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Overlapping and dissociable brain activations for fluid intelligence and executive functions

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Abstract

Cognitive enhancement interventions aimed at boosting human fluid intelligence (g*f*) have targeted executive functions (EFs), such as updating, inhibition, and switching, in the context of transfer-inducing cognitive training. However, even though the link between EFs and g*f* has been demonstrated at the psychometric level, their neurofunctional overlap has not been quantitatively investigated. Identifying whether and how EFs and g*f* might share neural activation patterns could provide important insights into the overall hierarchical organization of human higher-order cognition, as well as suggest specific targets for interventions aimed at maximizing cognitive transfer. We present the results of a quantitative meta-analysis of the available fMRI and PET literature on EFs and g*f* in humans, showing the similarity between g*f* and (i) the overall global EF network, as well as (ii) specific maps for updating, switching, and inhibition. Results highlight a higher degree of similarity between g*f* and updating (80% overlap) compared with g*f* and

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Declarations

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inhibition (34%), and gf and switching (17%). Moreover, three brain regions activated for both gf and each of the three EFs also were identified, located in the left middle frontal gyrus, left inferior parietal lobule, and anterior cingulate cortex. Finally, resting-state functional connectivity analysis on two independent fMRI datasets showed the preferential behavioural correlation and anatomical overlap between updating and gf. These findings confirm a close link between gf and EFs, with implications for brain stimulation and cognitive training interventions.

Keywords

Executive functions; Fluid intelligence; fMRI; Functional connectivity; Cognitive enhancement

Introduction

Fluid intelligence (gf) has been defined as the ability to cope with novel scenarios irrespective of previously acquired knowledge, identifying and manipulating chunks of available information to drive the emergence of a solution (Cattell, 1987; Cattell, 1963; Horn & Cattell, 1966). Remarkably, gf closely correlates with a vast number of cognitive activities and is suggested to be an important predictor of both academic and professional success (Deary et al., 2007; Ren et al., 2015; Rohde & Thompson, 2007; te Nijenhuis et al., 2007; Watkins et al., 2007), as well as overall health and mortality (Gottfredson & Deary, 2004). Given these implications, in the past 20 years a great deal of effort has been devoted to understand the neural correlates underlying gf (Ebisch et al., 2012; Jung & Haier, 2007; Prabhakaran et al., 1997). Various theories and models have been proposed (Basten, Hilger, & Fiebach, 2015; Cole, Yarkoni, Repovs, Anticevic, & Braver, 2012; Colom et al., 2009; Ebisch et al., 2012; Langer et al., 2012; Santarnecchi, Rossi, & Rossi, 2015a; Wang, Song, Jiang, Zhang, & Yu, 2011), with a general agreement on the contribution of a bilateral network of brain regions predominantly comprised of the prefrontal and parietal lobes. This has led to the Parieto-Frontal Integration theory of intelligence (P-FIT)(Jung & Haier, 2007), a model describing the most relevant regions involved in intelligence-related processing, as well as their specific role and interplay during cognitive processing. Moreover, a recent meta-analysis supports the frontoparietal network (FPN) as a pivotal component supporting abstract reasoning abilities (Santarnecchi et al., 2017) and also highlights the relevance of a subset of resting-state networks (RSNs) (Sporns, 2014) linked to awareness, salience processing, and attention (Santarnecchi et al., 2017). Moreover, several recent studies highlighted that similarity between task and rest functional connectivity within brain networks is related to behavioral performance (Schultz & Cole, 2016; Zuo et al., 2018).

While a better understanding of neurophysiological underpinnings of gf can help to identify neuroanatomic targets for gf enhancement, in the past decade a large number of studies have instead focused on developing cognitive training interventions to enhance executive functions (EFs), the complex set of functions that allows for voluntary behavior toward long-term goals. A widely used model of EFs by Miyake et al. (2000) proposes three core competencies that while being correlated show clear distinction between them, namely (i) switching of task or goal sets (“switching”), (ii) updating and monitoring of

working memory representations ("updating"), and (iii) inhibition of prepotent responses ("inhibition") (Miyake et al., 2000). This concept of "unity and diversity of EFs" has been replicated in many subsequent studies (Fisk & Sharp, 2004; Friedman et al., 2006; Hedden & Yoon, 2006; Huizinga et al., 2006; Lehto et al., 2003; van der Sluis et al., 2007), including neuroimaging ones showing activation of frontoparietal brain regions during EF tasks but also different activation in frontal and/or posterior areas unique to switching and updating (Collette et al., 2005; Sylvester et al., 2003). Of these core EFs, the vast majority of studies has focused on training updating ability (Baltes et al., 1989; Harrison et al., 2013; Jaeggi et al., 2008; Jaeggi et al., 2011) (for a review see: Au et al., 2015) with the goal of inducing a positive transfer to g*f*. This research was guided by a large set of psychometric studies showing a correlation between g*f* and EF abilities (Friedman et al., 2006; McCabe et al., 2010; Miyake et al., 2000; Salthouse et al., 2003; Salthouse & Davis, 2006; Salthouse & Pink, 2008) and lesion studies showing EF and g*f* being both susceptible to frontal lobe lesions (Barbey et al., 2014; Duncan et al., 1995; Roca et al., 2010; Woolgar et al., 2010). In more detail, there are approximately 40 published studies designed to enhance g*f* by means of EF training, most of them based on single (Halford et al., 2007; Jaušovec & Jaušovec, 2012; Studer et al., 2009) or dual-task working-memory trainings tested in adults (Jaeggi et al., 2008; Thompson et al., 2013). In addition, some studies have attempted to enhance g*f* by improving WM in neurotypical children (Zhao et al., 2011) and children with attention-deficit/hyperactivity disorder (Klingberg et al., 2002). While latent factor analysis of behavioral data would suggest high overlap between EFs and g*f*, and therefore the potential for transfer of abilities to g*f*, results have been controversial so far. Some studies reported a benefit (Jaeggi et al., 2008; Jaušovec & Jaušovec, 2012), and others showed no impact of EF training on g*f* (Foroughi et al., 2016; Thompson et al., 2013). In addition, to date, the overlap between g*f* and switching, updating, and inhibition remains purely psychometric; no study investigated the quantitative functional overlap of brain networks across EFs and g*f*. While several studies found a highly correlation between EFs (especially updating) and g*f*, others reported that this could be a methodological error regarding the measurement of the EFs (Frischkorn et al., 2019; Hedge et al., 2018; Rey-Mermet et al., 2019). Looking further, they suggested that the highly correlation between updating and g*f* could be the results of the reliability, characteristic of updating and not of shifting and inhibition. Moreover, these studies point out that updating may be more strongly related to g*f* than any other EFs, because it is not derived as a difference measure. It may simple reflect general WM capacity, while shifting and inhibition may reflect the processing speed (Jewsbury et al., 2016). However, highly correlated behavioral measures may not share the same neural substrates, but only show high levels of covariance, potentially due to the known phenomenon of positive manifold (for a review see: Colom et al., 2016). This term refers to the possibility that different neural regions are the basis of performance on two very different tasks, which, however, are positively related to each other at the behavioral level. This might lead to scenarios where training function "A" might not activate areas relevant for function "B," therefore leading to no transfer of abilities. At the same time, it might be that increasing one's ability in function "A" might just be sufficient to increase performance at "B" by making a cognitive subtask linked to "B" -but also relevant for "A"- more efficient. Distinguishing between functional overlap and the phenomenon of positive manifold is not only highly relevant for further advancing the field of cognitive

enhancement but for identifying potential target regions for noninvasive brain stimulation interventions. Thus, characterizing the overlap between the functional networks supporting EF and g*f* represents a critical next step toward improving interventions designed to augment EFs and g*f*. In attempt to quantify the overlap between the functional networks supporting EFs and g*f*, here we present a systematic quantitative meta-analysis of functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) data collected during EFs- and g*f*-related processing. Data were gathered from 163 papers and analyzed within the Activation Likelihood Estimate (ALE) analytic framework (Eickhoff et al., 2009). Separate meta-analytic maps were created for switching, updating, and inhibition, classifying existing literature on the basis of the Miyake's influential EF model (Miyake et al., 2000). As the core of the present investigation, a DICE similarity index was calculated between each EF map and the g*f* meta-analytic map recently published by our group (Santarnecchi et al., 2017). Specific cortical and subcortical overlaps were identified for each pair, allowing us to generate potential hypotheses about switching-, updating-, and inhibition-specific overlaps with g*f*. In addition, regions showing full overlap among all three EFs and g*f* were identified, because they would likely represent the most suitable target for generating EF→g*f* transfer. According to previous behavioral evidence, we predicted a greater degree of overlap for updating and g*f*, with a major contribution by shared regions in the prefrontal and parietal lobes, bilaterally.

Finally, given previous evidence of a specific correlation structure between g*f* and EFs scores in neurotypical individuals (Salthouse et al., 1998; Salthouse et al., 2003), suggesting a stronger link between updating and g*f* and close to no correlation between g*f* and inhibition/switching (Engle et al., 1999; Salthouse & Pink, 2008), we analyzed behavioral data from two independent databases, looking at behavioral correlations across the four cognitive functions. According to prior literature, we hypothesized a stronger similarity/correlation between behavioral data related to g*f* and updating and lower correlations between g*f*, inhibition, and switching.

Materials and Methods

A quantitative ALE meta-analysis of the available literature about g*f*, inhibition, updating, and switching was performed. A statistical comparison of the resulting maps was performed using the software GingerALE. The similarity between g*f* and EF was tested by analyzing resting-state fMRI data from two datasets collected in Boston (MA, USA) and Siena (Italy). Moreover, a parallel analysis on behavioral data was performed to verify whether observed similarity in connectivity was reflected in the psychometric interaction of EF and gf tasks. Details about the analysis are reported below.

Quantitative meta-analysis comparison

Literature search and database creation

Executive functions: Potentially relevant articles were retrieved by performing a search in PubMed and Google Scholar databases without temporal restrictions. To specify the object of the present review, terms such as "executive function," "inhibition," "updating," "flexibility," "switching," "switching," "frontal functioning," and "working memory" were

individually combined with “functional magnetic resonance imaging,” “position emission tomography,” and related abbreviations (fMRI, PET). The searches for methods and research topics were combined with AND operator. We screened 268 publications from which we excluded 105 using several exclusion criteria: (i) studies including patients with organic illness, (ii) review papers, (iii) studies not reporting fMRI/PET activations coordinates in Montreal Neurologic Institute (MNI) or Talairach space, (iv) studies using a priori-defined regions of interest, and (v) studies not reporting activation foci in table format or reporting statistical values without corresponding coordinates. The final sample was composed by 163 publications (updating = 65; switching = 21; inhibition = 77) (Figure S1). As shown in Table S1, for each study, the following information was retrieved: (i) sample size, (ii) cognitive task, (iii) coordinate system, and (iv) number of foci. Different maps were created, carefully inspecting each manuscript and extracting activation foci from tables referring to the contrast of interest. A list of the publications considered is reported in Table S1.

Fluid intelligence: Data from a recently published set of maps by our group were used (Santarnecchi et al., 2017). Specifically, even though the available ALE database includes ten g*f*-related maps (including functional activations, e.g., for verbal and visuospatial material, as well as related to cognitive processing stages, such as Rule Inference and Rule Application), we focused on identifying of a more general overlap between EFs and g*f*. Therefore, we used the general g*f* map (corresponding to the data shown in Fig. 1 and Table 1 in Emiliano Santarnecchi et al., 2017).

ALE maps computation—The quantitative evaluation of spatial fMRI patterns was carried out using the activation likelihood estimate (ALE) method implemented in GingerALE software v2.3.2 (www.brainmap.org) (Eickhoff et al., 2012; Eickhoff et al., 2009). Differently from within-study SPM analysis where every voxel in the image space is tested against a null hypothesis of no activation, the ALE method assumes that for each study of interest there is a given spatial distribution of activity and an associated set of maximal coordinates. Therefore, the algorithm tests to what extent the spatial locations of the activation foci correlate across independently conducted fMRI studies investigating the same construct.

First, the lists of coordinates were carefully checked for duplication of data across publications in order to avoid artefactual inflation of a given foci significance. Coordinates collected from studies reporting activation foci in Talairach space were converted into the MNI space using the tal2mni algorithm implemented in GingerALE. Activation foci from each study were modeled as Gaussian distributions and merged into a single 3D volume. The ALE algorithm modeled spatial uncertainty of each activation focus (Turkeltaub et al., 2012), using an estimation of the intersubject and interstudy variability typically observed in neuroimaging experiments, rather than applying a priori full-width half maximum (FWHM) kernel. Therefore, the number of participants in a given study influenced the spatial extent of the Gaussian function used. We first modeled the probability of activation over all the studies at each spatial point in the brain, returning localized “activation likelihood estimates” or ALE values. Values were then compared with a null distribution created from simulated datasets with randomly placed foci, in order to identify significantly activated clusters

(permutations test = 1,000 run). Following Eickhoff and colleagues arguments supporting a better balance between sensitivity and specificity for Cluster-based corrections over False-Discovery-Rate (FDR) and Family Wise Error (FWE) approaches (Eickhoff, Bzdok, Laird, Kurth, & Fox, 2012), we applied cluster correction for multiple comparisons with a $p < 0.001$ threshold for cluster-formation and a $p < 0.05$ for cluster-level inference. Only clusters with a size exceeding the cluster size recommended by ALE were reported (range 500–1,000 mm^3).

Quantitative ALE overlap analysis—Specific statistical comparisons were computed in order to identify segregated neurobiological signatures of each EFs component as well as conjunction maps showing (i) conjunctions and disjunctions between EFs and g*f* (e.g., updating and g*f*) and (ii) significant overlap between core EFs (e.g., updating and inhibition, see supplementary information). The procedure involved the creation of a combined map, including the two maps of interest (i.e., including all the activation foci), using the voxel-wise minimum value of the input ALE images. Contrast images were created from the subtraction of each pair of ALE maps, together with a map showing their statistically significant overlap. Given that the resulting subtraction image has the major drawback of not considering the differences in the dataset sizes between the two original maps, GingerALE's simulated data of the pooled foci datasets, obtained by randomly dividing the pooled data into two new groupings of the same size as the original datasets. An ALE image was created for each new dataset, subtracted from the other and then compared to the real data. The process was computed 10,000 times, and a voxel-wise *p* value image was obtained. Values in each voxel represent the position of real data with respect to the distribution of values obtained during the permutation test. To ease the comprehension of the results, ALE contrast images were converted to Z scores.

This procedure was applied to each of the aforementioned coordinate lists. In particular, we created significant maps showing conjunction and disjunction between updating and g*f*, switching and g*f*, and inhibition and g*f*. Moreover, only the conjunction maps have been created for updating and switching, updating and inhibition, and switching and inhibition (see supplementary information). Results were then expressed as clusters of activation using Z score values in the image statistics and maxima value. Anatomical labels of final cluster locations were provided by the Talairach Daemon (http://www.talairach.org/daemon.html). ALE maps were visualized using MriCronGL64 (Rorden & Brett, 2000) on an MNI standard brain.

Connectivity and behavioral analysis

fMRI datasets—In order to test the similarity between g*f* and EFs in terms of connectivity profile, two independent fMRI datasets including resting-state fMRI and cognitive data were used to provide more robust estimates. Data were collected as part of two initiatives respectively looking (i) at the possibility of enhancing g*f* via a combination of cognitive training and non-invasive brain stimulation (i.e., Flexible Adaptive Synergistic Training [FAST], a study funded under the scope of the IARPA SHARP program, collected at the Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA; "FAST" dataset hereafter), and (ii) investigating a possible link between spontaneous

fMRI connectivity, cognitive profile, and response to brain stimulation (i.e., the APOLLO study, collected at the University of Siena School of Medicine, Italy; "APOLLO" dataset hereafter). Both initiatives included the acquisition of resting-state fMRI data and behavioral assessments of g*f* and EFs. The **FAST dataset** includes 84 healthy participants (mean age 29 years, range 21–49, standard deviation [SD] = 12; mean education 15 years, range 11–23, SD = 3) with fMRI data and two g*f* measures, namely the Raven Advanced Progressive Matrices (RAPM) (Raven et al., 1998) and the Sandia matrix (Matzen et al., 2010)+. The **APOLLO dataset** includes 130 healthy participants (mean age 25 years, range 19–32, SD = 7; mean education 16 years, range 14–23, SD = 3) with fMRI data and RAPM scores. In **FAST**, the average RAPM accuracy was 0.77 (SD = 0.14), while the Sandia accuracy was 62% (SD = 17). In **APOLLO**, average RAPM accuracy was 54% (SD = 15). To provide estimates of the correlation between connectivity and behavior as test-unspecific as possible, RAPM and Sandia scores were averaged in the FAST dataset. In **FAST**, the average updating accuracy was 68% (SD = 16); the average stop-signal reaction time, a marker of inhibition ability with lower values indicating better inhibition, was 257 ms (SD = 84 ms); the average switch costs, an established indicator of switching ability with lower values indicating better switching, were 71 ms (SD = 37 ms). In **APOLLO**, the average updating accuracy was 70% (SD = 19); the average inhibition reaction time was 235 ms (SD = 63 ms); the average switch costs were 64 ms (SD = 60 ms). Details about the fMRI protocols for the FAST and APOLLO datasets, the g*f* and EF tasks used in both datasets, as well as fMRI preprocessing procedures are included in the supplementary materials.

fMRI and behavioral analysis

Seed-based analysis: First, a seed-based connectivity analysis was performed looking at the qualitative similarity of voxel-wise connectivity maps of the g*f* and EFs maps. The average BOLD time course during resting-state was retrieved by averaging the signal from all the voxels included in each EFs and g*f* ALE maps. Subsequently, the signal from each map was correlated with that of the remaining voxels in the rest of the brain, resulting in a 3D volume where each voxel value represents the correlation coefficient between its BOLD activity and that of the seed map of interest. Moreover, we also verified whether EF and g*f* maps display strong connectivity with other resting-state fMRI networks, and if EFs and g*f* maps display a stronger correlation between themselves compared with other resting-state networks (RSNs). To this end, the same seed-based connectivity procedure explained previously was performed by using RSNs maps as seed regions. Specifically, the BOLD activity from 14 RSNs (Shirer et al., 2012) was computed. The connectivity between EFs/g*f* maps and the 14 RNSs was then calculated using a multivariate general linear model (GLM).

Behavioral analysis: In an attempt to replicate previous behavioral results, individual g*f* scores were correlated (Pearson correlation coefficient) with updating, inhibition, and switching scores in the two independent datasets.

Results

ALE Meta-analysis

The results of the ALE meta-analysis are available for download as a nifti. nii volumetric file at www.tmslab.org/santalab.php. The maps include both network-level volumes representing the entire set of regions activated, e.g., for updating, as well as separate .nii files for each node composing the network. For the sake of synthesis, the lists of regions representing each map are presented in separate paragraphs. Detailed information on the anatomical localization of each significant cluster and the relative statistics are reported in dedicated figures and tables. A more in-depth discussion about the meaning of the patterns identified as well as the role of specific regions is provided in the Discussion section.

EFs maps

A summary of the anatomical profile of the ALE maps for g*f* and EFs is reported in Fig. 1. Details about each EF map are reported below and are part of the supplementary materials of this meta-analysis.

Updating—Map and coordinates for the activation pattern elicited during completion of updating tasks are shown in Figure S2 and Table S2. The map includes ten separate clusters highlighting a bilaterally distributed functional organization mainly involving (left) prefrontal and parietal lobes, with additional contribution from cerebellum, fusiform gyrus and precuneus, and subcortical structures, including lentiform nucleus and insula.

Inhibition—Map and coordinates for the inhibition tasks are reported in Figure S3 and Table S3. Consistent with many reports on right hemispheric involvement in inhibition tasks (Garavan et al., 1999), the maps include seven distinct nodes localized mostly in the (right) FPN with further activation of cingulate gyrus, superior temporal lobe, and insula.

Switching—Activations during tasks involving switching and their respective sets of coordinates are reported in Figure S4 and Table S4. Qualitatively, seven clusters with a more left lateralized activation seem to be present, mostly related to inferior and middle frontal gyrus activation, as well as activation in the inferior parietal lobule and cingulate gyrus.

Overlap within EFs—Activations overlapping between each pair of EFs also were computed, looking at e.g. brain regions activated during both updating and switching processing. Results of each pairwise comparison are displayed in Figures S5, S6, and S7 with details about each activation cluster and corresponding MNI coordinates visible in Tables S5, S6, and S7.

Conjunction and disjunction between g*f* and updating

The resulting map and coordinates for both conjunction and disjunction patterns of activation between g*f* and updating is reported in Fig. 2A and Table 1. The map of overlap includes 12 separate clusters (i.e., nodes) highlighting a bilaterally distributed functional

organization mainly involving (left) prefrontal and parietal lobes with additional contribution from precuneus and subcortical structures including insula.

Moreover, disjunctive maps of activation show a bilateral frontoparietal activation pattern, which was greater for updating than for g*f*, with additional contribution from cerebellum and subcortical regions, such as insula and claustrum, while 2 separate clusters are clearly visible for g*f* rather than for updating, which include the left inferior frontal gyrus and the right caudate (Fig. 3A; Table 1).

Conjunction and disjunction between g*f* and switching

Map and coordinates for both conjunction and disjunction pattern of activation between g*f* and switching is reported in Fig. 2A and Table 2. A more left-lateralized pattern of activation is present looking at the conjunctive maps with activation of regions placed mostly in the FPN. For the disjunctive maps, five separate clusters of bilateral frontal, parietal, and occipital regions were present for switching, whereas a single node in the left precentral gyrus characterized the pattern of activity during g*f* tasks (Fig. 3B; Table 2).

Conjunction and disjunction between g*f* and inhibition

Map and coordinates for both conjunction and disjunction pattern of activation between g*f* and inhibition are reported in Fig. 2A and Table 3. A more left-lateralized activation involving FPN is visible for the overlap map, with further participation of precuneus and insula. Disjunctive maps of activation displayed a more right-lateralized activation for inhibition involving regions of frontal, parietal, and temporal lobes. Conversely, a frontoparietal pattern of activation involving the left hemisphere was found for g*f* tasks (Figure 3C; Table 3).

Volume, coordinates, and corresponding Brodmann area, lobe, hemisphere, and regional labels are reported for each cluster included in the ALE map.

Full overlap between g*f* and EFs

Map and coordinates for the overlapping activation clusters between g*f* and EFs is reported in Fig. 4. A set of regions in the left hemisphere was identified, with coactivation in the anterior cingulate gyrus (ACC) (MNI = −2, 11, 44), middle frontal gyrus (MFG) (MNI = −50, 13, 22), and inferior parietal lobule (IPL) (MNI = −36, −61, 45).

Functional connectivity profile and behavioral data

Seed-based analysis of g*f* and EFs maps is displayed in Figs. 5 and 6, showing the similarity between g*f* and EFs connectivity patterns. As previously shown in the case of g*f* (Santarnecchi et al., 2017), EFs maps also show resemblance of frontoparietal “cognitive” networks, such as the dorsal attention network (DAN) (Corbetta & Shulman, 2002) and the frontoparietal network (FPN) (Spreng et al., 2012), and display negative connectivity with medial structures of the default mode network (DMN) (Fox et al., 2005; Fransson, 2005). Less similarity is observed between gf/EFs connectivity profile and remaining RSNs related to auditory, visual, motor, and language processing (Fig. 6).

To verify the similarity between functional connectivity patterns (Fig. 5) and behavioral data, the correlation between g*f* and EFs scores was computed for both the FAST and APOLLO datasets. Results are shown in Fig. 7, with a pattern suggesting a stronger similarity (i.e., positive correlation) between g*f* and updating, with weak or null correlation between g*f* and inhibition and switching in both FAST (updating-g*f*, r = 0.59, $p < 0.004$; switching-g*f*, r = 0.13, $p < 0.146$; inhibition-g*f*, r = 0.28, $p < 0.046$) and APOLLO (updating-g*f*, r = 0.38, $p < 0.016$; switching-g*f*, r = 0.11, $p < 0.326$; inhibition-g*f*, r = 0.18, $p < 0.389$) datasets.

Discussion

We performed a meta-analysis of 163 studies using fMRI or PET while participants completed tasks engaging the three core EFs, i.e. updating, switching and inhibition, and created functional localization maps for each function. Classifying available studies on the basis of EF components (update, inhibition, and switching) allowed us to identify spatially segregated networks of cortical and subcortical regions underlying each core EF and their overlap with brain regions associated with g*f*. The ALE meta-analysis showed greater overlap between g*f* and updating (80%) with less similarity between g*f* and switching (17%) and inhibition (34%). An analysis of behavioral data from two independent datasets also confirmed results of the ALE MRI meta-analysis, as well as previously reported behavioral associations between EFs and g*f*, with an almost exclusive positive correlation between updating and g*f* scores.

Overlap between updating and g*f*

From a psychometric point of view, previous studies on non-clinical populations have reported evidence of a close correlation between EFs and g*f* (Carpenter et al., 1990; Engle et al., 1999; Miyake et al., 2001; Salthouse et al., 1998; Salthouse et al., 2003). We demonstrate that this overlap is mostly driven by a similarity in fMRI activation patterns observed for g*f* and updating (80% overlap), with significantly smaller similarity for inhibition (34%) and switching (17%).

Our results are consistent with previous behavioral evidence (Friedman et al., 2006; Gray et al., 2003; Salthouse, 2005) in older adults (Salthouse et al., 2003), young adults (Ackerman et al., 2005), and children (Klingberg et al., 2002; Zhao et al., 2011). The relationship between updating (i.e., WM) and g*f* still represents an open debate, with multiple theories explaining their link. One of the main propositions posits a pivotal role of executive control. Engel, Kane, and Conway believe that executive control, WM and g*f* are connected to each other, and the association between WM and g*f* could be the result of the correct use of domain-wide attentional control, consisting of focusing attention on crucial task-relevant information (Engle & Kane, 2004; Kane & Engle, 2002). In essence, the stability of mental representations of task features is supported by the WM system, where such stability also allows for control and manipulation of information, which in turns facilitates reasoning ability (Shipstead et al., 2016).

On the contrary, others have postulated that the crucial cognitive mechanism underlying g*f* and updating lies in storage capacity more than stability of mental representations,

which allows one to actively maintain distinct chunks of information and flexibly construct task-relevant bindings among them (Chuderski et al., 2012). Moreover, further studies have reinforced such a concept by suggesting that storage capacity could depend on the ability to set flexible and temporary bonds between elements and their positions within a certain mental structure (Oberauer et al., 2008; Shipstead et al., 2012; Sternberg, 2008). According to this theory, storage capacity would be the common denominator between WM and g*f* and, subsequently, serve a functional role for the overlapping regions identified in our study (see below).

To examine whether individual differences in EFs are influenced by genetic or environmental influences, Friedman et al. (2008) conducted a multivariate analysis in twins. They used ACE models to analyze similarity and diversity in the genetic substrate supporting the three core EFs (updating, inhibition, and switching), as well as to study whether genetic variance in a general EFs factor "common EF" (representing the aspect of ability that is common across the three different types of EF tasks) reflects variability in intelligence. They found EF correlation to be 99% hereditable while diversity was due primarily to substantial genetic influences only in updating (56%) and switching (42%), showing that unity and diversity between EF are genetic. However, this does not mean that EF abilities are immutable, as hereditability explains only about half of the variance between tasks. Moreover, the results demonstrated different genetic substrates between "common EF" and intelligence except for some overlap with updating. This confirmed a previous finding by the same group showing significant behavioral correlations between updating and intelligence but no link between the latter and inhibiting or switching (Friedman et al., 2006). It is important to note that the measure of intelligence used was The Wechsler Adult Intelligence Scale III (WAIS-III), which primarily assesses the crystalized component of intelligence, while our research focused on the fluid component of intelligence.

In the past decade, numerous studies have used this overlap to investigate the claim that gains in WM training might transfer to gains in g*f* by means of increased updating capacity (Au et al., 2015). Despite multiple promising studies reporting g*f* enhancement, other attempts at replication failed to show any sign of cognitive transfer (Thompson et al., 2013). The heterogeneity of results across studies, which aimed to create transfer on g*f* by stimulation of EFs, could be attributed to many factors (Rudebeck et al., 2012; Stephenson & Halpern, 2013; Jaeggi et al., 2008; Redick et al., 2013; Thompson et al., 2013). For example, much of the data on how intelligence relates to different EFs comes from studies using standard clinical neuropsychological tests as measures of EF, but these often have poor reliability (Miyake et al., 2000). Shipstead et al. (2012) have proposed a series of methodological weaknesses that could explain this heterogeneity, including inadequate measurement (i.e., using a single task to measure a construct, such as g*f*), conflation of working memory with short-term memory, and inadequate control groups. Moreover, Jaeggi et al. have suggested that variability may depend on how intrinsically motivated the subjects are as well as on personality differences (Jaeggi, Buschkuehl, Shah, & Jonides, 2014), while an old pioneering work has shown that for transfer of learning to be possible one has to apply skills in a variety of different contexts, as the form in which problems are expressed can limit the extent to which well-developed skills can be seen as being relevant and be applied (Simon & Hayes, 1976). Additionally, the contributions of inhibition and switching

may be very important in transfer contexts, whereas updating could be more relevant in a novel domain. The lack of emphasis of these crucial processes may account for some of the limited transfer. Overall, there is not yet a consensus on the relationship between EF and g*f*. It also is important to consider that most psychometrical data regarding the relation between updating and g*f* have been collected via cross-sectional studies (Shipstead et al., 2016) and that well-designed training-related studies might provide causal evidence of such link. The debate about the correlation between EF and g*f* is still ongoing, and recent studies point out the idea that the lack in understanding this correlation could be related in a difference in EFs measurement (while shifting is derived from difference scores, updating and inhibition are calculated as performance in a specific task condition) (Frischkorn et al., 2019; Hedge et al., 2018; Rey-Mermet et al., 2019).

In a nutshell, our findings suggest that the overlap between EFs and g*f* observed in terms of fMRI activation patterns is not mirrored at the behavioral level, with high similarity for g*f* and updating but smaller overlap for switching and inhibition. However, even though a dominant overlap for updating and g*f* has been reported at both behavioural and neural level, the nature of our analysis does not allow to draw a causal link between neuroanatomical and behavioural similarity. Further studies should disentangle this matter.

Overlapping Core Regions for g*f* and EFs

In our analysis, we identified a set of brain regions that overlap across g*f* and each EFs functions. These regions, mostly related to a left lateralized FPN similar to what described in the P-FIT theory of intelligence (Jung & Haier, 2007), include the left dorsolateral prefrontal cortex (dlPFC; BA9/46), the left inferior parietal lobule (IPL; BA39/40), and left anterior cingulate cortex (ACC; BA8).

The central role of dlPFC both in EFs and intelligence has been amply demonstrated by multiple fMRI studies in healthy subjects and individuals with dlPFC damage (Barbey et al., 2014; Kievit et al., 2014). Spearman (1927) was the first to theorize that dlPFC has a unique functional role leading to a unified neural architecture for higher cognition (Duncan, 2010; Duncan et al., 2000). Subsequent studies support this framework, demonstrating the activation of dlPFC during EF (Duncan & Owen, 2000), general intelligence (Bishop et al., 2008; Duncan et al., 2000; Esposito et al., 1999; Prabhakaran et al., 1997), as well as g*f* tasks (Blair, 2006; Cole et al., 2012, 2015; Woolgar et al., 2010). The dlPFC is particularly involved during updating tasks but is also engaged in manipulation of information (D'Esposito et al., 1998; Haxby et al., 2000; Mencarelli et al., 2019; Smith & Jonides, 1999). Duncan (2005) confirmed the involvement of dlPFC in both updating and inhibition tasks, as well as perceptual tasks. The literature to date support our finding, suggesting left dlPFC as a crucial hub for high-order cognition.

The role of ACC has been also extensively associated with EFs in humans, given its involvement in error monitoring and top–down control over sensory (Crottaz-Herbette & Menon, 2006) and limbic brain regions (Etkin et al., 2006). Moreover, the correlation between these areas and EFs was supported by several studies underscoring the fundamental role of ACC in relating actions to their consequences (Rushworth et al., 2004), learning and predicting action outcomes, providing a control signal to other brain regions (Alexander

& Brown, 2011), as well as in late-stage aspects of response selection (Banich, 2009). These functions are easily associated with core components of brain activity during EF and g*f* tasks (Bush, 2000; Carter et al., 1998, 1999; Crottaz-Herbette and Menon, 2006). Thus, the presence of ACC among the regions showing full overlap across EFs and g*f* is not surprising. Interestingly, other authors also have stressed the potential role of ACC in modulating arousal-related processes (Braver & Barch, 2006), showing ACC as mostly activated during task-initiation cues and error cues, a potential further proof of its role in both control/monitoring of attention.

Finally, different fMRI studies have suggested that both frontal cortex and parietal regions are involved in switching (Braver et al., 2003; Crone et al., 2006; Liston et al., 2006; Sylvester et al., 2003). Several prefrontal regions seem to be involved in cognitive switching, with regional specializations depending upon the specific type of switching required (e.g., Wisconsin Card Sorting Task, attention shift, location rule switch, etc.) (Kim et al., 2012). The inferior parietal lobule (IPL), on the other hand, seems to be crucial in task switching, specifically in transforming stimulus representations into associated response codes (Andersen et al., 1997; Culham & Kanwisher, 2001). IPL is a heterogeneous area responsible for a vast array of cognitive functions, including sensory motor processing (Iacoboni, 2005; Keysers & Gazzola, 2009), executive control (Seeley et al., 2007; Uddin et al., 2011), automatic attentional process (Mark D'Esposito & Grossman, 1996; Nobre et al., 1997), WM maintenance and manipulation (Tsukiura et al., 2001), and WM processing of auditory verbal and nonverbal information tasks (Mencarelli et al., 2019; Yoo et al., 2004).

Left ACC, IPL, and MFG could represent a network of regions at the very core of high-order cognitive functioning in humans. However, focal lesions to areas with high system density and participation coefficient produce more severe and widespread cognitive deficits than focal lesions to areas of high-degree centrality (Warren et al., 2014). Furthermore, lesion-based studies should look into the differential impact of lesions to "overlapping" regions compared with regions mainly related to one specific function.

Similarity of behavior-connectivity correlational structures

Recent studies have shown how resting-state functional connectivity patterns hold predictive value over evoked brain activity (Tavor et al., 2016), suggesting the idea that spontaneous inter-regional coupling "shape" the metabolic changes required for cognitive performance. Moreover, the same principle seems to apply to behavioral performance, with evidence of a link between resting-state fMRI patterns, performance in a specific behavioral task (e.g., g*f* task) and the degree of "shaping" taking place during such task: the more an individual displays high level of performance in a given function, the more his/her functional activation sustaining such function (i.e., an fMRI activation measured in the MRI scanner during a task) is similar to resting-state activity in the MRI scanner. Additionally, Shultz and Cole (2016) showed that subjects with an optimized intrinsic network configuration for domain-general task performance are more efficient in updating functional networks, suggesting that this ability is a hallmark of high intelligence and highlighting that similarity between task and rest functional connectivity within brain networks is related to behavioral performance (Schultz & Cole, 2016; Zuo et al., 2018).

Following this reasoning, one could hypothesize that, given the previously reported positive correlation between updating and gf behavioral performance at rest—also confirmed in our two datasets—the patterns of intrinsic fMRI connectivity for gf might show more similarity with updating as well, especially compared with that with inhibition of switching. Although our results could not prove this hypothesis, the notion of the positive/negative correlation among EFs and gf is of value, e.g., planning noninvasive brain stimulation interventions where stimulation targets should be selected differently, depending on whether it precedes (i.e., it is delivered at rest) or takes place concurrently to cognitive training (i.e., when a stronger specificity of brain activation could be present; see next paragraph). These concepts should be evaluated and discussed in future studies.

Exploiting the overlap for cognitive enhancement

The present results shed light into the shared neural basis of gf and EFs and might inform studies aimed at generating transfer on gf by cognitive training programs based on EFs. Specifically, these maps could indicate the best overlapping areas between gf and EF, which, if engaged properly, might maximize the chance of cognitive transfer/enhancement in both neurotypical and atypical populations. Alternatively, interventions based on NIBS could be used to engage such regions with high spatial precision, using both magnetic or electrical transcranial stimulation (Filmer, Dux, & Mattingley, 2014; Rossi, Hallett, Rossini, & Pascual-Leone, 2009; Santarnecchi et al., 2015b; Tatti, Rossi, Innocenti, Rossi, & Santarnecchi, 2016). For example, low-voltage electrical stimulation provides an efficient tool to modulate—excite or inhibit—the activity of an entire network with potential for cognitive enhancement (Santarnecchi, Brem, et al., 2015). Previous neuromodulatory studies have reported an enhancement on gf (Santarnecchi et al., 2016; Santarnecchi et al., 2013) and working memory (Polanía et al., 2012) following stimulation of a single region in the left prefrontal lobe or a FPN. We have previously reported the effect of 40hz-tACS on the left middle frontal gyrus (MFG) inducing enhancement of gf performance (Santarnecchi et al., 2013) but not of spatial working memory abilities (Santarnecchi et al., 2016). Noninvasive brain stimulation could be used both to causally test and validate the maps of EFs-gf overlap identified in the present study, as well as to target shared EF-gf substrate to maximise the chance for transfer.

Limitations of the study and future directions

In this study, we created specific meta-analytic maps for updating, inhibit, and switching and compared them with previously published maps for gf. However, it must be considered that such maps represent the average activity over multiple tasks addressing the same function (e.g., for updating, we used fMRI data on N-back and AX-CPT). To fully leverage the power of this “functional overlap” approach, future investigations should evaluate the overlap between specific gf and EF tasks, especially when selecting the potential target responsible for cognitive transfer. Moreover, it would be interesting to validate these maps with behavioral data. Future investigations should focus on the relationship between EFs/gf maps before and after cognitive training, to understand, e.g., whether baseline correlation between EF as well as their overlap with gf are predictive of transfer over gf abilities.

Unfortunately, measuring EF is a challenging topic because of the difficulty with its definition and measurement (Jurado & Rosselli, 2007). Tasks considered specific in measuring a particular aspect of EF may not be sensitive for defining the entire process, because it often requires other EF and non-EF processes. Current evidence indicates that each EF ability (e.g., updating) can be separate into what is common across the three main EFs, i.e., "unity," and what is unique to that ability, or "diversity." This drives what is commonly known as the "impurity task" problem: an EF task is composed of (i) specific aspect of EF targeted by that task (e.g., shifting), (ii) common aspects of EF, and (iii) non-EF aspects of the task (e.g., visual processing), as well as (iv) nonsystematic variance (Snyder et al., 2015). In addition, the low reliability of EF tasks is an important issue because of the poor correlations they have with other measures (Paap & Sawi, 2016). Considering the interpretation by Miyake et al. (2000), complex EF tasks tend to have relatively low internal and/or test–retest reliability, also because subjects adopt different strategies at different times when performing the tasks. The issue of task sensitivity and reliability need to be considered, because they may lead to false negative/positive findings and conclusions, for example that EF is not impaired in a clinical group (Gustavson et al., 2020; Snyder et al., 2015). Given this background, a limitation of our results is that they cannot prove the reliability of EFs measures, but only their generalizability, because the data have been collected at two different sites. Combining multiple EF and gf tasks into larger longitudinal studies, further work is needed to understand the associations between these high-order cognitive functions.

Additionally, we collected the fMRI dataset following the recommendations given in past reports (Damoiseaux et al., 2006; Shehzad et al., 2009; Van Dijk et al., 2009). However, as pointed out in a recent study (Laumann et al., 2015), this could be a limitation when we look at resting state functional connectivity individual differences. Moreover, the FD and DVARS thresholds used for motion censoring (0.5) are both quite liberal by current standards, and this could represent a limitation in our study. However, considering that large resting-state networks with known topographies are being extracted and correlated with equally distributed and large networks (g*f*, EF), we do not believe that these thresholds have a significant influence on the results.

Conclusions

Understanding the shared neural mechanisms underlying g*f* and EFs might help to understand further the structure of high-order cognition and design better cognitive enhancement/rehabilitation approaches. We stress a major overlap between g*f* and updating compared with inhibition and switching, with a strong involvement of regions of the left FPN supporting both EFs and g*f*.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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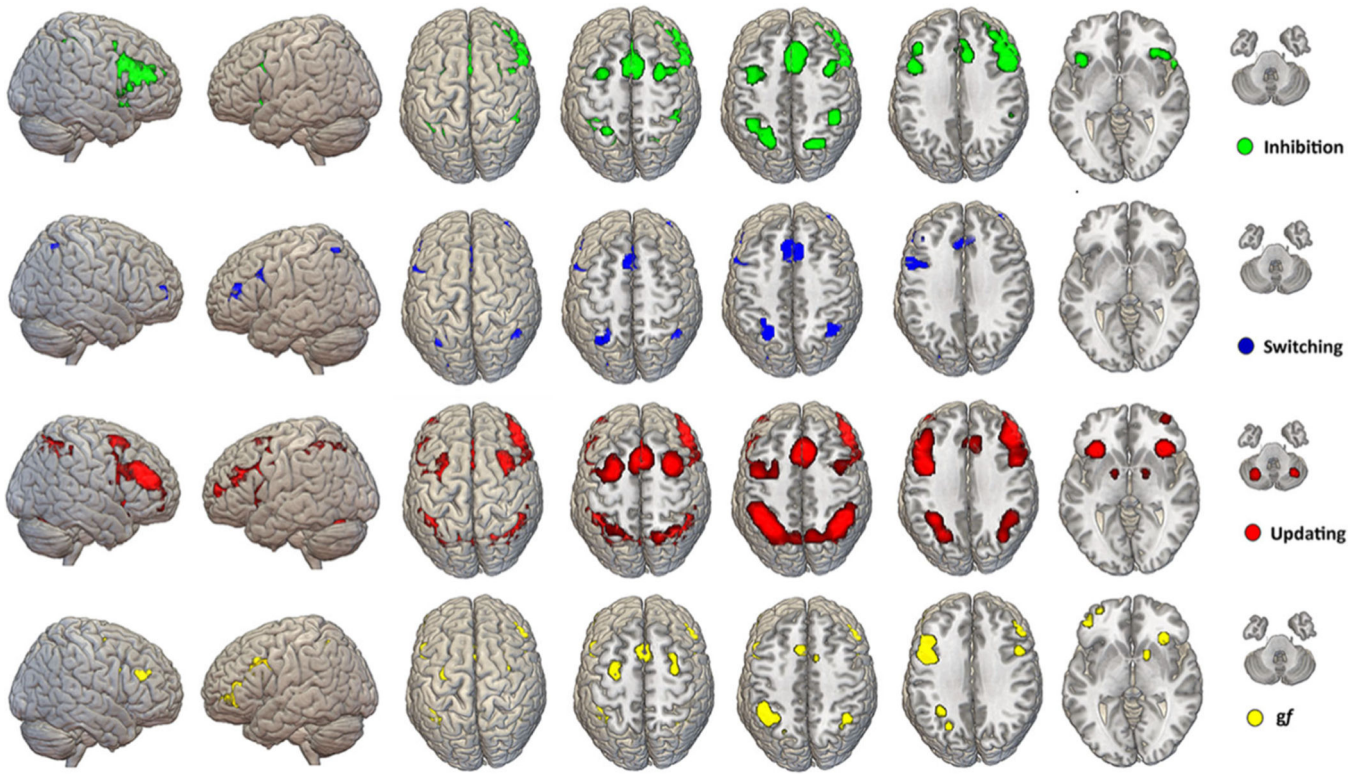

Fig. 1.

ALE maps. Results of the quantitative analysis of fMRI activation for g*f* and EFs tasks are shown on a template brain in MNI space. The maps are the results of voxel-wise analysis based on $p < 0.001$ threshold for cluster-formation and a $p < 0.05$ for cluster-level inference. On average, clusters had a dimension between 500–1,000 mm^3. Amore comprehensive depiction of each EF map is available as part of the supplementary materials of the manuscript (Figures S1-2-3; Tables S2-3-4). Note: MNI = Montreal Neurologic Institute

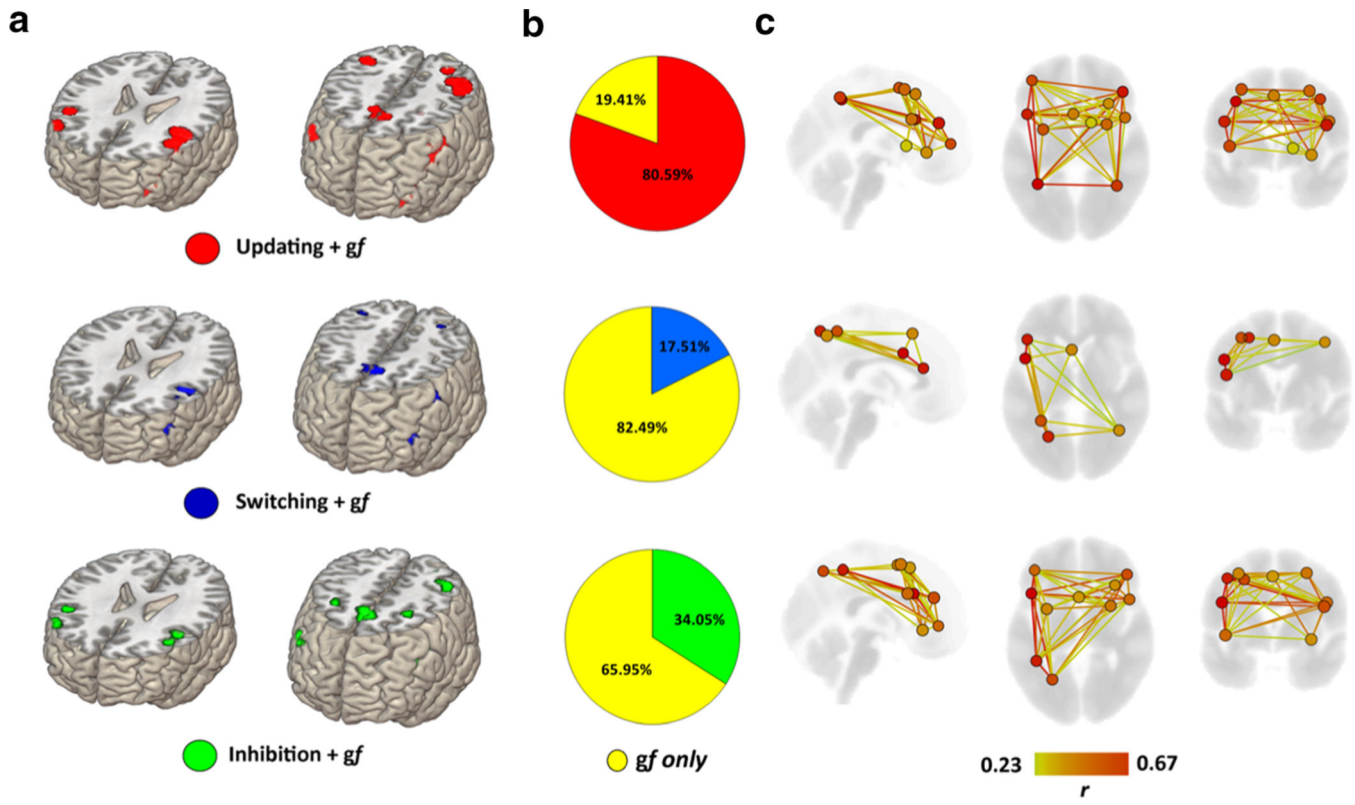

Fig. 2.

Statistical overlap between EFs and g*f*. Brain regions displaying statistically significant overlap between g*f* and each EFs are displayed (**A**), as well as the percentage of overlap of each EFs with the g*f* ALE map (**B**). A greater overlap between updating and g*f* was present, with the two networks showing strong overlap especially in bilateral frontoparietal regions (**C**). Overlapping regions are displayed as nodes of a network; their corresponding spontaneous functional connectivity pattern (Pearson *r* coefficient) also is shown, highlighting stronger and weaker connections within the overlapping nodes.

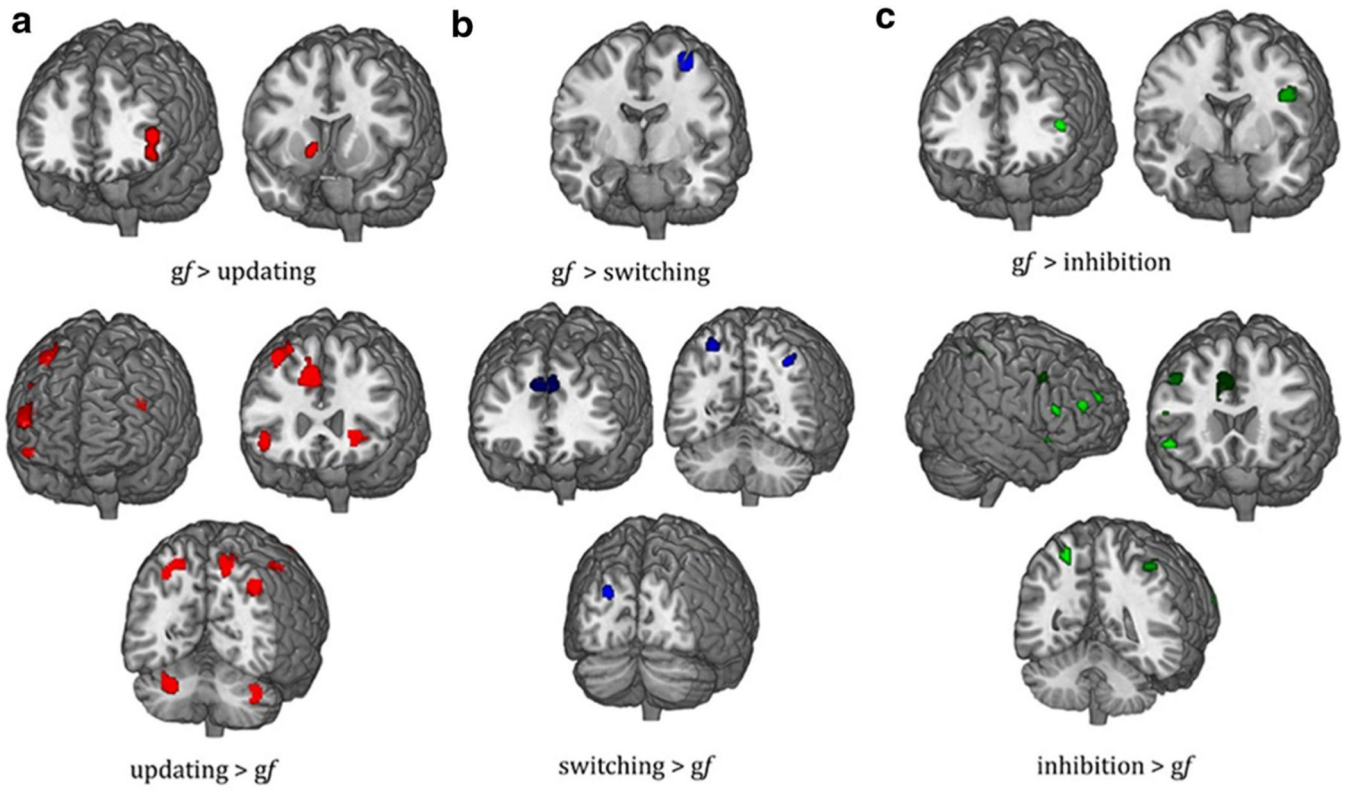

Fig. 3.

Disjunction maps for EFs and g*f*. Significant differences in brain activation for updating and g*f* (**A**), switching and g*f* (**B**), inhibition and g*f* (**C**) are reported.

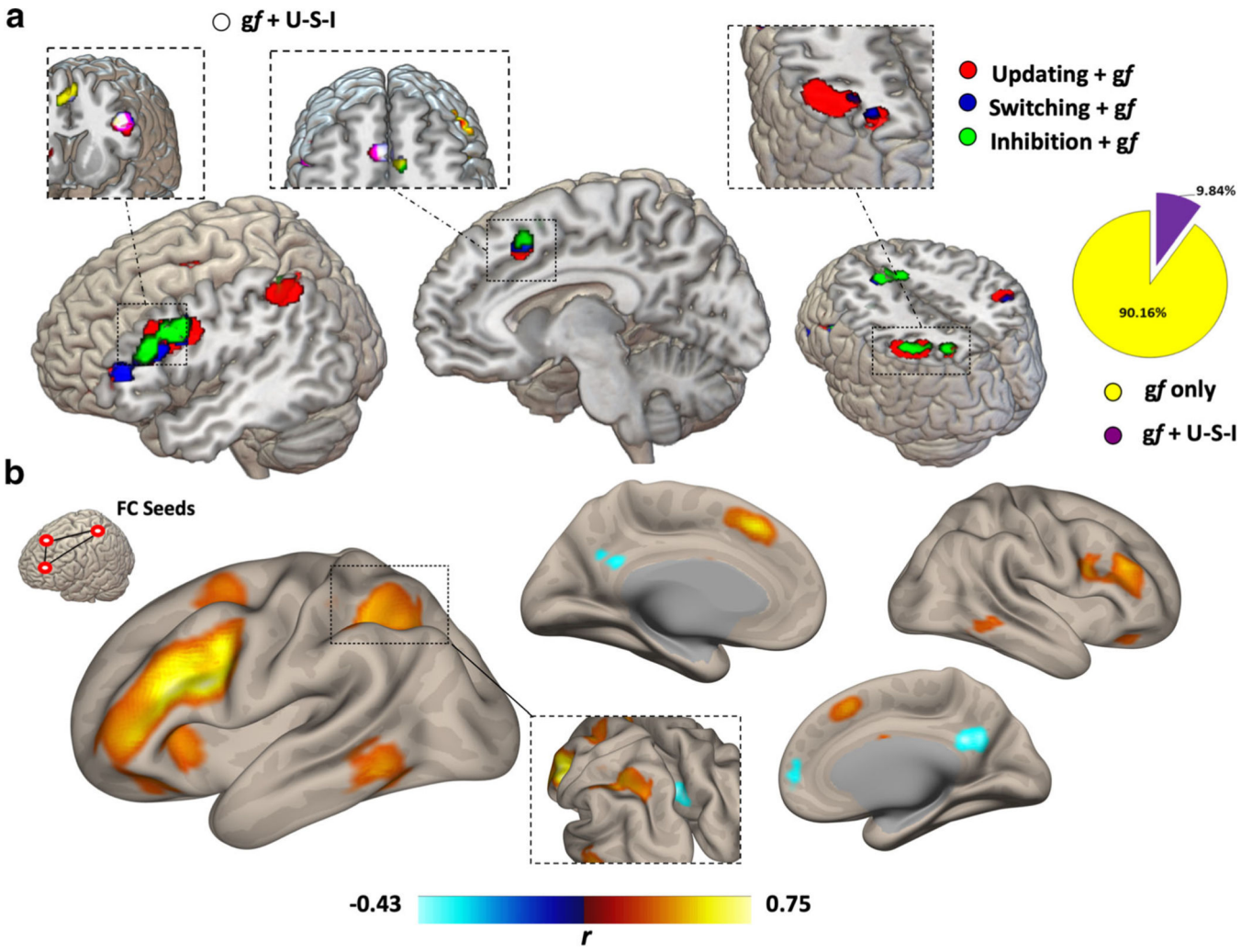

Fig. 4.

Full overlap between EFs and g*f*. A subset of brain regions composing each EFs and g*f* maps displayed full overlap across the four functions, even though only composing roughly 10% of the g*f* map (**A**). The regions included the ACC, left MFG, and left IPL, with the higher overlap displayed by the left MFG as also highlighted by looking at the functional connectivity profile of the three overlapping regions (**B**)

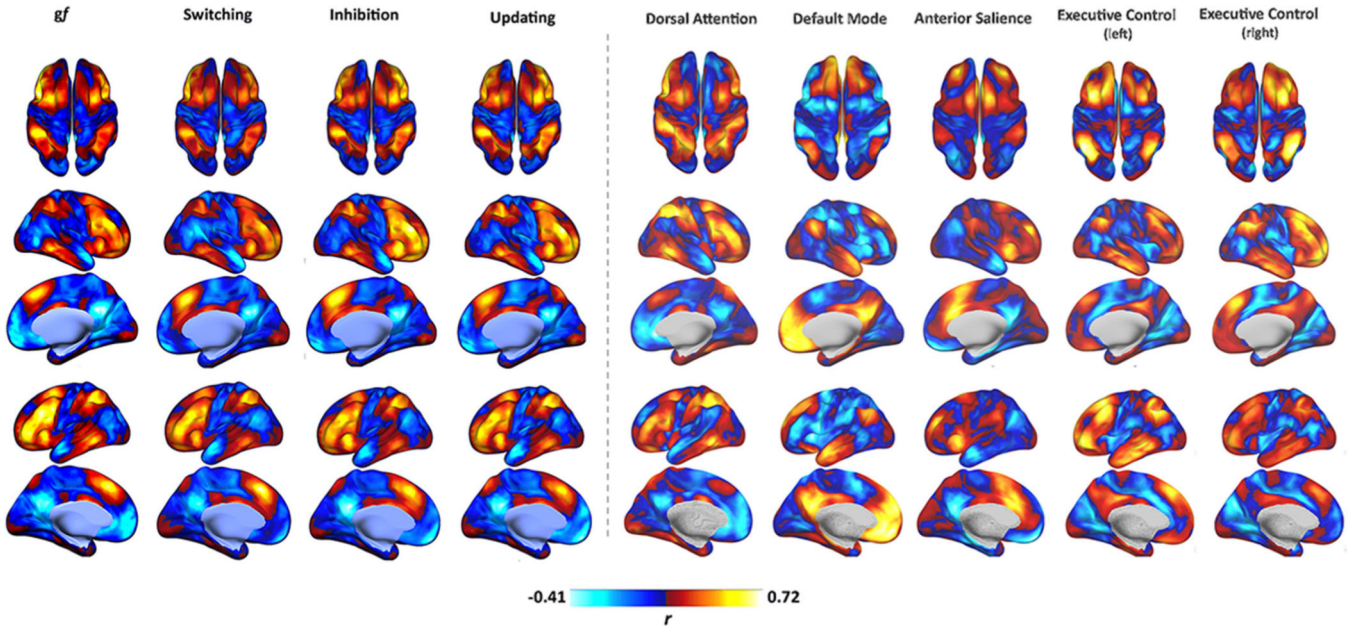

Fig. 5.

Functional connectivity profile. As visible, gf and EFs maps display high similarity in terms of their functional connectivity fMRI profile, with a strong positive connectivity between their nodes and a negative connectivity with brain regions resembling the DMN. Their overall pattern also seems to resemble those of other “cognitive” RSNs, such as the FPCN and DAN. Connectivity is expressed as correlation coefficient between the average BOLD signal extracted from each map and that of any other voxel in the brain. Data refer to the FAST dataset. DMN = Default Mode Network.

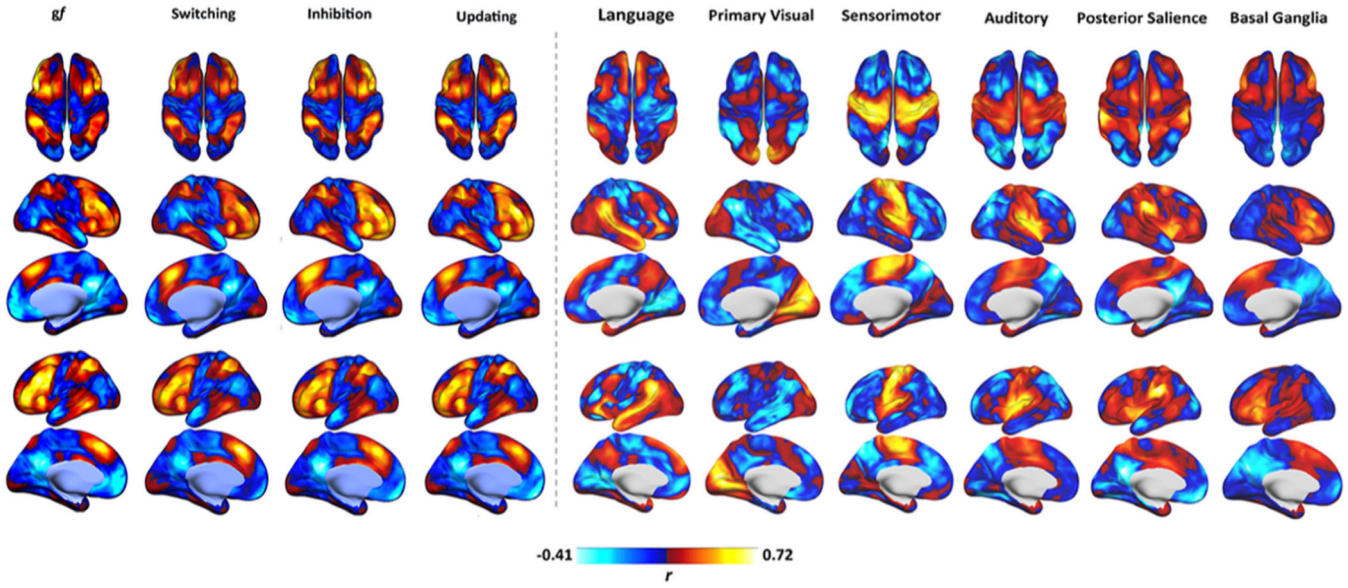

Fig. 6.

Functional connectivity profile or other RSNs. Gf and EFs maps display lower similarity with other RSNs, such as the visual, auditory, language, and motor networks. Connectivity is expressed as correlation coefficient between the average BOLD signal extracted from each map and that of any other voxel in the brain. Data refer to the FAST dataset. DMN = Default Mode Network.

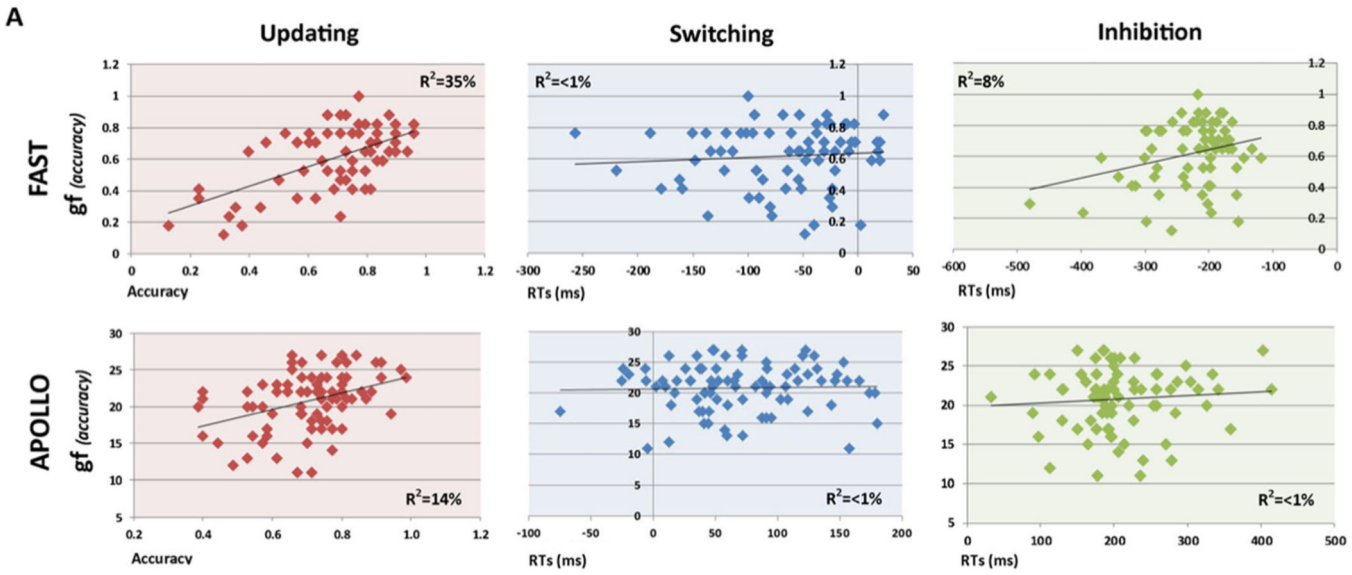

Fig. 7.

Behavioral correlation. As previously reported, g*f* scores in both the FAST (**A**) and APOLLO (**B**) datasets display a positive correlation with updating scores, while no significant correlation is present with switching and inhibition scores in both datasets.

Table 1

Updating and g*f*. Conjunction and disjunction activation pattern for updating and g*f*

Cluster number	Volume (mm^3)	Gyrus Weighted Center			Extrema Value	Extrema value coordinates			Brodmann Area	Hemisphere	Lobe	Label
		x	*y*	*z*		*x*	*y*	*z*				
conjunction coordinates												
1	6608	−47.2	16	27.1	0.045	−48	8	30	9	L	Frontal	Frontal Gyrus
					0.034	−46	24	22	46	L	Frontal	Middle Frontal Gyrus
2	5544	−37.8	−49.9	45.9	0.034	−46	−44	48	40	L	Parietal	Inferior Parietal Lobule
					0.029	−36	−48	42	40	L	Parietal	Inferior Parietal Lobule
					0.026	−24	−64	44	7	L	Parietal	Precuneus
3	2928	−1.2	15.1	49.9	0.025	−4	18	48	6	L	Frontal	Superior Frontal Gyrus
					0.021	4	10	50	6	R	Frontal	Superior Frontal Gyrus
					0.021	8	10	48	24	R	Limbic	Cingulate Gyrus
4	1088	39.1	−53.1	47.5	0.027	40	−52	48	40	R	Parietal	ParietalInferior Parietal Lobule
					0.016	34	−60	54	7	R	Parietal	Superior Parietal Lobule
5	1072	−30	−.3	56	0.020	−32	−6	58	6	L	Frontal	Precentral Gyrus
					0.019	−30	2	52	6	L	Frontal	Middle Frontal Gyrus
6	61064	32.2	25	−3.7	0.034	32	26	−4	13	R	Sub-lobar	Insula
7	784	47.9	11.5	26.9	0.028	48	12	28	9	R	Frontal	Inferior Frontal Gyrus
8	776	48.5	33.7	25.9	0.026	52	32	28	9	R	Frontal	Middle Frontal Gyrus
					0.017	44	42	30	9	R	Frontal	Middle Frontal Gyrus
9	536	30.2	4.7	54.1	0.020	30	−2	56	6	R	Frontal	Middle Frontal Gyrus
					0.019	30	10	52	6	R	Frontal	Sub-Gyral
10	408	−40.9	49.1	1.5	0.023	−42	44	10	46	L	Frontal	Middle Frontal Gyrus
					0.020	−42	50	−2	46	L	Frontal	Middle Frontal Gyrus
					0.020	−36	54	−4	10	L	Frontal	Middle Frontal Gyrus
11	280	15.5	6.8	2.7	0.023	16	8	2	.	R	Sub-lobar	Lentiform Nucleus
12	8	−36	56	−6	0.018	−36	56	−6	.	L	Frontal	Middle Frontal Gyrus
gf>updating												
1	1504	−46	42.4	.5	38.905.919	−47.3	43	−2.9	46	L	Frontal	Inferior Frontal Gyrus
2	200	13.4	9.9	−2.8	2.820.158	14	10	−4	.	R	Sub-lobar	Caudate

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Cluster number	Volume (mm^3)	GyrusWeighted Center			Extrema Value	Extrema value coordinates			Brodmann Area	Hemisphere	Lobe	Label
		x	*y*	*z*		*x*	*y*	*z*				
updating>gf												
1	3408	41.2	43.7	20.7	38.905.919	44.2	48.7	17.4	10	R	Frontal	Middle Frontal Gyrus
2	1992	−31.9	−65.2	−27.8	38.905.919	−32.1	−65.3	−25.7	.	L	Cerebellum	Uvula
3	1616	31	14.6	57.2	38.905.919	31.2	17.2	59.2	6	R	Frontal	Middle Frontal Gyrus
4	1512	8	19.3	40.9	38.905.919	10	20	40	6	R	Frontal	Middle Frontal Gyrus
					37.190.166	6	18	40	32	R	Limbic	Cingulate Gyrus
					3.540.084	11	18	40	32	R	Limbic	Cingulate Gyrus
					24.521.637	12	18	54	6	R	Frontal	Superior Frontal Gyrus
5	1424	42.5	22.7	−8.3	38.905.919	45.7	20.7	−8.2	47	R	Frontal	Inferior Frontal Gyrus
					37.190.166	46	28	−10	47	R	Frontal	Inferior Frontal Gyrus
6	1208	44.4	−44.5	49.7	3.540.084	48	−42	56	40	R	Parietal	Inferior Parietal Lobule
					2.820.158	44	−46	46	40	R	Parietal	Inferior Parietal Lobule
7	1072	35.6	−62.1	43.4	3.540.084	36	−62	42	39	R	Parietal	Angular Gyrus
8	848	−26	−57.4	54.5	32.905.266	−20	−58	56	7	L	Parietal	Precuneus
					29.112.377	−30	−58	48	7	L	Parietal	Superior Parietal Lobule
9	760	15.6	−62.4	58.4	37.190.166	18	−60	61	7	R	Parietal	Superior Parietal Lobule
					34.316.144	16	−60	56	7	R	Parietal	Precuneus
10	704	−40	39.8	25.4	34.316.144	−38	44	25	9	L	Frontal	Superior Frontal Gyrus
					31.213.892	−36	40	24	9	L	Frontal	Middle Frontal Gyrus
11	624	38.6	−65.6	−32.8	32.388.802	40	−70	−34	.	R	Cerebellum	Cerebellar Tonsil
12	400	−44.4	12.1	13.9	29.112.377	−44	12	12	13	L	Sub-lobar	Insula
13	368	−28.1	25.1	−3.3	2.820.158	−26	26	−4	.	L	Sub-lobar	Claustrum
14	328	7.7	35.1	29	29.888.823	10	38	30	9	R	Frontal	Medial Frontal Gyrus
15	248	48.7	16.6	13	29.290.497	48	16	12	13	R	Sub-lobar	Insula
16	208	39.6	57.2	−9	30.114.539	40	60	−12	10	R	Frontal	Medial Frontal Gyrus

Table 2

Switching and g*f*. Conjunction and disjunction activation foci for flexibility and g*f*

Cluster number	Volume (mm^3)	Gyrus Weighted Center			Extrema Value	Extrema value coordinates			Brodmann Area	Hemisphere	Lobe	Label
		x	*y*	*z*		*x*	*y*	*z*				
conjunction coordinates												
1	1592	−45.6	30.9	15.8	0.024	−44	28	18	46	L	Frontal	Middle Frontal Gyrus
					0.024	−46	22	18	46	L	Frontal	Middle Frontal Gyrus
					0.023	−46	40	10	46	L	Frontal	Middle Frontal Gyrus
2	1352	−49.6	10.6	30.8	0.026	−52	10	34	9	L	Frontal	Middle Frontal Gyrus
					0.019	−46	10	26	9	L	Frontal	Inferior Frontal Gyrus
3	1328	−2.6	17.9	47.9	0.025	−4	18	48	6	L	Frontal	Superior Frontal Gyrus
4	200	39	−54.9	49.2	0.021	40	−54	50	7	R	Parietal	Inferior Parietal Lobule
5	48	−31.3	−50.3	50.6	0.017	−32	−50	50	40	L	Parietal	Inferior Parietal Lobule
6	40	−25.6	−61.2	48	0.018	−26	−62	48	7	L	Parietal	Superior Frontal Gyrus
7	16	−25	−64	50	0.015	−26	−64	50	7	L	Parietal	Superior Frontal Gyrus
8	8	42	−58	46	0.014	42	−58	46	39	R	Parietal	Inferior Parietal Lobule
gf>switching												
1	696	−32.3	−4.3	58.5	29.112.377	−34	−2	58	6	L	Frontal	Precentral Gyrus
switching>gf												
1	1904	−2.2	31.8	34	38.905.919	−6	36	34	6	L	Frontal	Medial Frontal Gyrus
					37.190.166	6	36	30	6	R	Frontal	Medial Frontal Gyrus
					34.316.144	−8	30	28	32	L	Limbic	Cingulate Gyrus
					3.352.795	−8	26	31	32	L	Frontal	Cingulate Gyrus
2	888	−25.8	−57.7	55.1	34.316.144	−21.3	−58.7	54.7	7	L	Parietal	Superior Parietal Lobule
					3.352.795	−26	−60	56	7	L	Parietal	Superior Parietal Lobule
3	448	34.8	53.7	7.1	34.316.144	32	50	10	10	R	Frontal	Superior Frontal Gyrus
					27.163.806	34	58	2	10	R	Frontal	Middle Frontal Gyrus
					26.437.218	38	58	4	10	R	Frontal	Middle Frontal Gyrus
4	416	−21.1	−84.4	26.3	2.833.787	−20	−86	22	18	L	Occipital	Occipital Gyrus
					26.968.443	−20	−80	26	18	L	Occipital	Cuneus
5	216	37.7	−62.5	45.3	26.968.443	40	−64	45	19	R	Parietal	Precuneus

Volume, coordinates, and corresponding Brodmann area, lobe, hemisphere, and regional labels are reported for each cluster of contrasts between flexibility and g*f*.

Table 3

Inhibition and g*f*. Conjunction and disjunction activation pattern for inhibition and g*f*

Cluster number	Volume (mm^3)	Gyrus Weighted Center			Extrema Value	Extrema value coordinates			Brodmann Area	Hemisphere	Lobe	Label
		x	*y*	*z*		*x*	*y*	*z*				
conjunction coordinates												
1	12672	−.2	14.6	50.4	0.025	−4	18	48	6	L	Frontal	Superior Frontal Gyrus
					0.021	4	10	50	6	R	Frontal	Superior Frontal Gyrus
					0.021	8	10	48	24	R	Limbic	Cingulate Gyrus
2	2344	−46	18.4	27.3	0.037	−46	10	32	9	L	Frontal	Inferior Frontal Gyrus
					0.032	−46	26	22	46	L	Frontal	Middle Frontal Gyrus
					0.025	−48	20	28	9	L	Frontal	Middle Frontal Gyrus
					0.019	−40	26	32	9	L	Frontal	Middle Frontal Gyrus
3	1128	−41	−46.6	49.5	0.030	−42	−48	50	40	L	Parietal	Inferior Parietal Lobule
					0.029	−44	−44	50	40	L	Parietal	Inferior Parietal Lobule
4	648	48	12.1	27.1	0.028	48	12	28	9	R	Frontal	Inferior Parietal Lobule
5	632	48.3	33.4	25.3	0.025	50	32	26	9	R	Frontal	Middle Frontal Gyrus
					0.017	44	40	28	9	R	Frontal	Middle Frontal Gyrus
6	432	29.8	6.	53.2	0.019	30	−2	54	6	R	Frontal	Middle Frontal Gyrus
					0.019	30	10	52	6	R	Frontal	Sub-Gyral
7	384	−29.3	2.7	53.3	0.019	−30	2	52	6	L	Frontal	Middle Frontal Gyrus
8	296	−24.7	−63.4	47.2	0.025	−24	−64	46	7	L	Parietal	Precuneus
9	232	34	25.8	−6.3	0.026	34	26	−6	13	R	Sub-lobar	Insula
10	136	−42	38.4	3.8	0.024	−42	40	4	46	L	Frontal	Inferior Frontal Gyrus
11	8	−42	40	−2	0.016	−42	40	−2	46	L	Frontal	Inferior Frontal Gyrus
inhibition>gf												
1	1400	7.2	19.1	19.136.1	38.905.919	5.6	16.4	38.8	24	R	Limbic	Cingulate Gyrus
					37.190.166	9.3	16.7	36.7	32	R	Limbic	Cingulate Gyru
					32.388.802	12	22	22	32	R	Limbic	Cingulate Gyrus
					30.356.724	10	28	22	24	R	Limbic	Anterior Cingulate
2	1184	42.6	13.5	39.8	38.905.919	40.8	18.8	40.4	6	R	Frontal	Middle Frontal Gyrus
					37.190.166	41.8	10.4	40	6	R	Frontal	Middle Frontal Gyrus

Cluster number	Volume (mm^3)	Gyrus Weighted Center			Extrema Value	Extrema value coordinates			Brodmann Area	Hemisphere	Lobe	Label
		x	*y*	*z*		*x*	*y*	*z*				
					3.540.084	48	14	40	8	R	Frontal	Middle Frontal Gyrus
3	672	43.2	−37.5	54.1	38.905.919	41.1	−36.6	54.6	40	R	Parietal	Inferior Parietal Lobule
4	632	5.6	37.3	28.9	38.905.919	3.5	39	23.5	32	R	Limbic	Cingulate Gyrus
					37.190.166	6	40	28	9	R	Frontal	Medial Frontal Gyrus
					34.316.144	6	40	34	6	R	Frontal	Medial Frontal Gyrus
					30.902.324	8	38	38	8	R	Frontal	Medial Frontal Gyrus
5	592	48.5	22.3	−8.2	38.905.919	48	24	−8	47	R	Frontal	Inferior Frontal Gyrus
					3.540.084	48	20	−9	47	R	Frontal	Inferior Frontal Gyrus
					32.388.802	54	14	−10	22	R	Temporal	Superior Temporal Gyrus
					29.478.426	54	10	−6	22	R	Temporal	Superior Temporal Gyrus
6	512	−23	−54.9	58.4	38.905.919	−20.7	−54	60.7	7	L	Frontal	Precuneus
7	384	54.4	27.4	14.8	14.837.190.166	56.7	27.3	14	45	L	Frontal	Inferior Frontal Gyrus
					29.478.426	50	22	14	46	R	Frontal	Middle Frontal Gyrus
8	336	44.6	47	17.1	38.905.919	44	50	17	10	R	Frontal	Middle Frontal Gyrus
9	312	28.3	54	22.8	38.905.919	28.6	53.5	23.2	10	R	Frontal	Superior Frontal Gyrus
10	208	−37.6	43.1	20.6	37.190.166	−37	41	21	9	L	Frontal	Middle Frontal Gyrus
					32.905.266	−s38	46	22	9	L	Frontal	Middle Frontal Gyrus

Volume, coordinates, and corresponding Brodmann area, lobe, hemisphere, and regional labels are reported for each cluster included in the ALE map.