**Cortical surface area of the left frontal pole is associated with visuospatial working
memory capacity**

George Zacharopoulos^{1,2*}, Torkel Klingberg^{2†}, Roi Cohen Kadosh^{1†}

Affiliations:

¹Wellcome Centre for Integrative Neuroimaging, Department of Experimental Psychology,
University of Oxford, UK

²Department of Neuroscience, Karolinska Institute, Sweden

†=equal contribution

***Correspondence:**

George Zacharopoulos

Present Address: Department of Experimental Psychology, University of Oxford, Oxford,
OX2 6GG

E-mail: george.zacharopoulos@psy.ox.ac.uk

Abstract

Working memory, the ability to maintain and manipulate information over seconds, is central to cognition and it is impaired in many clinical populations. However, our knowledge of the structural properties associated with individual variation in visuospatial working memory capacity is currently poor. Across two locations (Stockholm and Oxford), we examined how regional surface area and cortical thickness in frontal and parietal regions were related to visuospatial working memory capacity. We found a negative association between visuospatial working memory capacity and the surface area of the left frontal pole across both locations, and this finding was consistently present in each of the two locations separately. Importantly, this association was specific to (i) the surface area (not cortical thickness), (ii) the left side of the brain, (iii) and the visuospatial rather than the verbal modality. This result reveals a novel and highly specific neurobiological association with visuospatial working memory which could be further explored in studies with a wider range of psychological tests and in clinical populations.

Keywords: visuospatial working-memory capacity, frontal pole, surface area

Introduction

Working memory (henceforth WM) is our ability to maintain and manipulate information over seconds (Baddeley, 1986), and individual differences in WM capacity are large and seem to be highly stable over time (Kane & Engle, 2002). WM is a pivotal factor for cognition (Conway, Kane, & Engle, 2003; Süß, Oberauer, Wittmann, Wilhelm, & Schulze, 2002). Individual variation in WM is a crucial factor in a wide range of cognitive domains including reasoning, attention, mathematics and language comprehension (Alloway & Alloway, 2010; Conway et al., 2003). WM impairments have been documented in several clinical populations (Cools & D'Esposito, 2011) including Down's syndrome, Williams syndrome, specific language impairment, and attentional deficits (Gathercole & Alloway, 2006; Luck & Vogel, 2013). Consequently, there has been great interest in uncovering the neurobiological underpinnings of WM capacity. The characterization of WM limitations can be described in terms of the number of items an individual can maintain in mind successfully, such as is measured by the K-value (Luck & Vogel, 1997),

A current review identified a fronto-parietal network as key to visuospatial (henceforth vs) WM capacity (Constantinidis & Klingberg, 2016). This review was based on research from animal neurophysiology, neural network simulation and functional neuroimaging studies in humans. There is still, however, less data on structural associations to vsWM capacity

Cortical morphology can be assessed with a volumetric representation of the brain implemented by the technique of voxel-based morphometry (Ashburner & Friston, 2000). Compared to voxel-based morphometry (Hutton, Draganski, Ashburner, & Weiskopf, 2009). surface-based morphometry (Dale, Fischl, & Sereno, 1999) separate the measurements of surface area and thickness, which is suggested to be more sensitive and specific

measurements. Moreover, morphological measures such as cortical thickness and surface area are among the parameters with the highest reliability in neuroimaging (interclass correlation >0.8) (Zuo, Xu, & Milham, 2019). Therefore, focusing on these structural parameters significantly optimizes measurement reliability which is often underappreciated in neuroscience research (Zuo et al., 2019).

A study (Colom et al., 2013) of young-adults that investigated the brain morphological overlap between intelligence and cognitive factors identified cortical grey matter volume and cortical surface area in the middle frontal gyrus to be associated with both fluid intelligence and WM, assessed using verbal and visuospatial subtests. Longitudinal developmental work (Tamnes et al., 2013) on children and adolescents (8–19 years-old) revealed that improvement in verbal WM was related to cortical volume reduction in bilateral prefrontal and posterior parietal regions and in regions around the central sulci. Another developmental study with children (from 6 to 16 years old) on the latent components of WM (verbal, visuospatial and executive component) found no relationship between cortical thickness and the verbal or vsWM components (Bathelt, Gathercole, Johnson, & Astle, 2018) but found that the executive component of WM could be predicted from an interaction between age and fractional anisotropy within a corpus callosum and an occipitotemporal region. In a structural investigation with healthy older adults, high verbal WM (n-back) performers exhibited more cortical surface area in the medial orbital frontal gyrus, the inferior frontal gyrus and the superior frontal gyrus in the right hemisphere (Nissim et al., 2017). Another study with a large adult sample identified a negative association between cortical thickness within the middle frontal gyrus and visual WM performance (Owens, Duda, Sweet, & MacKillop, 2018).

Despite our understanding of the neurostructural properties of WM capacity, the prior work mainly focused primarily on visual and verbal WM capacity and so very little

knowledge exists on the specific brain regions and structural properties that track individual variation in vsWM capacity. Importantly, several findings suggest that there may be distinct neural determinants between vsWM and other types of WM. First, several lesion studies revealed a regional dissociation between vsWM and other types of WM and even between visual and spatial WM. Lesion in the right, but not left, parietal cortex induced impairments in spatial WM (Koenigs, Barbey, Postle, & Grafman, 2009), while verbal WM impairments were affected in patients with left lesions (Shallice & Warrington, 1970; Vallar & Baddeley, 1984). Other studies showed that lesions to the temporal cortex affect visual but not spatial WM (Owen, Morris, Sahakian, Polkey, & Robbins, 1996), and the reverse pattern was documented in parietal-lesion patients (Pisella, Berberovic, & Mattingley, 2004). Second, investigations of animal models indicated a dorsal/ventral dissociation based on spatial vs. object WM (Levy & Goldman-Rakic, 2000). Third, meta-analytic studies revealed that the left prefrontal cortex (PFC) is engaged in verbal WM and the right PFC in spatial WM (Nee et al., 2012; Owen, McMillan, Laird, & Bullmore, 2005; Wager & Smith, 2003). Fourth, other studies showed that different types of WM may have different developmental trajectories and may be associated with different cognitive abilities. For example, a dissociation between mastering auditory and visual WM tasks has led to the suggestion of an earlier functional maturity for the visual than for the auditory WM system (Vuontela et al., 2003). Another developmental study (Swanson, 2017) indicated that performance on vsWM tasks as a function of age decreased at a faster rate than performance on verbal WM tasks. Moreover, an investigation employing confirmatory factor analyses in a study involving the measurement of verbal and spatial WM provided evidence of the need to distinguish verbal and spatial WM abilities (Hale et al., 2011). Lastly, a study revealed that different types of WM were related to different cognitive functions, in that vsWM was related to mathematics while verbal WM was related to reading (Giofrè, Donolato, & Mammarella, 2018).

These accumulated findings raise the possibility that vsWM capacity may stem from neurobiological mechanisms distinct from those of other WM modalities. This possibility stresses the importance of investigating the specific neurostructural underpinnings of vsWM capacity and its corresponding limitations. Given the aforementioned previous studies, an emerging open question is whether vsWM capacity resides in structural variation in key regions involved in WM in general (including frontal and parietal). Therefore, the present study aimed to examine the role of brain properties such as surface area and cortical thickness in predicting vsWM capacity. To this end, we employed a two-location study (Stockholm and Oxford) involved a scanning and a behavioural session. During the scanning session, we obtained a structural image that allowed the quantification of the structural properties of surface area and cortical thickness. During the behavioural session, participants completed a vsWM task (Figure 1) based on which we quantified the vsWM capacity. Lastly, in Stockholm participants completed another vsWM task, the span board, and one verbal WM task, the digit span. Our aims were: (i) to identify the structural determinant of vsWM capacity, (ii) to establish the structural, regional, and cognitive specificity of such a determinant.

Material and Methods

Participants

We recruited 39 participants (17 in Stockholm, 22 in Oxford) predominantly young adults and university students (Stockholm: mean age = 25.5, standard deviation = 3.6, 9 males, Oxford: mean age = 26.05, standard deviation = 6.5, 16 males). Participants were informed that the study investigated the behavioural and neural mechanisms of spatial memory. The completion of the structural acquisition lasted ~20min, and the completion of the WM testing lasted ~15min in Oxford and ~20min in Stockholm because of the additional behavioural

measures (see below). Participants received monetary compensation for their participation. Informed written consent was obtained and the study was approved by the Stockholm's Ethics Committee and by the University of Oxford's Medical Sciences Interdivisional Research Ethics Committee (MS-IDREC-C2_2015_016), respectively.

MRI data acquisition and pre-processing

In Stockholm, the anatomical high-resolution T1-weighted volume scans were acquired using a BRAVO sequence (repetition time (TR)=6.4040s; echo time (TE)=2.8080ms, 180 slices; voxel size = $1 \times 1 \times 1$ mm) in a 3T General Electrics MRI system. In Oxford, the anatomical high-resolution T1-weighted volume scans (1 mm^3) were acquired with a 3T Siemens MAGNETOM Prisma MRI System equipped with a 32 channel receiver only head coil. Anatomical high-resolution T1-weighted scans were acquired (MPRAGE sequence: TR = 1900ms; TE = 3.97ms; 192 slices; voxel size = $1 \times 1 \times 1$ mm). Since the two locations differed in their image acquisition parameters we controlled for location when applicable as discussed below.

The structural analysis was performed by utilising the recon-all function within the FreeSurfer image analysis software v6.0.0, which is documented and freely available for download online (surfer.nmr.mgh.harvard.edu). The cortical parcellation of our regions of interest was defined based on the Desikan-Killiany atlas. Our main structural parameters of interest were cortical thickness (mm) and surface area (mm^2). Based on the previous studies mentioned in the introduction, we focused our analyses on frontoparietal regions which were the following: caudal middle frontal, inferior parietal, lateral orbitofrontal, medial orbitofrontal, pars opercularis, pars orbitalis, pars triangularis, rostral middle frontal, superior frontal, superior parietal, supramarginal, frontal pole. In addition to the local measures, we

also calculated the total global surface area and the mean cortical thickness for each individual.

WM capacity measures

WM capacity for each participant was assessed in a single session. During the main vsWM task (Figure 1) participants were presented with three, four, five or six items followed by a delay and then the probe phase where they were required to make a yes/no button press depending on whether the encoding stimuli had appeared at the location indicated by the probe stimulus. Participants completed 10 trials for each of the four conditions (i.e., three, four, five or six items). The vsWM capacity was measured using a standard formula (Cowan, 2001; Vogel, McCollough, & Machizawa, 2005), $K = S * (H - F)$, where K is the vsWM capacity, S is the array size, H is the observed hit rate and F is the false alarm rate. We calculated the K for all four array sizes and average them into a single score representing the vsWM capacity.

In Stockholm, participants completed the forward versions of two additional WM capacity tests to assess the specificity of our findings: (i) the span-board forward (Wechsler, 1981), which measures vsWM, and (ii) the digit span, which measures verbal WM. In the span-board, a sequence of blocks (e.g., 6,7,8) positioned on brick was indicated by the experimenter and the participant was asked to reproduce the sequence in the same order. The test stopped after two consecutive incorrect responses and the final score was calculated. In the digit span forward (Wechsler, 2008), participants were asked to keep in memory a list of digits (e.g., 5, 8, 2) presented aurally at a rate of approximately one digit per second and were asked to immediately repeat the list in the same order. If they succeeded, a list one digit longer was presented. If they failed, a second list of the same length was presented. If subjects were successful on the second list, a list one digit longer was given, as before. The test stopped after two consecutive incorrect responses and the final score was calculated.

Figure 1. Graphical representation of a single trial across the sequential trial events: fixation, encoding, delay, probe and response phases (up to 2s). Participants were asked to remember the location of 3, 4, 5 or 6 stimuli (red dots, as displayed here).

Statistical Analyses

We employed multiple regression models in SPSS version 25 with the WM capacity score as the dependent variable. The P-values and the 95% confidence intervals (CI) presented in the results section were obtained from 10,000 samples bootstrapping methods (Ibm, 2017). The Bootstrapping method was used for deriving robust estimates of standard errors and confidence intervals for the regression coefficient of the neurostructural independent variables (see below). The independent variables were: (i) the regional cortical structure (surface area or cortical thickness within a region defined by the anatomical atlas); (ii) the global cortical structure (global surface area or mean cortical thickness), and (iii) the study

location (Stockholm or Oxford). We included global measures as covariates to correct for inter-individual differences in brain size and to separate local and global signals.

We included the study location in the models as a dichotomous covariate mainly because the two datasets were acquired using different scanners with different image sequences and acquisition parameters. Our main analyses involved combining the two datasets to improve power, as the sample size were small for each site separately and there were gender ratio differences. To correct for multiple comparisons we utilised Bonferroni correction with 48 comparisons (24 brain measures across the whole brain *2 structural properties), yielding any uncorrected P-value below .001042 to be significant (i.e., $P_{\text{CORRECTED}} < .05$).

Results

We first examined the association between vsWM capacity and the surface area (eq1.1) and cortical thickness (eq1.2) of 12 frontal and parietal regions (per hemisphere), using eq1.

$$\text{mean K-value (vsWM)} \sim \text{regional surface area} + \text{total surface area} + \text{study location}$$

(eq1.1)

$$\text{mean K-value (vsWM)} \sim \text{regional cortical thickness} + \text{total cortical thickness} + \text{study location}$$

(eq1.2)

Out of these 24 regions, only the surface area of the left frontal pole exhibited a significant association with vsWM capacity (eq1.1, Figure 2B, $B = -.011$, $t = -4.035$, $SE = .003$, $P_{\text{CORRECTED}} = .0048$, $CI = [-.016, -.006]$, uncorrected P-value = .0001), even after controlling for age and gender (eq1.3, $B = -.009$, $t = -3.457$, $Bias = -.000231$, $SE = .003$, $CI = [-.015, -.004]$, uncorrected P-value = .0023).

$$\begin{aligned} \text{mean K-value (vsWM)} \sim & \text{regional cortical thickness} + \text{total cortical thickness} + \text{study location} \\ & + \text{age} + \text{gender} \end{aligned}$$

229 (eq1.3)

230 Subsequently, we analysed the surface area of the left frontal pole in each of the two
231 study locations separately to assess whether this finding was consistently present, which was
232 indeed the case (Figure 2C, Stockholm, $B = -.012$, $t = -3.066$, $SE = .004$, $P = .006$, $CI = [-.018,$
233 $-.002]$; 2D, Oxford, $B = -.009$, $t = -2.273$, $SE = .004$, $P = .034$, $CI = [-.018, -.001]$). Of note, the
234 main effect of the left frontal pole surface area was not significant ($P = .159$) in predicting
235 vsWM without total surface area correction. For completeness, see Supplementary Material
236 for the statistical results of eq1 for all 48 brain regions.

237

238

239 **Figure 2.** Scatterplots depicting the associations between individual variation in the left
240 frontal pole surface area. (A) Parcelled left hemisphere where the frontal pole is displayed
241 in the most anterior part with a dark purple colour. The y-axis displays the surface area of
242 the left frontal pole (here the unstandardized residuals when correcting for total surface area

and study side) and the x-axis represents the vsWM capacity measured as the mean K-value (B) across locations, (C) in Stockholm, and (D) in Oxford locations separately.

After establishing a reliable predictor of vsWM capacity, we examined the specificity of this finding in terms of the (i) structural property (surface area, cortical thickness), the (ii) side of the brain (left, right) and the (iii) modality of WM (visuospatial, non-visuospatial). To investigate the extent to which the association between vsWM capacity and left frontal pole is specific to the property of surface area as opposed to the property of cortical thickness we added the left frontal pole cortical thickness as well as the total cortical thickness (i.e., mean cortical thickness across the whole brain) into the original model, yielding eq2. The addition of regional cortical thickness also requires the addition of total cortical thickness. Adding both the regional and total structural properties (surface area and thickness) in the same standard linear model allows us to examine the unique contribution of each on the vsWM.

$$\text{mean K-value (vsWM)} \sim \text{regional surface area} + \text{total surface area} + \text{regional thickness} + \text{total thickness} + \text{study location}$$

(eq2)

The left surface area of the frontal pole was still the only regional significant predictor ($B = -.011$, $t = -4.177$, $SE = .002$, $P = .0004$, $CI = [-.015, -.007]$). To examine if the association between vsWM capacity and the frontal pole was specific to the left as opposed to the right hemisphere, we added the right frontal pole surface area into the original model, yielding eq3. Adding both the frontal lobe of both the right and the left hemispheres in the same standard linear model allows us to examine the unique contribution of each on the vsWM.

$$\text{mean K-value (vsWM)} \sim \text{left regional surface area} + \text{right regional surface area} + \text{total surface area} + \text{study location}$$

267 (eq3)

268 Again, the surface area of the left frontal pole was the only regional significant predictor ($B=-$
269 $.010$, $t=-3.952$, $SE=.002$, $P=.0002$, $CI= [-.015, -.006]$).

270 Our last aim was to establish the cognitive specificity of the main findings. The following
271 analyses were conducted only in the Stockholm dataset as the additional WM tasks were
272 collected only for this location. For this reason, the following equations do not involve the
273 predictor study location. To confirm that the surface area of the left frontal pole is associated
274 with vsWM, we assessed whether the surface area of the left frontal pole can track individual
275 variation of another vsWM test, namely the span board, using eq4, which was indeed the case
276 ($B=-.026$, $t=-3.174$, $SE=.001$, $P=.006$, $CI= [-.038, -.006]$), even after additionally controlling
277 for age and gender ($B=-.023$, $t=-2.714$, $Bias=.002$, $SE=.009$, $CI=[-.038 -.001]$, uncorrected P-
278 value=.0027).

279 Span board (vsWM) ~ regional surface area + total surface area

280 (eq4)

281 To investigate if the surface area of the left frontal pole is associated with verbal WM we
282 employed the digit span forward test using eq5 but the main effect of the left frontal pole did
283 not reach significance ($B=-.025$, $t=-2.073$, $SE=.015$, $P=.07$, $CI= [-.053, .008]$).

284 Digit span (verbal WM) ~ regional surface area + total surface area

285 (eq5)

286 Lastly, using eq6 the significance of the left frontal pole in predicting vsWM was present
287 even after controlling for the verbal WM ($B=-.015$, $t=-3.221$, $SE=.005$, $P=.01$, $CI= [-.023, -$
288 $.002]$).

289 Mean K-value (vsWM) ~ regional surface area + total surface area + digit span (verbal WM)

(eq6)

Taken together, these results show that the surface area of the left frontal pole is specific to the visuospatial modality of WM.

Conclusions

In the present study, we examined the association between structural properties of the brain and vsWM capacity. This was done by combining brain imaging, allowing the quantification of several structural properties (surface area, cortical thickness) with a computerised task assessing vsWM capacity. We reported three main findings. First, we identified the role of the surface area within the left frontal pole in tracking vsWM capacity, which was independent of gender and age. Second, we found that the structural property of the left surface area was significantly associated with vsWM capacity while the structural property of cortical thickness was not. Third, the surface area of the left frontal pole tracked individual variation in vsWM but not verbal WM, indicating modal specificity.

WM capacity is a central factor of cognitive functioning and, consequently, WM capacity limitations impair cognitive functioning. Therefore, a central aim of neuroscience research is the characterization of the neurobiological underpinnings of such limitations. As mentioned in the introduction, several previous studies explored the neurostructural underpinnings of WM capacity but our knowledge of the specific neurostructural properties contributing to vsWM capacity, in particular, is very scarce. Here, we identified that individuals with relatively smaller surface area (i.e., controlling for the total surface area) within the left frontal pole exhibited higher vsWM capacity, and this finding was consistently present in each the two research locations separately. Our findings seem to be in line with prior microstructural work, which also found the involvement of frontal regions in tracking vsWM.

For example, a study showed that structural integrity in frontoparietal areas is correlated with vsWM capacity (Klingberg, 2006), and fractional anisotropy values within frontoparietal regions were positively correlated with vsWM performance and WM-related functional activity (Olesen, Nagy, Westerberg, & Klingberg, 2003).

However, the exact neurobiological nature of the role of the left frontal pole's surface area on vsWM capacity is currently unknown. The cortical surface area is thought to reflect the structural integrity of grey matter (Fischl & Dale, 2000; Lemaitre et al., 2012; Salat et al., 2004). In particular, neurons within the cerebral cortex are organized into columns that are perpendicular to the surface of the brain. The surface area is likely associated with the number of radial columns perpendicular to the pial surface whereas cortical thickness is defined by the horizontal layers in the cortical columns (Rakic, 2009). Indeed, the radial unit hypotheses postulate that the size of the cortical surface area is determined by the number of cortical columns (Rakic, 1995, 2007; Rakic & Swaab, 1988), and several proteins, such as β -catenin and caspase 3/9, have been showed to be significant contributors of surface area. Indeed, increases in surface area have been documented with both the stabilized β -catenin transgene (Chenn & Walsh, 2003) and with caspase 3/9 mutations which lead to a larger number of radial columns and cause decreased apoptosis of progenitor cells and radial glial cells respectively (Haydar, Kuan, Flavell, & Rakic, 1999). It is currently difficult based on the present study alone to make inferences as to which of these proteins may affect the surface area of the left frontal pole resulting in the formation of vsWM capacity. It was also suggested that surface area measures may even reflect the underlying white matter fibres with tension or shrinkage of these fibers leading to deeper sulci and extended surface area measures (Van Essen, 1997). This may suggest that shrinkage of the white fibres that reach the left frontal pole may be more shrunk in the individuals with lower vsWM capacity scores. Despite these speculations, future research incorporating a multi-modal approach combining

structural and functional connectivity indices can detail the neural network through which the surface area of the left frontal pole may impact the brain network level forming vsWM capacity. A limitation of the present study is that the frontal pole was treated as a unitary region. However, the frontal pole is subdivided into lateral (Fp1) and medial (Fp2) cytoarchitectonic subregions, which were shown to coactivate with a different set of regions as assessed using fMRI (Bludau et al., 2014). Regions involved in top-down control such as the dorsolateral prefrontal cortex and anterior cingulate cortex co-activate with the lateral frontal pole while medial frontal pole coactivates with temporal pole, posterior cingulate and posterior superior temporal sulcus (Gilbert, Gonen-Yaacovi, Benoit, Volle, & Burgess, 2010). Taken these findings together, the frontal pole is thought to be an attention control system responsible to select between attending to internal (lateral frontal pole) or attending to external (medial frontal pole) information collectively termed the gateway hypothesis (Orr, Smolker, & Banich, 2015). Indeed, succeeding in the present vsWM task requires switching between attending to external (stimulus encoding phase) and attending to internal (maintenance phase), and back to external information (response probe). Indeed prior resting-state functional connectivity study revealed that the lateral frontal pole is connected to the executive control network, associated with directed attention and WM (Moayed, Salomons, Dunlop, Downar, & Davis, 2015). There is also evidence suggesting that the lateral frontal pole is uniquely human as connectivity comparison between humans vs. macaques using structural and functional neuroimaging methods revealed that the ventrolateral frontal pole is specific to humans as it could not match any prefrontal cortex macaque region (Neubert, Mars, Thomas, Sallet, & Rushworth, 2014). Based on these findings we speculate that the surface area of the lateral frontal pole than the total frontal pole area should be more relevant to vsWM capacity.

Apart from the medial-lateral distinction recent evidence using anatomical and functional connectivity methodologies suggest the existence of ventral-dorsal gradient within the frontal pole (Orr et al., 2015). According to this view, dorsal regions are connected to process goals and action plan prefrontal regions, medial regions are connected to regions monitoring action outcomes and motivating behaviour and ventral regions connect to regions processing information about stimuli, values and emotions. However, it is still unclear which of this set of frontal pole connections are most relevant to vsWM. Future structural and connectivity work can examine whether the structural parameters or connectivity patterns of specific frontal pole's subregions is more critical in tracking vsWM capacity. In particular, such studies can reveal the impact of the left frontal pole surface area at the mechanistic network level, a piece of missing information that we could not addressed with our neuroimaging protocol. For instance, the effect of frontal pole's surface area on vsWM capacity may be mediated by the connectivity between left lateral frontal pole and seeds within the executive control network. This is a likely case as previous studies established these connections to frontal pole, and these regions are known to be underpin WM (Moayed, Salomons, Dunlop, Downar, & Davis, 2015).

Another limitation of the current investigation is the fact that the main finding is of a correlation in nature. Although several specific proteins discussed above were shown to experimentally alter the surface area, measuring vsWM capacity in mice has its limitations and may not be applicable. A more viable method to probe causality may involve cognitive training, where individuals are trained in the vsWM and verbal WM task described here and assessed as to whether this training intervention induced training alterations, specifically in the surface area of the left frontal pole and in response to the visuospatial rather than the verbal WM training.

In summary, by investigating cortical morphometric properties such as surface area and thickness the present research allowed the identification of an objective neurostructural marker for vsWM capacity. Given the central role of WM capacity on cognitive processes, these findings pave the way for further studies into the causal role of the neural differences influencing core components of cognition which are impaired in different clinical populations.

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Contributions

G.Z., T.K., R.C.K. designed the experiments; G.Z. performed the experiments; G.Z. analysed the data, G.Z., T.K., R.C.K. wrote the paper.

Competing interests

The authors declare no competing financial interests.

Corresponding author

Correspondence to George Zacharopoulos (george.zacharopoulos@psy.ox.ac.uk)

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Supplementary Material

Statistical results (t= t-statistic, B= beta-weight, SE= Standard error, P= P-value, not corrected) of eq1 for all 24 regions (12 in each hemisphere) (L=left, R=right, CT= cortical thickness, SA= surface area). The analyses are listed according to p-values.

	t	B	SE	P
L frontopole (SA)	-4.0355	-0.011	0.0027	0.0003
R parsopercularis (SA)	2.1901	0.0012	0.0005	0.0353
L caudalmiddlefrontal (CT)	2.1683	2.468	1.1382	0.037
R rostralmiddlefrontal (SA)	-1.8758	-0.0003	0.0002	0.069
R parstriangularis (CT)	1.838	1.8385	1.0003	0.0746
L rostralmiddlefrontal (SA)	-1.7858	-0.0003	0.0002	0.0828
L parstriangularis (CT)	1.7127	1.9055	1.1126	0.0956
L parsorbitalis (SA)	-1.457	-0.0021	0.0015	0.154
L parsorbitalis (CT)	1.4445	0.9737	0.6741	0.1575
L lateralorbitofrontal (CT)	1.3584	1.6895	1.2437	0.183
R parsorbitalis (SA)	-1.3425	-0.0013	0.0009	0.1881
L rostralmiddlefrontal (CT)	1.2575	1.9399	1.5427	0.2169
L superiorfrontal (SA)	1.1825	0.0002	0.0002	0.245
R caudalmiddlefrontal (CT)	1.1155	1.3835	1.2403	0.2722
R medialorbitofrontal (SA)	1.0467	0.0007	0.0007	0.3024
L superiorfrontal (CT)	-0.8668	-1.4507	1.6736	0.3921
R inferiorparietal (CT)	0.8588	1.4165	1.6493	0.3963
R superiorfrontal (SA)	-0.858	-0.0002	0.0002	0.3967
L supramarginal (CT)	-0.8054	-1.1646	1.4461	0.426

R inferiorparietal (SA)	-0.7953	-0.0002	0.0002	0.4318
R caudalmiddlefrontal (SA)	-0.7895	-0.0003	0.0004	0.4351
R superiorparietal (SA)	0.7718	0.0002	0.0002	0.4454
R parsorbitalis (CT)	0.7597	0.583	0.7675	0.4525
R superiorparietal (CT)	-0.7277	-1.2721	1.7481	0.4718
R rostralmiddlefrontal (CT)	0.6857	0.9722	1.4179	0.4974
L caudalmiddlefrontal (SA)	0.5964	0.0002	0.0004	0.5547
L inferiorparietal (SA)	-0.5939	-0.0002	0.0003	0.5565
R frontalpole (CT)	0.56	0.283	0.5054	0.5791
L supramarginal (SA)	0.5479	0.0001	0.0002	0.5872
R lateralorbitofrontal (SA)	-0.5398	-0.0002	0.0004	0.5928
L medialorbitofrontal (SA)	-0.4946	-0.0003	0.0006	0.624
R supramarginal (SA)	-0.4806	-0.0001	0.0003	0.6338
R medialorbitofrontal (CT)	0.4408	0.3776	0.8567	0.6621
L frontalpole (CT)	0.4391	0.2305	0.525	0.6633
R supramarginal (CT)	-0.4291	-0.7974	1.8584	0.6705
R frontalpole (SA)	-0.4118	-0.0015	0.0036	0.683
R parsopercularis (CT)	-0.3751	-0.3826	1.0198	0.7098
R parstriangularis (SA)	-0.3522	-0.0002	0.0004	0.7268
L superiorparietal (CT)	-0.3428	-0.5981	1.7447	0.7338
R lateralorbitofrontal (CT)	0.302	0.2547	0.8435	0.7644
L lateralorbitofrontal (SA)	0.2633	0.0002	0.0006	0.7939
L parstriangularis (SA)	-0.2389	-0.0002	0.0007	0.8126
L inferiorparietal (CT)	-0.1566	-0.2308	1.4742	0.8765
L superiorparietal (SA)	-0.1094	0	0.0002	0.9135

L parsopercularis (CT)	-0.1019	-0.11	1.0797	0.9194
R superiorfrontal (CT)	-0.0678	-0.0977	1.4414	0.9464
L parsopercularis (SA)	-0.0435	0	0.0006	0.9655
L medialorbitofrontal (CT)	0.0254	0.0276	1.0851	0.9799