The multiple-demand system but not the language system supports fluid intelligence

Alex Woolgar[1,2*], John Duncan[3,4], Facundo Manes[5,2] and Evelina Fedorenko[6,7*]

[1] Perception in Action Research Centre, Department of Cognitive Science, Faculty of Human Sciences, Macquarie University (16 University Avenue, Sydney, NSW 2109, Australia)

[2] Australian Research Council Centre of Excellence in Cognition and its Disorders, Macquarie University (16 University Avenue, Sydney, NSW 2109, Australia)

[3] Medical Research Council Cognition and Brain Sciences Unit, University of Cambridge (15 Chaucer Road, Cambridge CB2 7EF, U.K.)

[4] Department of Psychology, University of Oxford (South Parks Rd, Oxford, OX1 3UD, U.K.)

[5] Laboratory of Experimental Psychology and Neuroscience (LPEN), Institute of Cognitive and Translational Neuroscience (INCyT), INECO Foundation, Favaloro University, Buenos Aires, Argentina; National Scientific and Technical Research Council (CONICET), Buenos Aires, Argentina;

[6] Department of Psychiatry, Harvard Medical School (55 Fruit Street, Boston, MA 02114, U.S.A.)

[7] Department of Psychiatry, Massachusetts General Hospital (149 East 13th Street, Charlestown, MA 02129, U.S.A.)

*Correspondence: alexandra.woolgar@mq.edu.au (A.W.) or evelina.fedorenko@mgh.harvard.edu (E.F.)

Keywords

Fluid intelligence, language, cognitive control, frontal, parietal, multiple-demand, neuropsychology, brain injury

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, cf. 25-26], intelligence loss following damage to frontoparietal cortex could have important contributions from damage to language-selective regions. To evaluate the relative contributions of MD vs. language-selective regions, we employed large fMRI 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), suggesting 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, cf. 25-26]. Following brain damage, loss of fluid intelligence has long been linked to lesions of the frontal lobes [6, 7] – which do 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 fronto-parietal network of brain regions active during diverse demanding tasks [11, 12, 34-38], and has been linked to such important constructs as cognitive control [e.g. 39, 40, 41], working memory [38], attention [2, 42], and goal-directed behaviour [1, 43]. Consequently, this system has been argued to underlie the human ability for flexible thought and problem solving – 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 a) MD regions and language-selective regions lie side-by-side on the lateral surface of frontal cortex [9], and b) 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 obtains 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 fMRI datasets from healthy participants.

The eighty 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 a) a probabilistic fMRI activation overlap map (from 63 healthy participants) for a contrast targeting the MD system [12, 46], and b) a probabilistic 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 which reliably activates the frontoparietal MD network [12]. For the language system map, we used data from a language task in which participants read sentences vs. lists of pseudowords. The sentence > pseudoword-list contrast robustly and reliably activates the fronto-temporo-parietal 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 vs. within the language system.

For each patient, we estimated the deficit in fluid intelligence resulting from their lesion (i.e., 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 of these tests based on a multiple regression, derived from healthy controls, predicting 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 to estimated premorbid scores 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 i) the relationship between the MD-weighted lesions and postmorbid change in fluid intelligence, and ii) the relationship between the language-weighted lesions and postmorbid change in fluid intelligence. The key result is shown in **Fig. 1:** MD-weighted, but not language-weighted, lesions predicted fluid intelligence deficit (MD: Pearson's $r = -0.304$, $p = 0.003$, all p-values one-tailed; language: $r = 0.043$, $p = 0.351$). 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 suggests 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 obtains specifically in the frontal lobe, which has historically been at the core of the 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. 1**, see also **Supplementary Figure 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 Figure 2**).

In two further analyses, we asked whether the results were robust to the details of how the MD and language maps were derived. First, we re-ran the analysis deriving the MD probabilistic map from the composite map of [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 activation for sentences > passive viewing in at least 9/220 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 Figure 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 IQ [see 53]. Indeed, we found that language-weighted lesion volume predicted verbal fluency (r = -.275, p = 0.007) whereas MD-weighted lesion volume did not (r = .171, p = .066) (**Fig. 2**). Moreover, language-weighted lesion volume predicted verbal fluency residual after MD-weighted lesion volume was partialled out (r = -.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 [54].

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 [55], with the finding that executive function is unrelated to language ability in deaf pre-schoolers [56], and with fMRI findings that language-responsive brain regions are not active when individuals engage in diverse executive function and problem-solving tasks [25, 57]. Thus, although linguistic abilities may be important in the *development* of certain cognitive abilities [e.g. 19, 25, 58, 59-62], our data suggest 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 (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] 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 .3 with a one-tailed alpha of .05 and a type II error rate of .15 [63]. Participants were not included if they had a visual field cut, overt aphasia, pre-insult history of epilepsy, or 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. 3**). Mean premorbid IQ, assessed using either the revised National Adult Reading Test [50] or the equivalent Word Accentuation Test [51], as appropriate for the participant's first language, was 109.1 (SD = 13.1).

33 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; SD = 12.9 years) and premorbid IQ (mean = 109.5; SD = 12.3). All participants gave written informed consent and were paid under the approval of the Cambridge Local Research Ethics Committee, Cambridge, UK.

Probabilistic activation overlap maps

We created probabilistic maps for the MD and language system based on extant fMRI activation data. For the MD system map, we used data from 63 healthy participants (47 female and 16 male, mean age 27.6, SD = 4.31, partially overlapping with datasets from [12, 46]). Participants performed a spatial working memory task in which they had to remember a set of four vs. eight locations in a 3x4 grid in the easy and hard conditions, respectively. The hard > easy contrast robustly and reliably activates the fronto-parietal 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, SD = 5.09). Participants read sentences vs. lists of pseudowords (participants either read these materials passively, or performed a memory probe task at the end of each sentence/sequence; see [47, 64] for evidence that similar activations obtain regardless of the task). The sentence > pseudoword-list contrast robustly and reliably activates the fronto-temporo-parietal 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 vs. 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 (SPGR) MRI scans (1x1x1mm 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 (Wellcome Department of Imaging Neuroscience, London, UK; www.fil.ion.ucl.ac.uk/spm; script available at osf.io/wm8a3). Values were converted to cm^3 by multiplying by the volume of one voxel.

Assessment of fluid intelligence

We assessed current fluid intelligence functioning using two problem-solving tests which 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 (R = .682 in the regression for Culture Fair, R = .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 standard deviation 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 as they can beginning with the letters F, A, and S, in blocks of one minute per letter. Data were available for 79/80 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 53]. Indeed, this relationship obtained in our sample: regression of Cattell Culture Fair against Verbal Fluency was reliable (r = .412, $F_{(1,77)}$ = 15.776, p = .0002, two-tailed). In order 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 pre-specified (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 scores did not change the results.

Code availability

Code used to calculate the weighted lesion volumes in this study is available on the Open Science Framework at 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.

COMPETING INTERESTS

The authors declare no competing interests.

AUTHOR CONTRIBUTIONS

Conceptualization, E.F.; Methodology, E.F. and A.W.; Formal Analysis, A.W.; Writing –

Original Draft, E.F. and A.W.; Writing – Reviewing and Editing, J.D., E.F., and A.W;

Visualization, A.W.; Supervision, E.F. and J.D. F.M. traced the patient lesions.

REFERENCES

1.	Duncan, J. (2010). The multiple-demand (MD) system of the primate brain: mental programs for intelligent behaviour. Trends Cogn Sci *14*, 172-179.

2.	Duncan, J. (2013). The structure of cognition: attentional episodes in mind and brain. Neuron *80*, 35-50.

3.	Gray, J.R., Chabris, C.F., and Braver, T.S. (2003). Neural mechanisms of general fluid intelligence. Nat Neurosci *6*, 316-322.

4.	Duncan, J., Seitz, R.J., Kolodny, J., Bor, D., Herzog, H., Ahmed, A., Newell, F.N., and Emslie, H. (2000). A neural basis for general intelligence. Science (New York, N.Y *289*, 457-460.

5.	Woolgar, A., Parr, A., Cusack, R., Thompson, R., Nimmo-Smith, I., Torralva, T., Roca, M., Antoun, N., Manes, F., and Duncan, J. (2010). Fluid intelligence loss linked to restricted regions of damage within frontal and parietal cortex. Proc Natl Acad Sci *107*, 14899-14902.

6.	Duncan, J., Burgess, P., and Emslie, H. (1995). Fluid intelligence after frontal lobe lesions. Neuropsychologia *33*, 261-268.

7.	Glascher, J., Rudrauf, D., Colom, R., Paul, L.K., Tranel, D., Damasio, H., and Adolphs, R. (2010). Distributed neural system for general intelligence revealed by lesion mapping. Proc Natl Acad Sci *107*, 4705-4709.

8.	Amunts, K., Lenzen, M., Friederici, A.D., Schleicher, A., Morosan, P., Palomero-Gallagher, N., and Zilles, K. (2010). Broca's region: novel organizational principles and multiple receptor mapping. PLoS Biol *8*.

9.	Fedorenko, E., Duncan, J., and Kanwisher, N. (2012). Language-selective and domain-general regions lie side by side within Broca's area. Curr Biol *22*, 2059-2062.

10.	Wise, R.J., Scott, S.K., Blank, S.C., Mummery, C.J., Murphy, K., and Warburton, E.A. (2001). Separate neural subsystems within 'Wernicke's area'. Brain : a journal of neurology *124*, 83-95.

11.	Duncan, J., and Owen, A.M. (2000). Common regions of the human frontal lobe recruited by diverse cognitive demands. Trends Neurosci *23*, 475-483.

12.	Fedorenko, E., Duncan, J., and Kanwisher, N. (2013). Broad domain generality in focal regions of frontal and parietal cortex. Proc Natl Acad Sci *110*, 16616-16621.

13.	Fedorenko, E., Behr, M.K., and Kanwisher, N. (2011). Functional specificity for high-level linguistic processing in the human brain. Proc Natl Acad Sci *108*, 16428-16433.

14.	Carruthers, P. (2002). The cognitive functions of language. Behav Brain Sci *25*, 657-674; discussion 674-725.

15.	Hinzen, W. (2012). The philosophical significance of Universal Grammar. Lang Sci *34*, 635-649.

16. Hinzen, W. (2013). Narrow syntax and the language of thought. Philos Psychol *26*, 1-23.

17. Dennett, D.C. (1997). The role of language in intelligence. Sprache und Denken/Language and Thought, 42.

18. Bickerton, D. (1995). Language and human behavior, (University of Washington Press).

19. Gentner, D. (2003). Why we're so smart. In Language in mind: Advances in the study of language and thought, D. Gentner and S. Goldin-Meadow, eds. (Cambridge: MIT Press).

20. Kuczaj, S.A., and Hendry, J.L. (2003). Does language help animals think? In Language in mind: Advances in the study of language and thought, D. Gentner and S. Goldin-Meadow, eds. (Cambridge, MA: MIT Press).

21. Bermúdez, J.L. (2003). Thinking without words, (Oxford University Press).

22. Baldo, J.V., Bunge, S.A., Wilson, S.M., and Dronkers, N.F. (2010). Is relational reasoning dependent on language? A voxel-based lesion symptom mapping study. Brain Lang *113*, 59-64.

23. Baldo, J.V., Dronkers, N.F., Wilkins, D., Ludy, C., Raskin, P., and Kim, J. (2005). Is problem solving dependent on language? Brain Lang *92*, 240-250.

24. Baldo, J.V., Paulraj, S.R., Curran, B.C., and Dronkers, N.F. (2015). Impaired reasoning and problem-solving in individuals with language impairment due to aphasia or language delay. Front Psychol *6*, 1523.

25. Fedorenko, E., and Varley, R. (2016). Language and thought are not the same thing: evidence from neuroimaging and neurological patients. Ann N Y Acad Sci *1369*, 132-153.

26. Varley, R. (2014). Reason without much language. Lang Sci *46*, 232-244.

27. Tomasello, M., and Herrmann, E. (2010). Ape and Human Cognition What's the Difference? Current Directions in Psychological Science *19*, 3-8.

28. Sherwood, C.C., Subiaul, F., and Zawidzki, T.W. (2008). A natural history of the human mind: tracing evolutionary changes in brain and cognition. J Anat *212*, 426-454.

29. Premack, D. (2007). Human and animal cognition: continuity and discontinuity. Proc Natl Acad Sci *104*, 13861-13867.

30.   Penn, D.C., Holyoak, K.J., and Povinelli, D.J. (2008). Darwin's mistake: explaining the discontinuity between human and nonhuman minds. Behav Brain Sci *31*, 109-130; discussion 130-178.

31.   Broca, P. (1861). Remarks on the seat of the faculty of articulated language, following an observation of aphemia (loss of speech). Bulletin de la Société Anatomique *6*, 330-357.

32.   Monti, M.M., Parsons, L.M., and Osherson, D.N. (2012). Thought beyond language: neural dissociation of algebra and natural language. Psychol Sci *23*, 914-922.

33.   Woolgar, A., Jackson, J., and Duncan, J. (2016). Coding of Visual, Auditory, Rule, and Response Information in the Brain: 10 Years of Multivoxel Pattern Analysis. J Cogn Neurosci, 1-22.

34.   Yeo, B.T., Krienen, F.M., Eickhoff, S.B., Yaakub, S.N., Fox, P.T., Buckner, R.L., Asplund, C.L., and Chee, M.W. (2014). Functional Specialization and Flexibility in Human Association Cortex. Cereb Cortex.

35.   Niendam, T.A., Laird, A.R., Ray, K.L., Dean, Y.M., Glahn, D.C., and Carter, C.S. (2012). Meta-analytic evidence for a superordinate cognitive control network subserving diverse executive functions. Cogn Affect Behav Neurosci *12*, 241-268.

36.   Dosenbach, N.U., Visscher, K.M., Palmer, E.D., Miezin, F.M., Wenger, K.K., Kang, H.C., Burgund, E.D., Grimes, A.L., Schlaggar, B.L., and Petersen, S.E. (2006). A core system for the implementation of task sets. Neuron *50*, 799-812.

37.   Naghavi, H.R., and Nyberg, L. (2005). Common fronto-parietal activity in attention, memory, and consciousness: shared demands on integration? Consciousness and cognition *14*, 390-425.

38.   Owen, A.M., McMillan, K.M., Laird, A.R., and Bullmore, E. (2005). N-back working memory paradigm: a meta-analysis of normative functional neuroimaging studies. Hum Brain Mapp *25*, 46-59.

39.   Cole, M.W., and Schneider, W. (2007). The cognitive control network: Integrated cortical regions with dissociable functions. NeuroImage *37*, 343-360.

40.   Dosenbach, N.U., Fair, D.A., Cohen, A.L., Schlaggar, B.L., and Petersen, S.E. (2008). A dual-networks architecture of top-down control. Trends Cogn Sci *12*, 99-105.

41.   Power, J.D., and Petersen, S.E. (2013). Control-related systems in the human brain. Current opinion in neurobiology *23*, 223-228.

42.   Corbetta, M., and Shulman, G.L. (2002). Control of goal-directed and stimulus-driven attention in the brain. Nature Reviews Neuroscience *3*, 201-215.

43. Miller, E.K., and Cohen, J.D. (2001). An integrative theory of prefrontal cortex function. Annu Rev Neurosci *24*, 167-202.

44. Gray, J.R., and Thompson, P.M. (2004). Neurobiology of intelligence: science and ethics. Nature reviews *5*, 471-482.

45. Fedorenko, E., Hsieh, P.J., Nieto-Castanon, A., Whitfield-Gabrieli, S., and Kanwisher, N. (2010). New method for fMRI investigations of language: defining ROIs functionally in individual subjects. J Neurophysiol *104*, 1177-1194.

46. Blank, I., Kanwisher, N., and Fedorenko, E. (2014). A functional dissociation between language and multiple-demand systems revealed in patterns of BOLD signal fluctuations. J Neurophysiol *112*, 1105-1118.

47. Mahowald, K., and Fedorenko, E. (2016). Reliable individual-level neural markers of high-level language processing: A necessary precursor for relating neural variability to behavioral and genetic variability. Neuroimage *139*, 74-93.

48. Institute for Personality and Ability Testing (1973). Measuring Intelligence with the Culture Fair Tests, (Champaign, IL: Institute for Personality and Ability Testing).

49. Ekstron R. B., French J. W., Harmon H. H., and D., D. (1976). ETS Kit of Factor-Referenced Cognitive Tests, (Educational Testing Service, Princeton, NJ).

50. Nelson, H.E., and Willison, J.R. (1991). The Revised National Adult Reading Test-Test manual, (Windsor, UK: NFER-Nelson).

51. Burin, D.I., Jorge, R.E., Arizaga, R.A., and Paulsen, J.S. (2000). Estimation of premorbid intelligence: the word accentuation test-- Buenos Aires version. J Clin Exp Neuropsychol *22*, 677-685.

52. Benton, A.L., and Hamsher, K. (1976). Multilingual aphasia examination, (Iowa City: University of Iowa Press).

53. Roca, M., Parr, A., Thompson, R., Woolgar, A., Torralva, T., Antoun, N., Manes, F., and Duncan, J. (2010). Executive function and fluid intelligence after frontal lobe lesions. Brain : a journal of neurology *133*, 234-247.

54. Glascher, J., Rudrauf, D., Colom, R., Paul, L.K., Tranel, D., Damasio, H., and Adolphs, R. (2010). Distributed neural system for general intelligence revealed by lesion mapping. Proc Natl Acad Sci *107*, 4705-4709.

55. Hartshorne, J.K., and Germine, L.T. (2015). When does cognitive functioning peak? The asynchronous rise and fall of different cognitive abilities across the life span. Psychol Sci *26*, 433-443.

56.   Vernon, M. (1967). Relationship of language to the thinking process. Arch Gen Psychiatry *16*, 325-333.

57.   Monti, M.M., Parsons, L.M., and Osherson, D.N. (2009). The boundaries of language and thought in deductive inference. Proc Natl Acad Sci *106*, 12554-12559.

58.   De Villiers, J. (2000). Language and theory of mind: What are the developmental relationships. Understanding other minds: Perspectives from developmental cognitive neuroscience *2*, 83-123.

59.   Piaget, J. (1926). The language and thought of the child. Oxford, England: Harcourt, Brace

60.   Vygotskiĭ, L.S. (2012). Thought and language, (MIT press).

61.   Gentner, D., and Loewenstein, J. (2002). Relational language and relational thought. Language, literacy, and cognitive development: The development and consequences of symbolic communication, 87-120.

62.   Winsler, A., and Naglieri, J. (2003). Overt and covert verbal problem-solving strategies: Developmental trends in use, awareness, and relations with task performance in children aged 5 to 17. Child Dev *74*, 659-678.

63.   Hulley, S.B., Cummings, S.R., Browner, W.S., Grady, D.G., and Newman, T.B. (2013). Designing clinical research, (Lippincott Williams & Wilkins).

64.   Fedorenko, E. (2014). The role of domain-general cognitive control in language comprehension. Front Psychol *5*, 335.

FIGURE LEGENDS

Figure 1. Correlation of a) MD-weighted lesion volume, and b) language-weighted lesion volume with postmorbid change in fluid intelligence.
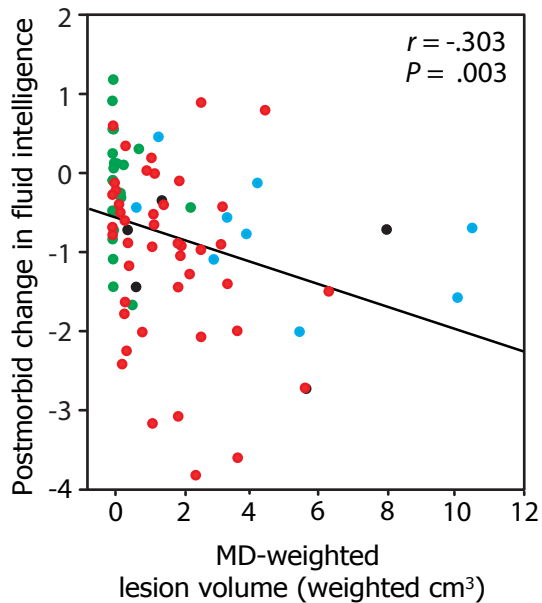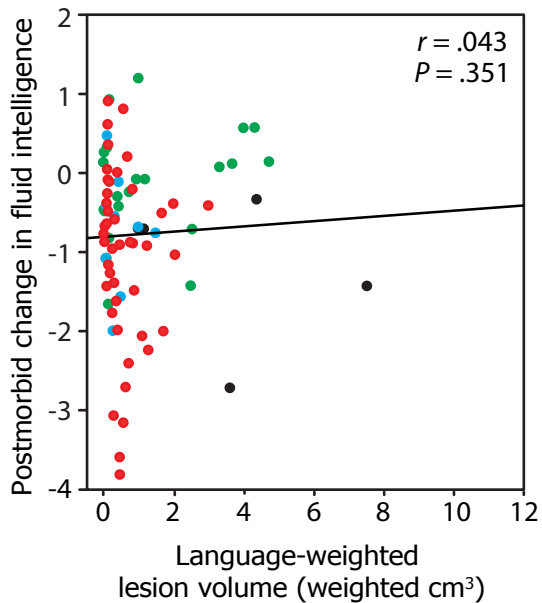
For each patient (N=80), lesion volume was weighted for the extent of damage to the MD and language systems using probabilistic maps which indicate the likelihood that each voxel belongs to the MD and language systems in healthy participants. We estimated postmorbid change in fluid intelligence by comparing current function to estimated premorbid function (postmorbid minus premorbid: a negative score indicates a deficit). Point colour indicates lesion anatomy: frontal (red), parietal (blue), occipitotemporal (green), or parietal and occipitotemporal (black). r is Pearson's correlation coefficient, *P* is the corresponding one-tailed *P*-value of the correlation. r, *P,* and fit lines are shown for the whole group. The extent to which lesions affect the MD, but not language, system predicts fluid intelligence deficit.

Figure 2. Correlation of a) MD-weighted lesion volume, and b) language-weighted lesion volume 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). Point colour indicates lesion anatomy: frontal (red), parietal (blue), occipitotemporal (green), or parietal and occipitotemporal (black). r is Pearson's correlation coefficient, *P* is the corresponding one-tailed *P*-value of the correlation. r, *P,* and fit lines 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, but not the MD, system.
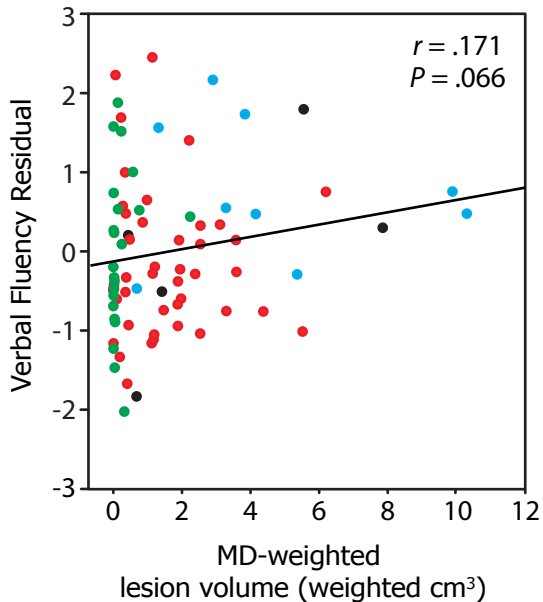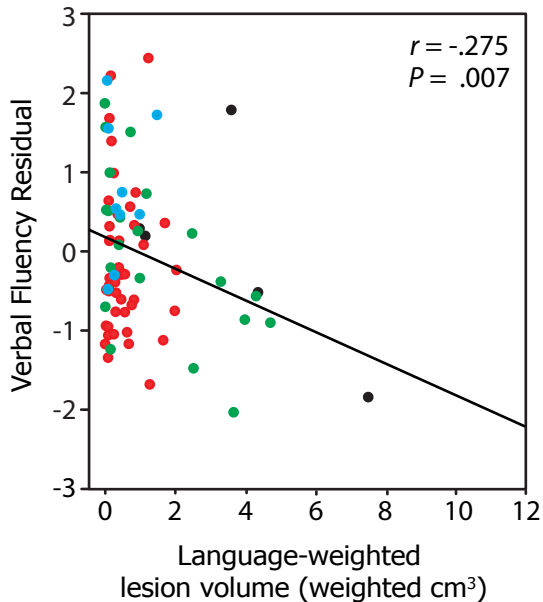
Figure 3. Anatomical distribution of lesions.

Gold colours indicate number of participants 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 which we used to derive MD and language weighted lesion volume. 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.

a

Postmorbid change in fluid intelligence vs MD-weighted lesion volume (weighted cm³)

$r = -.303$
$P = .003$

b

Postmorbid change in fluid intelligence vs Language-weighted lesion volume (weighted cm³)

$r = .043$
$P = .351$

Lesion anatomy:
- Frontal
- Parietal
- Occipitotemporal
- Parietal and occipitotemporal

a

r = .171
P = .066

Verbal Fluency Residual

MD-weighted
lesion volume (weighted cm^3)

b

r = -.275
P = .007

Verbal Fluency Residual

Language-weighted
lesion volume (weighted cm^3)

Lesion anatomy:
- Frontal
- Parietal
- Occipitotemporal
- Parietal and occipitotemporal