parcel.

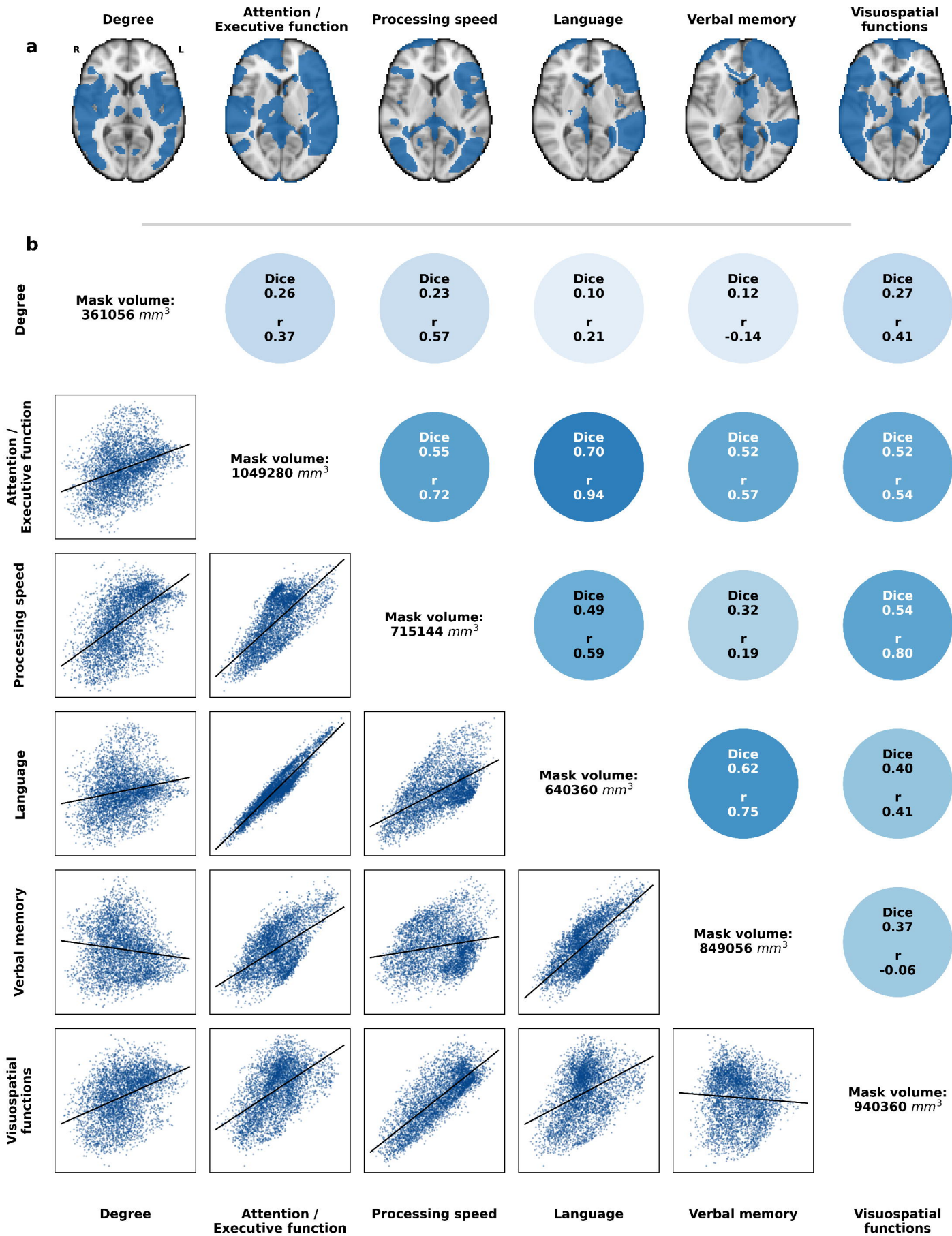
Abbreviations: R/RH, right hemisphere; L/LH, left hemisphere; WM, white matter; Cont, control network; Default, default-mode network; DorsAttn, dorsal attention network; SalVentAttn, salience/ventral attention network; SomMot, somatomotor network; Vis, visual network; Limbic, limbic network.



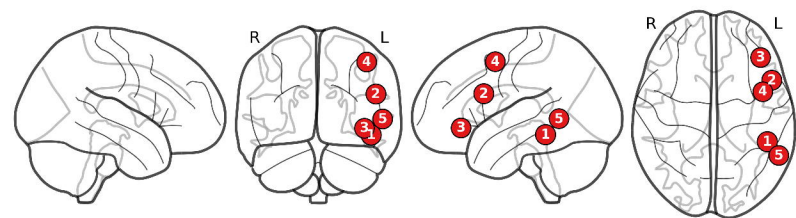
Visuospatial memory

n.s.



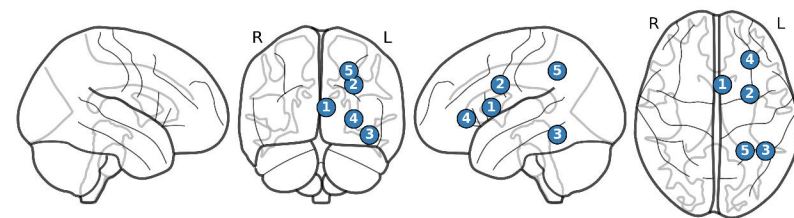


Attention / Executive function



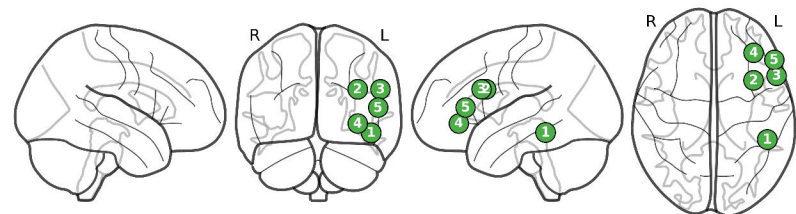
1. WM ~ LH DorsAttn Post 1 (T = +8.3)
2. LH Cont PFCL 10 (T = +7.7)
3. LH Default PFC 15 (T = +7.1)
4. LH Default PFC 42 (T = +7.0)
5. LH Default Temp 20 (T = +7.0)

Processing speed



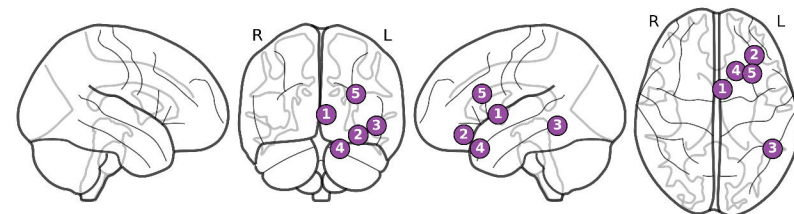
1. WM ~ CAU-VA-lh (T = +4.4) (n.s.)
2. WM ~ LH Cont PFCL 16 (T = +4.3)
3. LH DorsAttn Post 3 (T = +4.2)
4. WM ~ LH SalVentAttn FrOperIns 5 (T = +3.9)
5. WM ~ LH DorsAttn Post 32 (T = +3.6)

Language



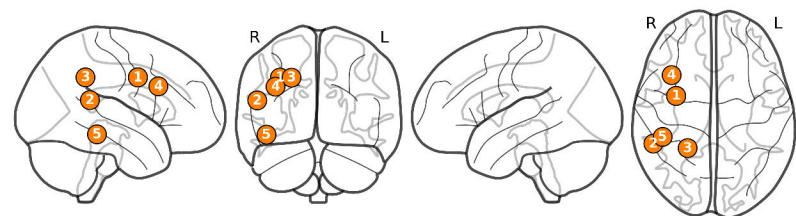
1. WM ~ LH DorsAttn Post 1 (T = +7.5)
2. LH Cont PFCL 16 (T = +7.1)
3. LH Cont PFCL 10 (T = +6.6)
4. WM ~ LH Default PFC 3 (T = +6.6)
5. LH Cont PFCL 4 (T = +6.4)

Verbal memory



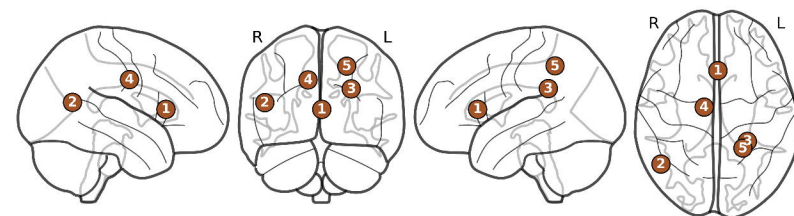
1. WM ~ CAU-VA-lh (T = +7.7)
2. LH Default PFC 3 (T = +7.5)
3. WM ~ LH DorsAttn Post 4 (T = +6.8)
4. LH Limbic OFC 3 (T = +6.3)
5. WM ~ LH Cont PFCL 14 (T = +6.3)

Visuospatial functions



1. WM ~ RH Cont PFCL 23 (T = +5.4)
2. RH SalVentAttn TempOccPar 5 (T = +4.8)
3. WM ~ RH Cont Par 17 (T = +4.8)
4. RH Cont PFCL 21 (T = +4.8)
5. WM ~ RH DorsAttn Post 3 (T = +4.7)

Visuospatial memory



1. WM ~ LH Default PFC 25 (T = +3.6) (n.s.)
2. RH DorsAttn Post 9 (T = +3.5) (n.s.)
3. WM ~ LH Cont Par 14 (T = +3.4) (n.s.)
4. WM ~ RH SalVentAttn Med 7 (T = +3.4) (n.s.)
5. WM ~ LH SalVentAttn Med 9 (T = +3.1) (n.s.)