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Fluid intelligence is supported by the multiple-demand system not the language system

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A set of frontoparietal brain regions—the multiple-demand (MD) system[1,2]—has been linked to fluid intelligence in brain imaging[3,4] and in studies of patients with brain damage[5–7]. For example, the amount of damage to frontal or parietal, but not temporal, cortices predicts fluid intelligence deficit[5]. However, frontal and parietal lobes are structurally[8] and functionally[9,10] heterogeneous. They contain domain-general regions that respond across diverse tasks[11,12], but also specialized regions that respond selectively during language processing[13]. Since language may be critical for complex thought[14–24] (compare with refs [25,26]), intelligence loss following damage to the frontoparietal cortex could have important contributions from damage to language-selective regions. To evaluate the relative contributions of MD versus language-selective regions, we employed large functional magnetic resonance imaging datasets to construct probabilistic maps of the two systems. We used these maps to weigh the volume of lesion (in each of 80 patients) falling within each system. MD-weighted, but not language-weighted, lesion volumes predicted fluid intelligence deficit (with the opposite pattern observed for verbal fluency), indicating that fluid intelligence is specifically tied to the MD system, and undermining claims that language is at the core of complex thought.

Humans are unique in the animal kingdom in that they possess a highly sophisticated communication system that can be used to exchange complex ideas. Humans are also vastly more intelligent than even our closest primate relatives[27–30]. Some have therefore argued that language is the foundation of complex thought, including our abilities for hierarchical structured thought, our ability to reason flexibly about novel problems and our ability for future-oriented thought and planning[14–24] (compare with refs [25,26]). Following brain damage, loss of fluid intelligence has long been linked to lesions of the frontal lobes[6,7], which house an important component of the language system[31]. However, the frontal lobes are highly structurally[8] and functionally[9] heterogeneous. In particular, they contain not only language-selective brain regions[13,32] but also highly domain-general regions of the multiple-demand (MD) system[11,12,33]. The MD system is an extensive bilateral frontoparietal network of brain regions active during diverse demanding tasks[11,12,34–38], and has been linked to such important constructs as cognitive control (see, for example, refs [39–41]), working memory[38], attention[2,42] and goal-directed behaviour[1,43]. Consequently, it has been argued that this system underlies the human ability for flexible thought and problem solving, which are the core ingredients of fluid intelligence[1]. Some have even hypothesized that it is specifically the expansion of the MD system in humans that endowed us with our unique cognitive capacities[44].

However, given that (1) MD regions and language-selective regions lie side by side on the lateral surface of the frontal cortex[9] and (2) the precise locations of these sets of regions are highly variable across individual brains[9], it is difficult to interpret findings that link frontal lobe damage to loss of fluid intelligence. A similar picture is obtained in the parietal cortex, which also houses both MD and language regions[1,45] and whose damage has also been linked to intelligence loss[5]. Thus, the relative contributions of the domain-general regions of the MD system and adjacent language-selective regions are unclear. We here attempt to disentangle the contributions of these two systems by combining data from 80 patients with focal brain lesions with large functional magnetic resonance imaging (fMRI) datasets from healthy participants.

The 80 patients in our study had chronic, focal, adult-onset brain lesions. Patients were chosen so that lesions were confined to either frontal or posterior (occipital, temporal, parietal) lobes. Each patient's lesion was weighted with respect to (1) a probabilistic fMRI activation overlap map (from 63 healthy participants) for a contrast targeting the MD system[12,46] and (2) a probabilistic fMRI activation overlap map (from 220 healthy participants) for a contrast targeting the high-level language processing system[45]. For the MD system map, we used data from a spatial working memory task that reliably activates the frontoparietal MD network[12]. For the language system map, we used data from a language task in which participants read sentences versus lists of pseudowords. The sentence > pseudoword-list contrast robustly and reliably activates the frontotemporoparietal language system[45,47]. For each contrast, the individual fMRI participants' maps were thresholded and overlaid in template space to create probabilistic activation overlap maps. In these maps, each voxel contains information on how many participants show an effect at the specified ($P < 0.001$) threshold. Thus, for any given voxel, we can calculate the probability that it falls within the MD system versus within the language system. The distribution of patient lesions relative to the two probabilistic maps is shown in Fig. 1.

For each patient, we estimated the deficit in fluid intelligence resulting from their lesion (their postmorbid change in fluid intelligence), by comparing current functioning to an estimate of premorbid function. We measured current fluid intelligence using two well-established tests[48,49], and estimated premorbid scores on each

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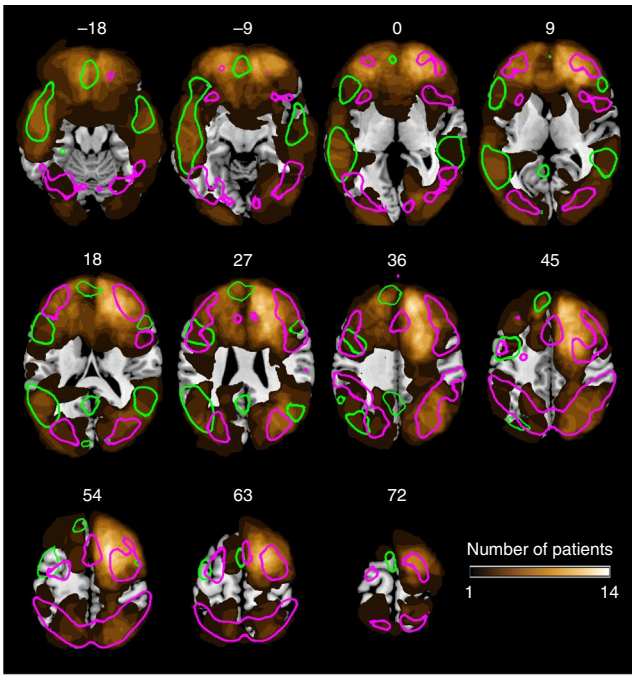

Fig. 1 | Anatomical distribution of lesions. Gold colours indicate the number of patients with a lesion at each voxel. Coloured outlines indicate regions of probability >5% in the probabilistic MD (magenta, $N=63$) and language (green, $N=220$) maps that we used to derive MD- and language-weighted lesion volume. Slices are numbered by z level in Montreal Neurological Institute (MNI) space. Our patient sample ($N=80$) provided good coverage of both the MD and language systems, with the exception of superior lateral regions of the left frontal cortex.

of these tests based on a multiple regression, derived from healthy controls, predicting the fluid intelligence score from age and crystalized intelligence[50,51], as in our previous work[5]. (Using only one of the tests[48] to assess current function, and comparing current scores with estimated premorbid scores on this test in the same way, produced a similar pattern of results.)

We then weighted each patient's lesion against the probabilistic activation maps for the MD and language system, to examine (1) the relationship between the MD-weighted lesions and postmorbid change in fluid intelligence and (2) the relationship between the language-weighted lesions and postmorbid change in fluid intelligence. The key result is shown in Fig. 2: MD-weighted, but not language-weighted, lesions predicted fluid intelligence deficit (MD-weighted: Pearson's correlation coefficient $r=-0.304$, $P=0.003$, all P values are one-tailed; language-weighted: $r=0.043$, $P=0.351$). Moreover, MD-weighted lesion volume predicted fluid intelligence deficit after language-weighted lesion volume was partialled out ($r=-0.303$, $P=0.003$), whereas the converse partial correlation was not significant ($r=0.031$, $P=0.393$). This indicates that MD lesion volume is a better predictor of fluid intelligence deficit than language lesion volume and that, after lesions to the MD system are taken into account, no further fluid intelligence deficit is accounted for by the extent to which the lesion affects language regions.

To evaluate whether this effect is also obtained specifically in the frontal lobe, which has historically been at the core of debates about human intelligence, we carried out a further analysis restricted to patients with frontal lesions only ($N=44$). Here again, MD-weighted lesion volume predicted behavioural deficit ($r=-0.258$, $P=0.046$), whereas language-weighted lesion volume did not ($r=-0.087$, $P=0.287$) (red points in Fig. 2; see also Supplementary Fig. 1). The result was the same if we instead restricted the analysis to patients with lesions affecting the left hemisphere ($N=46$): MD-weighted lesion volume predicted behavioural deficit ($r=-0.267$, $P=0.036$), whereas language-weighted volume did not ($r=0.152$, $P=0.156$) (Supplementary Fig. 2).

In two further analyses, we examined whether the results were robust to the details of how the MD and language maps were derived. First, we reran the analysis, deriving the MD probabilistic map from the composite map of Fedorenko et al.[12], in which the value at each voxel corresponds to the average t value for the contrast of hard > easy across seven cognitively demanding tasks (thresholded at $t>0$). Second, we derived a more restricted probabilistic map for the language system. For this, we masked our original map (derived from the contrast of sentences > pseudowords) with the equivalent map derived from the contrast of reading sentences > passive viewing of a fixation cross in the same 220 participants. Voxels were masked out of the restricted probabilistic language map if they did not show

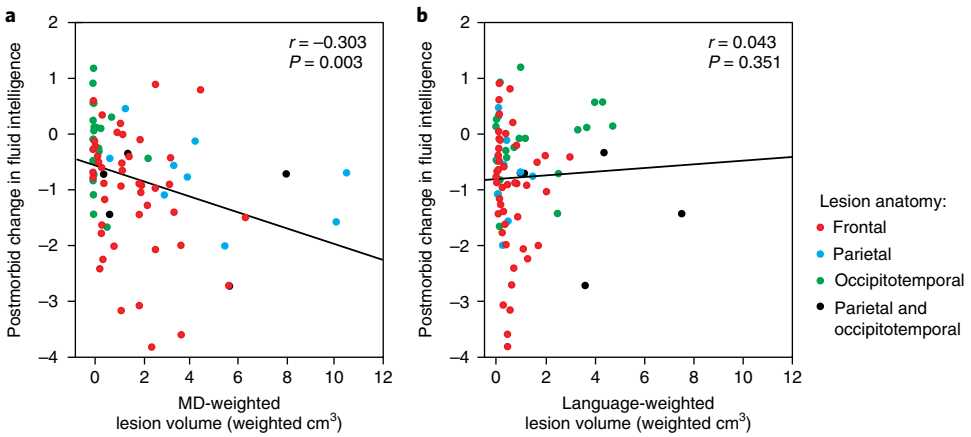

Fig. 2 | Correlation of lesion volumes with postmorbid change in fluid intelligence. a, MD-weighted lesion volume. **b**, Language-weighted lesion volume. For each patient ($N=80$), lesion volume was weighted for the extent of damage to the MD and language systems, using probabilistic maps that indicate the likelihood that each voxel belongs to the MD and language systems in healthy participants. We estimated postmorbid change in fluid intelligence by comparing current function with estimated premorbid function (postmorbid minus premorbid: a negative score indicates a deficit). Pearson's r, P values and lines of best fit are shown for the whole group. The extent to which lesions affect the MD system, but not the extent to which they affect the language system, predicts fluid intelligence deficit.

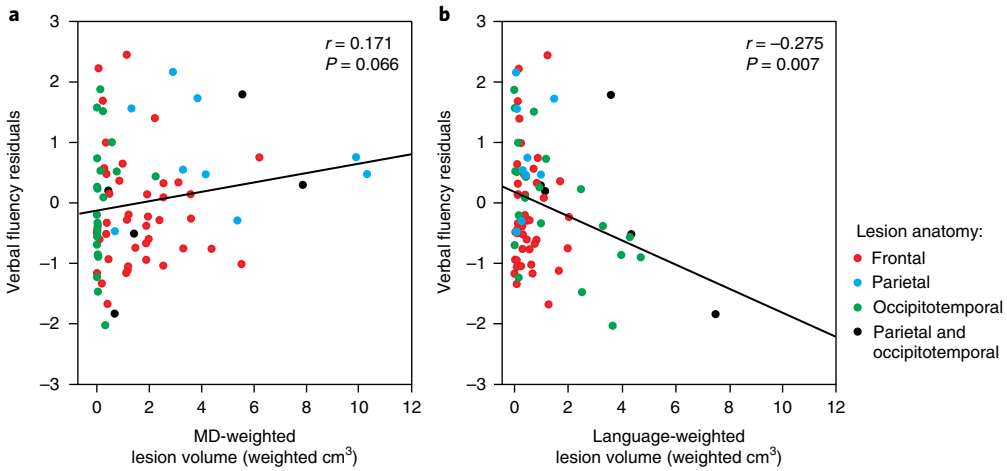

Fig. 3 | Correlation of lesion volumes with verbal fluency scores. Verbal fluency residuals are standardized residuals in the regression of Cattell Culture Fair scores against verbal fluency scores (a more negative score indicates poorer performance). Pearson's *r*, *P* values and lines of best fit are shown for the whole group ($N=79$). After partialling out variance attributable to IQ, verbal fluency is predicted by the extent to which lesions affect the language system but not by the extent to which they affect the MD system.

activation for sentences > passive viewing in at least 9 out of 220 participants (individual sentences > passive viewing maps thresholded at $P<0.001$ uncorrected). This masking procedure removed default mode network activity from the language map. The result did not change: MD-weighted lesion volume predicted fluid intelligence deficit ($r=-0.341$, $P=0.001$), whereas language-weighted lesion volume did not ($r=0.097$, $P=0.196$) (Supplementary Fig. 3).

Finally, to test whether performance on a task that relies on the language system can be predicted from language-weighted lesions, we examined our patients' performance on a test of verbal fluency[52], after regressing out variation attributable to intelligence quotient (IQ) (see ref. [53]). Indeed, we found that language-weighted lesion volume predicted verbal fluency residuals ($r=-0.275$, $P=0.007$), whereas MD-weighted lesion volume did not ($r=0.171$, $P=0.066$) (Fig. 3). Moreover, language-weighted lesion volume continued to predict verbal fluency residuals after MD-weighted lesion volume was partialled out ($r=-0.269$, $P=0.009$). In our sample, large language system lesions were usually posterior (occipitotemporal and parietal/occipitotemporal), and more data would be needed to examine the specific role of frontal language regions in fluency. Nonetheless, in the group as a whole, we observed a double dissociation between the MD and language systems and performance on fluid intelligence and language tasks.

Whereas our analyses point to the MD, and not language-selective, regions as central to fluid intelligence, they do not rule out the contribution of brain regions outside the boundaries of these two networks. A simple explanation based on total lesion volume is ruled out by the double dissociation and our previous observation that, for example, lesion volume in occipitotemporal patients does not predict fluid intelligence deficit[5]. However, contributions from other parts of the brain remain to be evaluated. For example, damage to white matter tracts plausibly plays an important role in fluid intelligence function[7].

Our results disentangle the relative causal contributions of domain-general MD regions and language-selective regions to fluid intelligence. We show that damage to the MD regions, but not to the language regions, causes fluid intelligence impairments. This work fits well with findings that individuals with severe aphasia retain the ability to engage in many forms of complex thought[25,26], with findings that show age-related decay in executive function in the presence of preservation, or even improvement, in verbal abilities[54], with the finding that executive function is unrelated to language ability in deaf preschoolers[55] and with fMRI findings that language-responsive brain regions are not active when individuals engage in diverse executive function and problem-solving tasks[25,56]. Thus, although linguistic abilities may be important in the development of certain cognitive abilities (see, for example, refs [19,25,57–61]), our data indicate that in mature human brains, the language system is not causally important for fluid intelligence.

Methods

Participants. Eighty (34 female and 46 male; mean age 51.3 (standard deviation s.d. = 12.9) years) patients with chronic, focal, adult-onset lesions (onset minimum two years before testing) of varied aetiology (see ref. [5] for details; where the same group of patients were used) were recruited from the Cambridge Cognitive Neuroscience Research Panel ($N=70$) and the Institute of Cognitive Neurology Research Database (Buenos Aires, Argentina) ($N=10$). A sample size of 80 is sufficient to detect a correlation of 0.3 with a one-tailed alpha of 0.05 and a type II error rate of 0.15 (ref. [62]). Participants were not included if they had a visual field cut, overt aphasia or pre-insult history of epilepsy, if they were unsuitable for MRI or if their lesion comprised both frontal and posterior (parietal, occipital, temporal) cortices. Lesions were traced by F.M., who was blind to the behavioural scores of the participants and experimental aims. Group lesion anatomy provided good coverage of the MD and language regions (Fig. 1). Mean premorbid IQ, assessed using either the revised National Adult Reading Test[50] or the equivalent Word Accentuation Test[51], as appropriate for each participant's first language, was 109.1 (s.d. = 13.1).

Data from thirty-three healthy control participants (21 female, 12 male), were used to create the multiple regression predicting fluid intelligence from age and premorbid IQ. These controls were recruited from the Medical Research Council Cognition and Brain Sciences Unit Volunteer Panel. They were selected to match the patient group on age (mean = 48.4 years; s.d. = 12.9 years) and premorbid IQ (mean = 109.5; s.d. = 12.3). All participants gave written informed consent and were paid under the approval of the Cambridge Local Research Ethics Committee.

Probabilistic activation overlap maps. We created probabilistic maps for the MD and language system based on extant fMRI data. For the MD system map, we used data from 63 healthy participants (47 female and 16 male, mean age 27.6 years, s.d. = 4.31 years, partially overlapping with datasets from refs [12,46]). Participants performed a spatial working memory task in which they had to remember a set of four versus eight locations in a 3×4 grid in the easy and hard conditions, respectively. The hard > easy contrast robustly and reliably activates the frontoparietal MD network, and the activations for this contrast overlap with hard > easy contrasts from numerous other tasks[12]. For the language map, we used data from 220 healthy participants (146 female and 74 male, mean age 29.1 years, s.d. = 5.09 years). Participants read sentences versus lists of pseudowords (participants either read these materials passively or performed a memory probe task at the end of each sentence/sequence; see refs [47,63] for evidence that similar activations are obtained regardless of the task). The sentence > pseudoword-list contrast robustly and reliably activates the frontotemporoparietal language system[45,47]. For each contrast, individual participants' maps were thresholded voxelwise at $P<0.001$ uncorrected, normalized and overlaid in template space to create probabilistic activation overlap maps. In these maps, the value at each voxel indicates the proportion of participants that show an effect at the specified

threshold, indicating the probability that the voxel falls within the MD system and the probability that it falls within the language system. The maps are available for download from the Fedorenko laboratory website (https://evlab.mit.edu/).

Lesion weighting. All patients had normalized lesion tracings drawn from T1-weighted Spoiled Gradient Echo MRI scans (1×1×1 mm resolution) as part of previous participation in the Panel. Each lesion was weighted twice: once for each of the probabilistic activation maps. At each voxel, the lesion (0 or 1) was multiplied by the value in the relevant probabilistic overlay map, and these values were summed to give MD-weighted and language-weighted lesion volume. This calculation was carried out in MATLAB using routines from SPM (Statistical Parametric Mapping, Wellcome Department of Imaging Neuroscience (www.fil.ion.ucl.ac.uk/spm)). Values were converted to cubic centimetres by multiplying by the volume of one voxel.

Assessment of fluid intelligence. We assessed current fluid intelligence functioning using two problem-solving tests that are known to load strongly on fluid intelligence: Cattell Culture Fair (Scale 2, Form A)[48] and Letter Sets from the Educational Testing Service Kit of Factor-Referenced Tests[49]. The tests consist of timed puzzles involving geometrical figures (Cattell) or sets of letters (Letter Sets). In Cattell, participants must determine the next in a series, odd one out, completion of a matrix or topological relations; in Letter Sets, they determine the odd one out. Patient and control participants had scores on file as part of previous participation in our Panel.

Postmorbid change in fluid intelligence. We estimated postmorbid change in fluid intelligence from the discrepancy between predicted premorbid scores, and observed postmorbid scores, on the Cattell and Letter Sets tests, as in our previous work[5]. First, we used control data to derive multiple regressions predicting Culture Fair and Letter Sets scores from age and premorbid IQ (regression co-efficient $R=0.682$ in the regression for Culture Fair, $R=0.712$ in the regression for Letter Sets). Then, we used these equations to predict premorbid Cattell and Letter Sets scores for each patient. Next, we subtracted the estimated premorbid score from the actual observed score and transformed the resulting score to a z score by dividing it by the s.d. of residuals in the relevant control group regression. Finally, we averaged the discrepancies from the two tests together to give a single measure of postmorbid fluid intelligence change, in which a negative score indicates behavioural deficit.

Assessment of verbal fluency. We assessed verbal fluency using the standard phonemic version of the verbal fluency task[52], in which participants generate as many words beginning with the letters F, A and S as they can in blocks of 1 minute per letter. Data were available for 79 out of 80 patients.

Factoring out the contribution of fluid intelligence from verbal fluency scores. As is the case with scores on many tasks across domains, verbal fluency scores are known to be predicted by fluid intelligence (see ref. [53]). Indeed, this relationship was obtained in our sample: regression of Cattell Culture Fair against verbal fluency was reliable ($R=0.412$, $F(1,77)=15.776$, $P=0.0002$, two-tailed). To test for the impact of brain lesions on the component of verbal fluency that is not attributable to fluid intelligence, we used the residuals of this regression in our correlations with language and MD-weighted lesion volumes.

Correlation of weighted lesion volume with behavioural scores. We assessed the correlation between weighted lesion volumes and derived behavioural scores by calculating Pearson's correlation coefficient (as appropriate for a linear relationship between continuous variables) and testing its significance. Reported P values are one-tailed, as the direction of the effect was prespecified (larger lesions leading to poorer performance). The data met the assumptions of the test, and additional analyses excluding points with high leverage and/or Cook's distance scores did not change the results.

Life Sciences Reporting Summary. Further information on experimental design is available in the Life Sciences Reporting Summary.

Code availability. The code used to calculate the weighted lesion volumes in this study is available on the Open Science Framework at https://osf.io/wm8a3.

Data availability. The probabilistic maps used in the current study are available for download from the Fedorenko laboratory website https://evlab.mit.edu/. The datasets generated and/or analysed during the current study are available from the corresponding author on request.

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Author contributions

E.F did the conceptualization, E.F. and A.W. did the methodology, A.W. did the formal analysis, E.F. and A.W. wrote the original draft of the paper, J.D., E.F. and A.W reviewed and edited the paper, A.W. did the visualization and E.F. and J.D. supervised. F.M. traced the patient lesions.

Competing interests

The authors declare no competing interests.

Additional information

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Experimental design

1. Sample size

   Describe how sample size was determined.

   Eighty participants from the Cambridge Cognitive Neuroscience Research Panel (N=70) and the Institute of Cognitive Neurology Research Database (Buenos Aires, Argentina) (N=10), with extant structural scans and behavioural data, met the criteria for inclusion in this study. A sample size of 80 is sufficient to detect a correlation of .3 with a one-tailed alpha of .05 and a type II error rate of .15 (see reference 63).

2. Data exclusions

   Describe any data exclusions.

   No data were excluded

3. Replication

   Describe whether the experimental findings were reliably reproduced.

   There was only one dataset: replication was not attempted. However, a series of control analyses indicated that the main result was robust to changes in the patient sample (holding when analysis was restricted to patients with lesions affecting the frontal lobe, Supplementary Figure 1; and patients with lesions affecting the left hemisphere, Supplementary Figure 2) and in the way that MD and language probabilistic maps were derived (Supplementary Figure 3).

4. Randomization

   Describe how samples/organisms/participants were allocated into experimental groups.

   Participants were not allocated to experimental groups. In the supplementary analyses we used the data for a subset of patients according to their lesion anatomy (patients with lesions affecting the frontal lobe, Supplementary Figure 1; and patients with lesions affecting the left hemisphere, Supplementary Figure 2).

5. Blinding

   Describe whether the investigators were blinded to group allocation during data collection and/or analysis.

   Investigators testing the patients were not blind to approximate lesion anatomy, however, lesion tracing and calculation of MD-weighted and language-weighted lesion volumes was done after behavioural testing had taken place. F.M., who traced the patient lesions, was blind to patient scores and experimental aims.

Note: all studies involving animals and/or human research participants must disclose whether blinding and randomization were used.

6. Statistical parameters

For all figures and tables that use statistical methods, confirm that the following items are present in relevant figure legends (or in the Methods section if additional space is needed).

n/a	Confirmed
☐	☒ The exact sample size (*n*) for each experimental group/condition, given as a discrete number and unit of measurement (animals, litters, cultures, etc.)
☐	☒ A description of how samples were collected, noting whether measurements were taken from distinct samples or whether the same sample was measured repeatedly
☒	☐ A statement indicating how many times each experiment was replicated
☐	☒ The statistical test(s) used and whether they are one- or two-sided (note: only common tests should be described solely by name; more complex techniques should be described in the Methods section)
☐	☒ A description of any assumptions or corrections, such as an adjustment for multiple comparisons
☐	☒ The test results (e.g. *P* values) given as exact values whenever possible and with confidence intervals noted
☐	☒ A clear description of statistics including central tendency (e.g. median, mean) and variation (e.g. standard deviation, interquartile range)
☒	☐ Clearly defined error bars

See the web collection on statistics for biologists for further resources and guidance.

Software

Policy information about availability of computer code

7. Software

Describe the software used to analyze the data in this study.	Weighted lesion volume was calculated in MATLAB using routines from SPM (Wellcome Department of Imaging Neuroscience, London, UK; www.fil.ion.ucl.ac.uk/spm; script available at osf.io/wm8a3). Statistical analysis was carried out in IBM SPSS Statistics Version 24.

For manuscripts utilizing custom algorithms or software that are central to the paper but not yet described in the published literature, software must be made available to editors and reviewers upon request. We strongly encourage code deposition in a community repository (e.g. GitHub). *Nature Methods* guidance for providing algorithms and software for publication provides further information on this topic.

Materials and reagents

Policy information about availability of materials

8. Materials availability

Indicate whether there are restrictions on availability of unique materials or if these materials are only available for distribution by a for-profit company.	No unique materials were used

9. Antibodies

Describe the antibodies used and how they were validated for use in the system under study (i.e. assay and species).	No antibodies were used

10. Eukaryotic cell lines

a. State the source of each eukaryotic cell line used.	No eukaryotic cell lines were used
b. Describe the method of cell line authentication used.	No eukaryotic cell lines were used
c. Report whether the cell lines were tested for mycoplasma contamination.	No eukaryotic cell lines were used
d. If any of the cell lines used are listed in the database of commonly misidentified cell lines maintained by ICLAC, provide a scientific rationale for their use.	No commonly misidentified cell lines were used

Animals and human research participants

Policy information about studies involving animals; when reporting animal research, follow the ARRIVE guidelines

11. Description of research animals

Provide details on animals and/or animal-derived materials used in the study.

No animals were used

Policy information about studies involving human research participants

12. Description of human research participants

Describe the covariate-relevant population characteristics of the human research participants.

The patient sample comprised eighty (34 female and 46 male; mean age 51.3 (SD = 12.9) years) patients with chronic, focal, adult-onset lesions (onset min 2 years prior to behavioural testing) of varied aetiology [see [5] for details; where the same group of participants were used]. Patients were selected so that they did not have a visual field cut, overt aphasia, pre-insult history of epilepsy, so that they were suitable for MRI, and so that their lesion comprised only frontal or posterior (parietal, occipital, temporal) cortices. Mean premorbid IQ was 109.1 (SD = 13.1).

We used data from 33 healthy control participants (21 female and 12 male) to create the multiple regression predicting fluid intelligence from age and premorbid IQ. These controls were selected to match the patient group on age (mean = 48.4 years; SD = 12.9 years) and premorbid IQ (mean = 109.5; SD = 12.3).

We additionally drew on extant fMRI data from 63 healthy controls (47 female and 16 male, mean age 27.6 years, SD = 4.31) from which we derived the probabilistic MD maps, and 220 healthy controls (146 female and 74 male, mean age 29.1 years, SD = 5.09) from which we derived the probabilistic language maps.