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Structural and functional brain correlates of theory of mind impairment post-stroke

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ABSTRACT

The ability to understand the mental states of others – also known as Theory of Mind (ToM) – is critical for normal social interactions. We combine behavioural probes with structural and functional brain imaging to provide the first comprehensive analysis of ToM deficits following stroke using the Reading the Mind in the Eyes Test (RMET). First, fMRI was used to identify the functional brain network involved in a non-clinical cohort. Results indicated that, relative to a control task, the RMET increased activity in a widespread functional bilateral network comprising frontal and temporo-parietal areas. To investigate how damage to grey and white matter components of this network can lead to ToM impairment, parcel-based lesion-symptom mapping (PLSM), white-matter tract-wise statistical analysis (TSA) and disconnectome symptom mapping (DSM) were performed using structural images from 64 stroke patients. PLSM results revealed that low scores on the RMET were associated with damage centered around the right posterior frontal gyrus and insula. TSA and DSM results further revealed that low RMET scores were associated with damage to white-matter tracts connecting frontal and temporo-parietal components of the RMET functional network. Together, these findings suggest that making judgements about the mental states of others imposes demands on a large functional network that can easily be disrupted, both by damage to grey matter areas that form part of the network directly, or the white-matter pathways that connect them.

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1. Introduction

A central tenet of the social brain hypothesis is that humans' unusually large brains may have evolved to meet the particular challenges of our complex social world (Dunbar, 2014). In light of the importance attributed to social cognitive function, a considerable body of literature has now emerged focused on the neuroscience of specific social cognitive abilities such as theory of mind (ToM). ToM refers to the ability to make inferences about the mental states of others, such as their beliefs, intentions, desires, and emotions, and to use this knowledge to understand and predict others' behaviour in a meaningful way (Frith & Frith, 2005). While we often take this 'mentalising' ability for granted, brain damage or dysfunction can lead to marked impairments that profoundly impact people's everyday lives by leading to difficulties in social interaction and communication (Martín-Rodríguez & León-Carrión, 2010; Moreau, Rauzy, Viallet, & Champagne-Lavau, 2016).

The eyes play a particularly important role in social interaction, often providing crucial information for decoding and understanding the mental states of others (Moor et al., 2012). The Reading the Mind in the Eyes Test (RMET) was developed as an advanced test of ToM that targets complex facial emotion recognition and mental state attribution processes (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). During this test, participants view black and white photographs that depict the eye region of protagonists' faces, and are asked to select from four options to identify each person's mental state. While face and emotion recognition are important components of the RMET, the task extends beyond simple emotion recognition tasks such as the Ekman faces (Ekman & Friesen, 1976) that depict the six basic emotions (disgust, anger, fear, surprise, sadness and happiness). In the RMET, participants are asked to determine what the person is thinking or feeling with choices including not only emotional but also cognitive responses that entail mental states (e.g., reflective, serious, playful, aghast, arrogant), far more complex than the basic emotions. An important strength of the RMET is that, in contrast to many other ToM tasks, it places only minimal demands on secondary cognitive operations such as working memory, top-down attention, and abstract reasoning. Consequently, it is often preferred for use in clinical populations that present with other types of neurocognitive impairment (Couto et al., 2013; Eddy & Rickards, 2015; Henry, von Hippel, Molenberghs, Lee, & Sachdev, 2016). The RMET is also frequently used over other ToM tasks because, rather than abstract inference alone, it also involves inferring mental states from facial cues, a naturalistic ToM skill used in everyday interactions. To date, the RMET has been used in over 250 studies, and has been reported to have good psychometric properties (Baron-Cohen et al., 2015; Fernandez-Abascal, Cabello, Fernandez-Berrocal, & Baron-Cohen, 2013; Vellante et al., 2013). For these reasons, the RMET was selected as the measure of ToM capacity in the present study. As detailed next, we were specifically interested in identifying the neural network involved when completing the RMET, and more precisely delineating how this particular ToM network can be affected by brain damage arising from stroke.

The neural substrates of ToM have been investigated in more than 400 neuroimaging studies (Koster-Hale & Saxe, 2013), which is reflective of the high degree of interest in this topic. The most consistent brain regions implicated are the dorsomedial prefrontal cortex (dmPFC) and bilateral temporoparietal junction (TPJ; Van Overwalle, 2009). Unsurprisingly, these studies have operationalised ToM using an extensive and varied range of measures. Depending on which ToM task is used (e.g., stories, cartoons, photographs, RMET, videos, animations or interactive games), additional regions such as the posterior super temporal sulcus (pSTS), superior and middle temporal gyrus (STG/MTG), temporal pole (TP), posterior inferior frontal gyrus (pIFG), ventromedial prefrontal cortex (vmPFC) and precuneus are also recruited (Molenberghs, Johnson, Henry, & Mattingley, 2016; Schurz, Radua, Aichhorn, Richlan, & Perner, 2014).

At least two distinct theories have been proposed to explain the brain networks involved in ToM functions: Simulation-Theory and Theory–Theory (Bohl & van den Bos, 2012; Coricelli, 2005; Keysers & Gazzola, 2007). Simulation-Theory (ST) suggests that individuals use their own minds as a model, and attribute mental states to others by implicitly imagining themselves in others' shoes. They are thus able to 'simulate' others' mental processes, allowing an automatic and almost instantaneous primitive understanding of another person's mind (Bohl & van den Bos, 2012; Völlm et al., 2006). According to ST, mental state attribution recruits areas such as the pIFG, rostral inferior parietal lobule (rIPL), pSTS, as well as emotion-related areas including the insula, amygdala and cingulate gyrus (Molenberghs, Cunnington, & Mattingley, 2012). It is important to highlight that pIFG and rIPL have also been implicated in affective aspects of ToM, including emotional contagion (Shamay-Tsoory, 2011), mirroring others' emotions (Pfeifer, Iacoboni, Mazziotta, & Dapretto, 2008) and, more broadly, empathy (Bernhardt & Singer, 2012). The contention here is that the ST network provides observers with direct insight into the mental states of others through simulation without the need for complex cognitive reasoning (Keysers & Gazzola, 2009; Rizzolatti & Fabbri-Destro, 2008).

By contrast, a central tenet of Theory–Theory (TT) is that ToM reasoning is supported by a constructed set of concepts (goal, desire, belief, et cetera), as well as governing rules or principles about how these concepts interact. This level of mentalising is relatively slower, cognitively taxing, and voluntary. In this way, mental state understanding is achieved through reasoning about others' likely states of mind (Saxe, 2005, 2006). TT accounts of ToM have implicated activity in a network of brain regions including the dmPFC, TPJ, as well as the bilateral TPs (Amodio & Frith, 2006; Gallagher & Frith, 2003; Saxe, 2005; Van Overwalle, 2009).

There is now a large body of literature showing that relatively more cognitive ToM tasks (such as stories, cartoons, and interactive games) rely predominantly on the TT network. However, fMRI meta-analyses have shown that the RMET implicates components of both the ST network such as the pIFG, pSTS and insula, as well as areas of the TT network such

as the TPJ, TP, and dmPFC (Molenberghs et al., 2016; Schurz et al., 2014). This is arguably because the RMET (contrary to most ToM tasks that involve stories, cartoons or interactive games) includes facial expressions of others which can be relatively easily simulated through the ST network. However, the slower and more deliberate TT network is also recruited as participants are additionally required to contemplate and explicitly report a person's mental state during the RMET.

While the brain networks involved in ToM are relatively well understood, surprisingly little is known about how damage to these networks impacts capacity for ToM. One clinical population that can shed particularly important insights are stroke patients. This is because stroke patients, as reported in several studies, are impaired on a broad range of ToM tasks, relative to non-clinical controls (Balaban, Friedmann, & Ziv, 2016; Happé, Brownell, & Winner, 1999; Muller et al., 2010; Shamay-Tsoory & Aharon-Peretz, 2007; Xi et al., 2013; Yeh & Tsai, 2014). The current study aims to further examine the relationship between *specific* lesion sites and ToM deficit, focusing in particular on the critical sites necessary for intact ToM performance.

As noted previously, prior literature has predominantly relied on fMRI to infer the brain regions underpinning ToM. However, interpretation of fMRI research is limited in that the brain regions involved in a task may not necessarily be crucial for that task (Rorden & Karnath, 2004). This is because identifying a correlation between task performance and brain activation does not establish that the region in question is necessary for the task, or the cognitive ability in general: it is possible that activation of certain regions only occurs because of their connections to other crucial regions (Rorden & Karnath, 2004). For example, Bird, Castelli, Malik, Frith, and Husain (2004) described a patient with extensive damage to the bilateral medial frontal lobe. Although this particular region has been frequently implicated in ToM function, the patient exhibited no signs of ToM impairment on a range of tasks. Another limitation of functional imaging studies is that they cannot detect the contribution of cortical regions that are constantly active irrespective of the task. Just because a cognitive task does not modulate blood flow to a particular brain region does not mean that the region is not involved in the task (Rorden & Karnath, 2004). These factors highlight the importance of not relying exclusively on functional neuroimaging studies when inferring the critical regions involved in a particular function (Molenberghs, Gillebert, Peeters, & Vandenberghe, 2008).

In contrast to fMRI, examining lesions in circumscribed areas of the brain can specify whether particular brain regions are *necessary* for particular behaviours. Lesion studies have a long history of yielding valuable information about the relationships between brain and behaviour. More recently, voxel-based lesion-symptom mapping (VLSM) and parcel-based lesion-symptom (PLSM) techniques have vastly improved the detailed knowledge that can be obtained via lesion mapping methods (Bates et al., 2003; Rorden, Karnath, & Bonilha, 2007; Hillis et al., 2018). However, the diffuse nature of lesions and the heterogeneous pathology of patients can potentially make it difficult to attribute specific behavioural changes to damage in a particular region (Carrington & Bailey, 2009). Furthermore, focal lesions can produce dramatic functional changes in other intact regions, presumably due to white-matter damage, further complicating the interpretation of lesion studies (Bartolomeo, Thiebaut de Schotten, & Doricchi, 2007; Corbetta, Kincade, Lewis, Snyder, & Sapir, 2005; He et al., 2007; Wang & Olson, 2018).

Both neuroimaging studies and lesion mapping techniques possess inherent limitations. Therefore, in the current study we used a multi-method design that leverages the complementary strengths of each approach, while simultaneously circumventing their specific limitations. First, an fMRI experiment was conducted on non-clinical controls, to determine the functional brain regions typically involved in the RMET. Participants were scanned during an fMRI-modified version of the RMET and a control task, allowing isolation of brain regions involved in ToM. It was expected that healthy individuals would show increased BOLD responses in both ST and TT regions in response to the RMET.

Second, we used PLSM to analyse the relationship between tissue damage and RMET performance on a region-by-region basis in a sample of stroke patients. The power of VLSM studies tends to be low because of the high number of voxels one has to correct for when performing the statistical analysis, thus creating a multiple comparison problem (Kimberg, Coslett & Schwartz, 2007). To increase power in our analyses, we therefore used PLSM, which instead of voxels uses larger regions of intrest (ROIs) as test units and thus significantly reduces the multiple comparison problem. PLSM and VLSM are preferable to traditional lesion study techniques because continuous behavioural measures can be used rather than binary categorisations (Bates et al., 2003; Rorden et al., 2007). Furthermore, lesions under this analysis are not restricted to pre-determined regions, allowing for inclusion of a larger number of participants, consequently increasing the reliability of analyses. It was expected that damage to specific lesion sites that overlapped or connected the functional regions involved in the fMRI ToM task would be associated with worse RMET performance.

An important limitation of PLSM and VLSM analyses is that they assume complete independence of all regions or voxels and perform the statistical tests without attempting to capture any potential existing correlations. However, damage to one region or voxel is necessarily contingent on damage to another non-adjacent region or voxel if they happen to pertain to the same damaged axon (Thiebaut de Schotten et al., 2014). Moreover, if white-matter tracts are damaged, behavioural disturbances may very well be due to the disconnection of cortical regions, irrespective of whether the regions themselves are damaged. It is becoming increasingly accepted that high level cognitive functions such as ToM are subserved by a dynamic interaction of distributed brain areas operating in large-scale neurocognitive networks (Bressler & Menon, 2010). Consequently, while the location of grey matter nuclei in the cortex are well-suited for PLSM or VLSM statistical analyses, the investigation of white-matter organisation and damage requires specific statistical analysis at the tract level (Thiebaut de Schotten et al., 2014).

Therefore, we also used an atlas of white-matter tracts based on a diffusion tensor imaging dissection of the human brain to take probabilistic measurements of white-matter tract disconnections. Specifically, using the newly developed

tract-wise statistical analysis (TSA; Foulon et al., 2018), we were able to provide detailed analyses of the relationships between ToM difficulties and lesioned white-matter pathways. Moreover, we generated a voxel-wise map of the disconnections for each patient, a *disconnectome*, and assessed its relationship with the RMET scores, a novel approach known as disconnectome symptom mapping (DSM; Foulon et al., 2018). These results were then qualitatively compared with the functional areas obtained from the healthy controls, to evaluate whether any reduction in white-matter integrity can provide a link between functional centres observed during fMRI, and the lesion sites associated with those changes. Importantly, TSA and DSM require as input structural, T_1-weighted data only, greatly expanding what scientists can learn about the impact of lesions to the brain from a single modality.

In summary, the present study used a unique and innovative multi-method approach to explore the functional regions involved in ToM in healthy participants, and the lesion correlates of ToM deficits post-stroke. We integrated fMRI, PLSM and white-matter TSA/DSM to determine the location of brain lesions both in the grey-matter cortical architecture as well as white-matter pathways that correlate with decreased ToM performance on the RMET.

2. Methods

2.1. Participants

All participants gave written informed consent according to the Declaration of Helsinki. Ethical review committees of the University of Queensland, the Metro North Hospital and Health Service (Brisbane, Australia), and the Metro South Hospital (Brisbane, Australia) approved the study.

2.2. Healthy participants

Twenty healthy older individuals (10 male, aged 51–84, $M = 68.40$, $SD = 8.54$) were recruited through the University of Queensland database, local clubs and word-of-mouth. A sample size of 20 participants was chosen a priori because it provides 92% power to detect a large effect size of $d = .8$ in a one-sample *t*-test fMRI experiment at $p < .05$ (Yarkoni & Braver, 2010). The healthy controls and stroke patients did not differ significantly in age, $t(82) = 1.69$, $p = .09$. All participants were right-handed native English speakers. None reported a history of psychiatric or neurological disease.

2.3. Stroke patients

VLSM or PLSM analyses typically require examining a relatively large group of patients (Rorden et al., 2007). This is due to the inherent variability in lesion volume and extent as well as difficulties in computing the true functional extent of a lesion (Rorden et al., 2007). Rorden et al. (2007), who developed the software, used a sample of 63 patients in their original VLSM paper, which can be considered a rough guideline for adequate sample size (see also Schwartz et al., 2009, who used 64 participants in their VLSM study). Ninety-five stroke patients with focal lesions were recruited as inpatients during their initial hospital admittance (days since admission: $M = 6.35$, $SD = 5.82$). All were recruited from the Princess Alexandra and Royal Brisbane and Women's Hospitals in Brisbane, Australia. None had any premorbid history of neurological disease, including stroke. Patients who did not have English as their first language were excluded. Additional exclusion criteria (determined a priori) included expressive dysphasia and spatial neglect, as these could potentially influence performance on the RMET. Language deficits were assessed using a naming task, which included a list of 10 common items (e.g., frog, banana, scissors; Snodgrass & Vanderwart, 1980). Eleven subjects unable to name at least nine of the objects from the list were excluded from further analysis. Spatial neglect was assessed with the Bells Test, using the Centre of Cancellation (CoC) task paradigm (Rorden & Karnath, 2010). Eighteen subjects scoring greater than .081 or less than −.081, indicating neglect behaviours, were excluded from any subsequent analysis (Rorden & Karnath, 2010). Finally, given that this analysis examined ToM deficits in the acute stages of stroke, two patients who were tested at a time point more than two standard deviations above the average of the cohort were also excluded. Together, a total of 31 patients (33%) were excluded, leaving 64 for subsequent investigation and analysis (42 male, aged 25–88, $M = 62.16$, $SD = 15.74$). Nine patients had a hemorrhagic stroke, while the other fifty-five patients had an ischemic stroke.

2.4. RMET

ToM was assessed using the revised version of Baron-Cohen et al.'s (2001) RMET. A computerised version was programmed using E-Prime 2.0 Software (Psychology Software Tools, Pittsburgh, 2005) and run on a Dell laptop computer. Verbal and written instructions were given by the experimenter and on the computer screen, respectively. The task consisted of 36 trials. In each trial, the participant was presented with a black and white photograph that showed the eyes region of a human face (Fig. 1A). Each photograph was surrounded by four centred words (two above, two below), one of which correctly identified the mindset of the person depicted. We decided to put the words in the centre of the photograph, two above and two below, rather than two on the left and two on the right side as in the traditional version of the RMET. The reason for this was to make sure that any residual left versus right visual field perceptual differences in patients (that were not detected by our neglect task), would not affect the performance on the RMET. On each trial, the participant was asked to indicate which word they felt best described what the person in the photograph was thinking or feeling. The RMET is a reliable and validated measure of ToM (Fernandez-Abascal et al., 2013; Olderbak et al., 2015). The test has been estimated to have an internal consistency of $\alpha = .61$, and good test-retest reliability (ICC = .83), as well as adequate convergent and discriminant validity (Vellante et al., 2013). The maximum possible score was 36 (100%), with less than 5% of the healthy adult population scoring below 21 (58.33%; Baron-Cohen et al., 2001). Scores below this cut-off were considered abnormal, and indicative of ToM impairment.

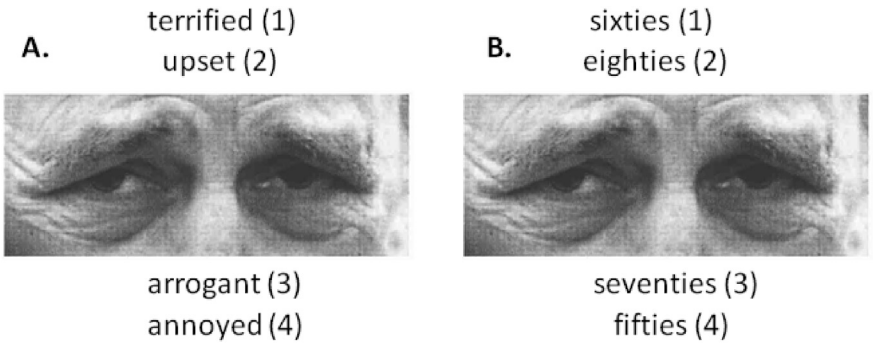

Fig. 1 – A. In the "mind" (RMET) condition, participants have to identify what a person is thinking or feeling. The four possible responses are presented in the middle of the visual field, two above the photograph, and two below. B. During the "age" control condition, participants have to indicate the age range of the person.

2.5. *fMRI-modified RMET*

The fMRI-modified RMET included an additional "age" condition with identical stimuli to the original task (referred to as the "mind" condition). The "age" condition served as a non-ToM control task, and involved asking participants to select the most likely age range of the person in the picture (Fig. 1B). Thus, the "age" control condition did not involve mentalising, and neural activity from this condition was subtracted from that of the "mind" condition to isolate the brain regions involved in ToM.

The fMRI RMET consisted of 108 trials per condition (216 in total), including four runs of 27 trials. Stimuli were selected pseudo-randomly from a pool of 72 stimuli, including 36 original RMET ("mind") stimuli and 36 matching control ("age") stimuli. Due to its greater statistical power, a block rather than event-related design was used (Aguirre & D'Esposito, 1999) such that stimuli were presented in blocks of 21 sec (including three mind or age trials of seven seconds each). Each run contained nine blocks of the "mind" condition and nine blocks of the "age" condition.

Trials were presented for exactly 6 sec each, followed by a crosshair '+' fixation point for 1 sec. As participants had limited time to respond to stimuli, accuracy percentages were calculated based on the total number of responses made, rather than the total number of trials, to account for trials when participants failed to respond on time.

2.6. *Image acquisition*

Functional (fMRI) and structural (T1) images were obtained with a Siemens 3-T MRI scanner, equipped with a 32-channel head coil. Functional images were acquired through gradient echo planar imaging (TR = 2.5 sec, TE = 36 msec, flip angle = 90°), in which 36 transversal slices were obtained with 64 × 64 voxels at 3 mm^2 in-plane resolution and 10% gap between slices, covering the whole brain. Each run contained 161 whole brain images, obtained every 2.5 sec. However, the first four of these were obtained prior to starting the experiment to ensure steady-state tissue magnetisation, and were not included in analyses. Additionally, T1-weighted whole brain structural images were acquired for anatomical reference (TR = 1900 msec, TE = 2.32 msec and flip angle of 9°, 192 cube matrix, voxel size = .9 cubic mm, slice thickness = .9 mm).

2.7. *Functional MRI data analysis*

All fMRI data were pre-processed and analysed using SPM8 software in Matlab (Wellcome Department of Imaging Neuroscience, Institute of Neurology, London; Mathworks Inc., USA). Pre-processing began with realignment to the first image of each run, correcting for head movements, matching images by voxels and controlling for between-run systematic differences. The T1 anatomical scan was then co-registered to the mean functional image created during realignment, and the co-registered anatomical scan was segmented. The parameters created in this step were then used to normalise the anatomical scan to the MNI T1 standard template using a voxel size of 1 × 1 × 1 mm. Additionally, these parameters were used to normalise the EPI images with a voxel size of 3 × 3 × 3 mm to map onto the MNI T1 template. Finally, the images were spatially smoothed with an 8 mm Gaussian Kernel to average data points across the brain, minimising noise across participants.

Each participant's data was then analysed using a general linear model (GLM). Specifically, BOLD signals from the onset of each block of "age" trials were subtracted from BOLD signals from each block of "mind" trials, creating a "mind" minus "age" contrast, isolating the neural activity due to the "mind" condition. A random effects GLM was then used to analyse the "mind" minus "age" contrast images. A one sample *t*-test was used to create a significance map for the random effects analysis (voxel-level threshold: $p < .05$, FWE corrected), and a voxel-level probability threshold of $p < .001$ was used to define each cluster.

2.8. *Parcel-based lesion-symptom mapping (PLSM)*

Stroke patients were recruited for PLSM analysis during the acute phase at the hospital and completed the computerised version of the RMET. Patients' CT or MRI scans and neurological reports were obtained directly from the hospital and the lesions were independently mapped by two different members of the research team. Any inconsistencies or discrepancies were discussed and decided on by an independent third party. Both researchers were blind to the behavioural deficits of the patients while undertaking the lesion reconstructions. All lesions were mapped onto 23 axial slices (MNI Z-coordinates: -58, -52, -46, -40, -34, -28, -22, -16, -10, -4, 2,

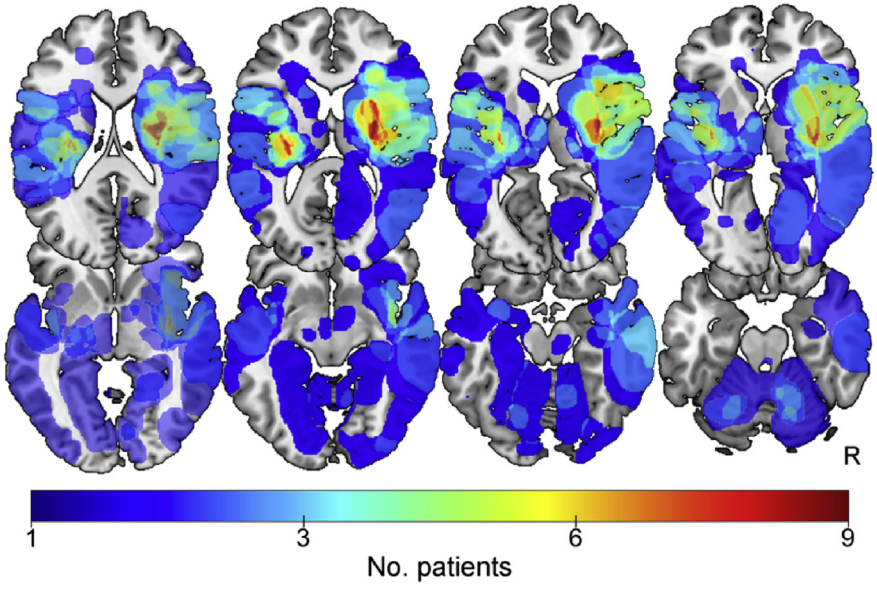

Fig. 2 – Lesion density map of all 64 stroke patients included in the final analysis. The colour code indicates the number of individuals that exhibited damage at a given voxel location (ranging from 1 to 9). Lesions are displayed on axial brain slices of the ch2bet template using MRIcroGL (http://www.mccauslandcenter.sc.edu/mricrogl). Shown, from left to right, top to bottom, slices 20, 14, 8, 2, −4, −10, −16, −22 (MNI Z-coordinates). R, right.

8, 14, 20, 26, 32, 38, 44, 50, 56, 62, 68 and 74) of the ch2bet template within MRIcron (https://www.nitrc.org/projects/mricron). Together, these slices formed a lesion map throughout the cortex, which was then saved as a VOI (volume of interest). The reliability of these mapping procedures has been verified by other laboratories using similar techniques (Borovsky, Saygin, Bates, & Dronkers, 2007; Karnath, Fruhmann Berger, Kuker, & Rorden, 2004). A lesion distribution map of all 64 patients is presented in Fig. 2.

Region of interest (ROI) PLSM analysis was then performed to determine the morphological correlates underpinning ToM processing as measured using RMET. A total of 384 grey-matter ROIs were defined based on the AICHA atlas (Joliot et al., 2015). For each region, the proportion of damage resulting from stroke together with RMET performance were entered into a general linear model. This model tested whether the proportion of damage to a given region of interest was significantly associated with impaired ToM (controlling for age). The family-wise error rate was controlled via 5000 permutations, with a threshold set at $p < .05$. The PLSM analysis was performed using NiiStat (www.nitrc.org/projects/niistat/), a set of Matlab scripts for analysing neuroimaging data from clinical populations.

2.9. *Tract-wise statistics analysis (TSA)*

White-matter correlates were first studied using the Tractotron software (part of the BCBtoolkit, http://www.brainconnectivitybehaviour.eu), which maps the lesion from each patient onto tractography reconstructions of white-matter pathways obtained from a group of healthy controls. For a given lesion, Tractotron provides a probability of disconnection for tracts from recently published white matter tract atlases (Rojkova et al., 2016). When a lesioned voxel overlaps on a white-matter tract with a probability superior to 50% (i.e., above chance), the tract is deemed to be disconnected. We then used linear regression to compare the performance on the RMET between patients with preserved and disconnected tracts (controlling for age), for tracts where at least 10 patients exhibited disconnection. To guard against violations of distributional assumptions, results are reported for bootstrapped regressions performed on the basis of 5000 permutations. Significance threshold was set at $p = .05$, corrected for multiple comparisons using the false discovery rate (FDR).

2.10. *Disconnectome symptom mapping (DSM)*

Given the variety of white matter atlases and the difficulty of atlas-based approaches to assess the disconnection of a subportion of tracts or the involvement of multiple tracts within a lesion, voxel-wise data-driven maps of disconnection or "disconnectomes" are also necessary. Disconnectomes for each participant were therefore calculated using the BCBtoolkit (Foulon et al., 2018). This approach uses diffusion weighted imaging datasets from a set of 35 healthy controls to track fibres passing through each lesion (Rojkova et al., 2016). Patients' lesions in the MNI152 space are first registered to native space in each control individual using affine and diffeomorphic deformations (Avants et al., 2011; Klein et al., 2009) and are subsequently used as seed for tractography in Trackvis (Wang, Benner, Sorensen, & Wedeen, 2007). Tractographies from the lesions are next transformed into visitation maps (Thiebaut de Schotten et al., 2011), binarised and brought to the MNI152 space using the inverse of the precedent deformations. Finally, a percentage overlap map is

generated by summing at each point in MNI space the normalized visitation map of each healthy subject. In the resulting disconnectome map, the value in each voxel incorporates the interindividual variability of tract reconstructions in controls and indicates a probability of disconnection from 0 to 100% for a given lesion (Thiebaut de Schotten et al., 2015).

We then used AnaCOM2 (available within the BCBtoolkit) in order to perform disconnectome symptom mapping proper, that is, identification of the disconnections that are associated with a deficit, in our case, ToM. AnaCOM2 is a cluster-based symptom mapping approach. Compared to standard VLSM (Bates et al., 2003), AnaCOM2 regroups voxels with the same distribution of neuropsychological scores into clusters of voxels. Additionally, AnaCOM2 performs comparisons between patients and controls as a first step in order to avoid drastic reduction of statistical power when two or more non-overlapping areas are responsible for patients' reduced performance (Kinkingnéhun et al., 2007). AnaCOM2 resulted in a statistical map revealing for each cluster the significance of a deficit on patients' performance in RMET, compared to controls. *p*-values were Bonferroni-Holm corrected for multiple comparisons at $p < .05$.

3. Results

3.1. RMET accuracy

A score summary map for the control and stroke group is presented in Fig. 3. Healthy participants' RMET scores ranged from 24 to 35 (66.67%–97.22%; $M = 78.05\%$, $SD = 9.62\%$), and were all classified in the normal range, based on Baron-Cohen et al.'s (2001) criteria (>58.33%). In contrast, stroke patients' scores ranged from 12 to 35 (33.33%–94.44%; $M = 61.50\%$, $SD = 13.90\%$), of which 34.38% of patients fell within the abnormal range. There was a significant group effect, $t(46) = 5.99$, $p < .001$, with stroke patients scoring lower than healthy controls.

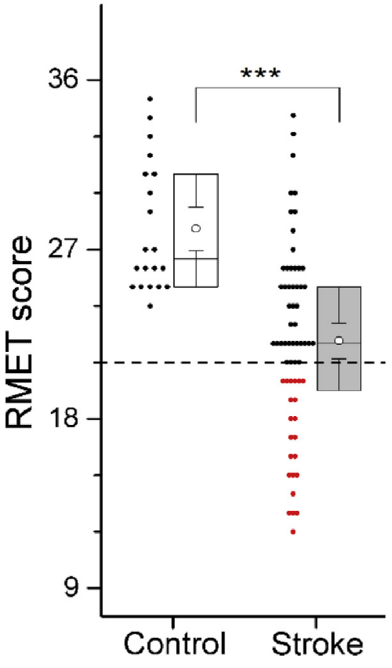

Fig. 3 – RMET score distribution for both the control group, and stroke patients. Red scores below the horizontal dash line represent stroke patients with impaired RMET performance by Baron-Cohen et al.'s (2001) criterion (<58.33%). Stroke patients performed significantly worse than healthy controls, $t(46) = 5.99$, $p < .001$.

3.2. fMRI results

Healthy participants exhibited significantly greater activation in the "mind" compared with the "age" condition in the left pIFG, insula, superior and medial temporal gyrus (STG/MTG), temporal pole, rIPL, pSTS, and adjacent TPJ (−51, 17, 13; $Z = 6.82$, $k = 2196$, $p < .001$), the right pIFG, insula, STG/MTG, temporal pole, rIPL, pSTS, and adjacent TPJ (63, −40, 4; $Z = 5.36$, $k = 1083$, $p < .001$), as well as the dmPFC (−3, 14, 67; $Z = 4.86$, $k = 356$, $p = .015$), left medial frontal gyrus (MFG; −42, −1, 55; $Z = 5.72$, $k = 211$, $p < .001$) and medial occipital cortex (MOC; -18 -94 28; $Z = 4.03$, $k = 377$, $p < .001$). See Fig. 4.

3.3. PLSM results

PLSM revealed that areas significantly associated with lower RMET performance comprised mainly the right inferior frontal gyrus (pars opercularis), insula, precentral gyrus and caudate tail. See Fig. 5.

3.4. TSA results

We found poorer performance in RMET was associated with a greater probability of disconnection in the right frontal aslant tract, FAT (FDR corrected $p < .05$). Such an association was also observed in the fornix, anterior commissure and right inferior longitudinal fasciculus (ILF). While in these tracts this association did not survive correction for multiple comparisons, it was close to significance (*i.e.*, *P*-values were close to the corrected critical *P*-value = < .002: fornix, $p = .003$; anterior commissure, $p = .004$; and right ILF, $p = .005$). See Fig. 6.

3.5. DSM results

In order to visualize the link between RMET scores and the pattern of disconnection, RMET scores were mapped onto each patient's disconnectome and averaged across participants (Fig. 7). Inspection of the resulting map showed impaired RMET performance (RMET scores below 21) is associated with right lateralized disconnection of tracts largely overlapping with the tracts identified by TSA. Results from AnaCOM2 revealed clusters exhibiting disconnection that led to a significant decrease in RMET performance in patients when compared to controls (Bonferroni-Holm corrected at $p < .05$). Clusters with the smallest *P*-values ($p < .01$) coincided with TSA results as they were found within the right FAT, as well as the fornix, anterior commissure and right ILF. Highly significant clusters were also observed in the body of the corpus callosum and the right external capsule. Less significant clusters were also found in the splenium of the corpus

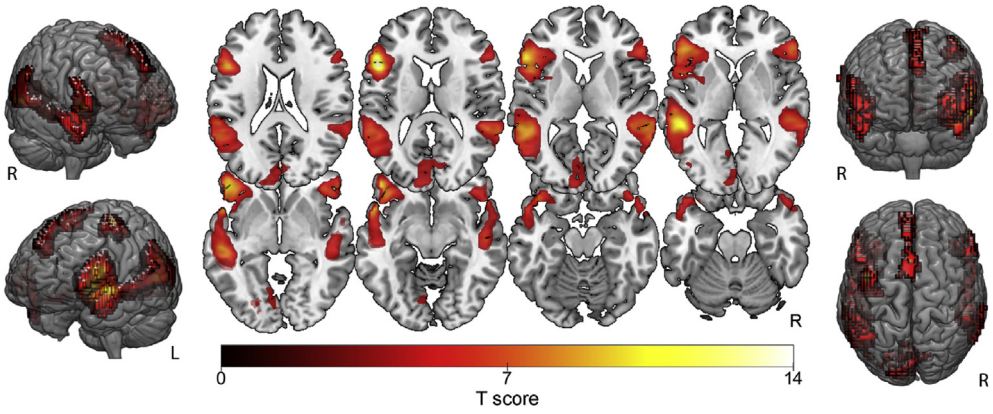

Fig. 4 – Significant clusters showing increased BOLD response (cluster corrected at $p < .05$) during the "mind" RMET versus the "age" control task for the twenty healthy older participants. Activations are displayed on axial brain slices of the ch2bet template using MRIcroGL. Shown, from left to right, top to bottom, slices 20, 14, 8, 2, −4, −10, −16, −22 (MNI Z-coordinates). Sides: fMRI results rendered in 3D. R, right; L, left.

callosum, arcuate fasciculus, bilaterally, and along the internal capsule in the left hemisphere (Fig. 7).

4. Discussion

In the present study, a multi-method approach was used to gain a clearer and more nuanced understanding of the relationship between ToM function and underlying brain damage in a sample of stroke patients. To operationalise ToM, the RMET was used, an extensively validated measure of this construct that involves interpreting the thoughts and feelings of others through visual eye gaze cues. The eyes provide vital information to assist us in deciphering the mental states of others, and to interpret other people's behaviour causally (Emery, 2000). By integrating RMET data across fMRI, PLSM, TSA and DSM analyses, the results provide important and novel insights into both the functional areas responsible for intact performance, as well as the location of brain lesions that are linked to impairment.

Four main findings emerged. First, as expected, stroke patients performed significantly worse (one-third scored in the abnormal range) than control subjects (no abnormal scores) on the RMET. This is in line with previous research that has shown extensive ToM deficits and social cognitive and communication impairments in patients with cerebral infarcts (Balaban et al., 2016; Happé et al., 1999; Muller et al., 2010; Shamay-Tsoory & Aharon-Peretz, 2007; Xi et al., 2013; Yeh & Tsai, 2014). The current data therefore further highlight the need to systematically assess ToM function in patients with brain damage, given the potentially profound impact of these social cognitive deficits on patients' functional status and prospects for recovery (Henry et al., 2016; Martín-Rodríguez & León-Carrión, 2010).

Second, consistent with recent evidence, healthy participants showed greater BOLD activation in both the ST (pIFG, rIPL, pSTS and insula) and TT (dmPFC, TPJ and TP) networks during the RMET (Molenberghs et al., 2016; Schurz et al., 2014). These data suggest that eye-based attribution of thoughts and emotions involves a combination of automatic simulation of

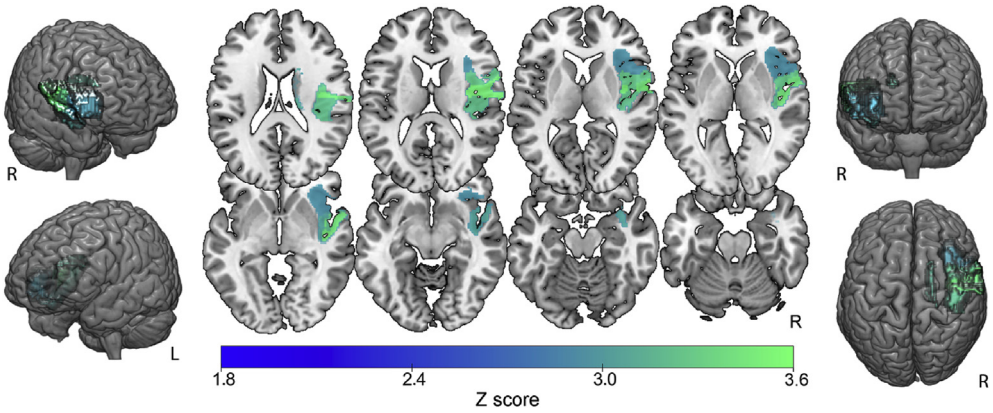

Fig. 5 – Results of PLSM analysis of 64 patients displayed on the ch2bet template in MRIcroGL. Areas marked represent voxels that led to significantly poorer scores on RMET when lesioned (FWE corrected $p < .05$). Shown, from left to right, top to bottom, slices 20, 14, 8, 2, −4, −10, −16, −22 (MNI Z-coordinates). Sides: results of PLSM analysis rendered in 3D. R, right; L, left.

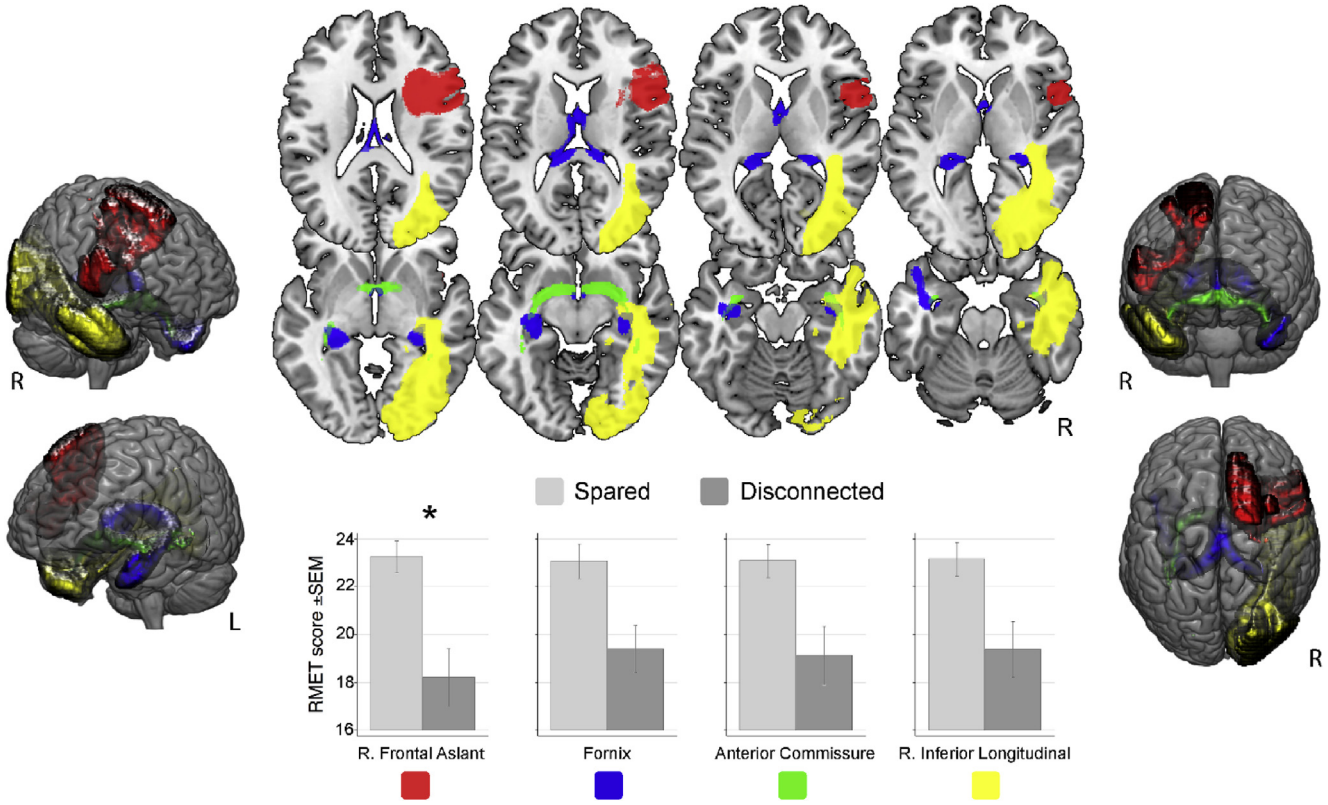

Fig. 6 – Results of track-wise statistical analysis. Poorer performance on the RMET was significantly associated with a greater probability of disconnection in the FAT, FDR corrected $p < .05$, *. Such an association was also observed in the fornix, anterior commissure and right ILF. While in these tracts this association did not survive correction, it was close to significance. Top: the tracts are depicted on axial slices of the ch2bet template in MRIcroGL. Shown, from left to right, top to bottom, slices 20, 14, 8, 2, −4, −10, −16, −22 (MNI Z-coordinates). Sides: the tracts are rendered in 3D. Bottom: RMET performance in patients with a disconnection versus patients without a disconnection in the relevant tracts. R, right; L, left.

others' likely mental processes (Molenberghs et al., 2012; Rizzolatti & Sinigaglia, 2010; Völlm et al., 2006), as well as deliberate cognitive reasoning about these mental states (Saxe, 2005).

Third, PLSM results showed that lesions in the right inferior frontal gyrus (pars opercularis), insula, precentral gyrus and caudate tail were associated with poorer performance on the RMET. This is in line with previous studies which have shown associations between lesions in similar brain regions with poorer ToM performance. For example, using a whole-brain VLSM approach, Dal Monte et al. (2014) showed that impaired RMET performance was associated with IFG damage in a sample of traumatic brain injured patients. Herbet, Lafargue, Bonnetblanc, Moritz-Gasser, and Duffau (2013) also found that lesions to the pars opercularis in the IFG specifically impaired RMET performance in a sample of patients with a slow growing brain tumour. Furthermore, Campanella, Shallice, Ius, Fabbro, and Skrap (2014) conducted a lobe-based and voxel-based analysis on a group of patients with brain tumours and showed that lesions in the insula, TPJ, MTG, anterior temporal lobe, as well as the superior temporal gyrus were all associated with poorer performance on the RMET.

The right hemisphere laterality found in the PLSM analysis is also consistent with previous lesion studies which have shown that damage to the right hemisphere is more strongly related to impaired ToM performance (Balaban et al., 2016; Happé et al., 1999; Shamay-Tsoory, Tomer, Berger, Goldsher, & Aharon-Peretz, 2004; Siegal, Carrington, & Radel, 1996; Stuss, Gallup, & Alexander, 2001; Weed, McGregor, Feldbaek Nielsen, Roepstorff, & Frith, 2010). However, a caveat in the present study was the fact that lesions in the right hemisphere were more densely sampled (Fig. 2), mainly because patients with language impairments (which is often associated with left hemisphere damage), were excluded. This lack of whole brain representation was compounded by another inherent problem when working with stroke patients: lesions are not uniformly dispersed throughout the brain, but tend to occur in the vicinity of major arteries. As a consequence, other cerebral regions such as the dmPFC for example which are often implicated in ToM abilities could not be identified because they were not part of the lesion distribution map.

Fourth, using TSA and DSM (Thiebaut de Schotten et al., 2011), we were able to analyse the effect of lesioned white-matter on ToM performance. These results revealed white-matter damage that negatively impacted RMET scores was also right lateralized. Both TSA and DSM implicated the right FAT, fornix, anterior commissure and right ILF in ToM

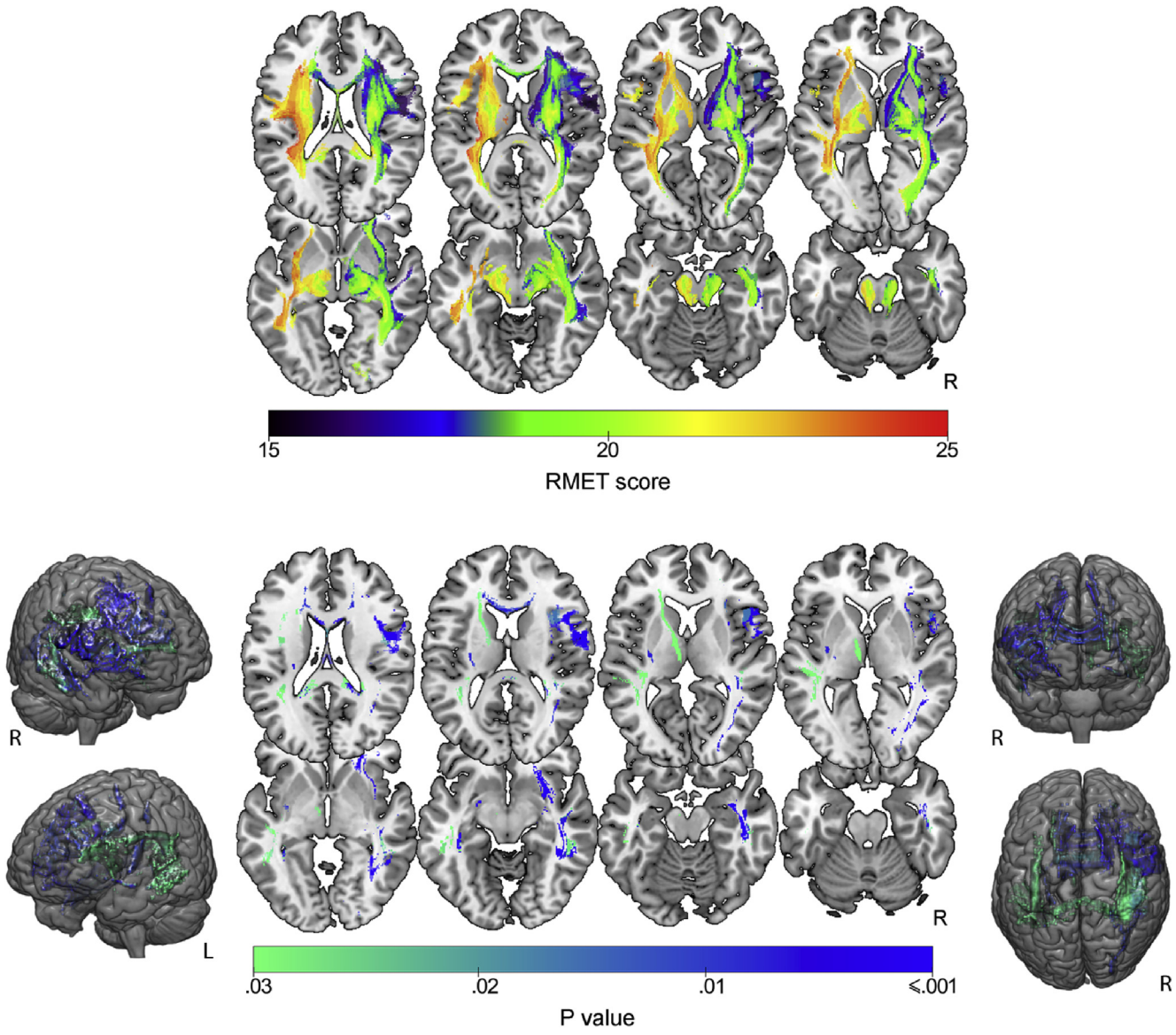

Fig. 7 – Results of disconnectome symptom mapping. Top: Representative slices from disconnectome maps computed for RMET score: blue indicates group average low performance and red clusters indicate higher performance. Bottom: Clusters exhibiting disconnection that led to a significant decrease in RMET performance in patients when compared to controls (Bonferroni-Holm corrected at $p < .05$). Results are displayed on the ch2bet template in MRIcroGL. Shown, from left to right, top to bottom, slices 20, 14, 8, 2, −4, −10, −16, −22 (MNI Z-coordinates). Sides: results of DSM analysis rendered in 3D. R, right; L, left.

dysfunction. The FAT is a recently discovered frontal intralobar white-matter tract that connects the pIFG with the cingulate gyrus and other medial frontal regions (Catani et al., 2013). The present finding, that lesions of the FAT disrupt ToM function, aligns with previous suggestions that the FAT extends and connects the pIFG to more anterior areas of the dmPFC that are consistently involved in ToM processing (Catani & Bambini, 2014). The pIFG belongs to the ST network and dmPFC to the TT network, so the FAT might link the two networks together. This suggests that damage to the ST network may lead to functional changes in the TT network, and vice versa.

The fornix is an interhemispheric area of white-matter between the hypothalamus and the hippocampus that forms part of the limbic system. A relationship between damage to this particular region and social cognitive function has recently been highlighted. Using the Awareness of Social Inference Test (TASIT), Downey et al. (2015) analysed white-matter tract signatures of impaired social cognition in a cohort of patients with either behavioural variant frontotemporal dementia (bvFTD) or semantic variant primary progressive aphasia (svPPA). The results indicated that both the bvFTD and the svPPA groups showed severe deficits in basic emotion recognition and more complex social inference

(identification of sarcasm). The emotion recognition deficits in particular were correlated with degeneration of the fornix, which links cognitive and evaluative processing with emotional responses. Other researchers have similarly argued that ToM reasoning is supported by a widely distributed neural system which is centred on the limbic system (Abu-Akel, 2003; Rajmohan & Mohandas, 2007), and the amygdala in particular (Siegal & Varley, 2002). Immediately anterior to the fornix is the anterior commissure, a compact fibre bundle that, like the fornix, interconnects several areas of the two hemispheres. Among other areas, the anterior commissure projects to the amygdala, the temporal lobe (including the temporal pole, TP) and the orbitofrontal cortex. The anterior commissure has been implicated in several functions including allocation of attention, memory and emotion (Patel, Toussaint, Charles-Edwards, Lin, & Batchelor, 2010; van Meer et al., 2016).

The ILF is a major occipitotemporal association tract. It connects occipital and occipitotemporal areas to the anterior temporal lobe and the amygdala. The ILF, particularly in the right hemisphere, plays a critical role in face recognition, conveying information between components of the core and extended face networks. Importantly, a number of studies have implicated the ILF in the recognition of facial emotions (Herbet, Zemmoura, & Duffau, 2018). ILF abnormalities have been reported in schizophrenia and 22q11.2 deletion syndrome (a genetic neurodevelopmental disorder with physical, neuropsychological, and psychiatric effects). It has been suggested that ILF dysfunction may be involved in the social cognition impairments reported for both of these disorders, which critically include, in the case of 22q11.2 deletion syndrome, theory of mind and empathy (Herbet et al., 2018). In addition to the tracts described above, DSM also revealed RMET performance was associated with disconnection in the body and splenium of the corpus callosum, and the arcuate fasciculus, bilaterally. Affected fibre bundles in the body of the corpus callosum connect respective areas of the right and left frontal lobes, while fibres travelling through the splenium connect right and left temporal lobes (Hofer & Frahm, 2006; Huang et al., 2005). The arcuate fasciculus, in turn, is a bundle of white-matter fibers that forms part of the superior longitudinal fasciculus (SLF), and connects the temporal cortex and inferior parietal cortex to locations in the frontal lobe (Catani & Thiebaut de Schotten, 2008).

In the present study, we also identified a role for the temporal poles (TPs; see Fig. 4) in ToM performance. A number of previous studies have also reported activation in the TPs in subjects engaged in mental state reasoning (Farrow et al., 2001; Völlm et al., 2006). Such findings have led a number of researchers to conclude that the TPs are a critical part for intact ToM ability and are a necessary component of the mentalising brain (Frith & Frith, 2006; Irish, Hodges, & Piguet, 2014; Mar, 2011). It has been argued that the TPs play an important role in social cognition by providing access to social knowledge in the form of scripts (Frith & Frith, 2006; Gallagher & Frith, 2003; Olson, Plotzker, & Ezzyat, 2007). In addition to the anterior commissure, the white-matter tracts that comprise the fornix have extensive connections into the TPs. Also, the fornix, through the hippocampal commissure, connects the two cerebral hemispheres across the midline (Thomas, Koumellis, & Dineen, 2011). This, together with RMET-related disruption of the body and splenium of the corpus callosum, helps illuminate how damage to one side of the brain can lead to contralateral functional changes and cognitive deficits, and explains the relationship between functional and lesion studies, even if dissimilar neuroanatomical regions are implicated.

Overall, the evidence presented here suggests that damage to white-matter pathways is an important predictor of poor ToM performance. The TSA and DSM results indicate that, relative to healthy individuals, abnormal scoring stroke patients have decreased white-matter integrity between their lesion site and the areas showing functional activation (see Fig. 8). This may help explain why, if damage (in blue in Fig. 8, top) is restricted to (and overlaps with) only a small portion of the ToM network (apparent in our fMRI results; in red in Fig. 8, top), we still see impaired RMET performance. Impairment seems therefore to be not only caused by direct damage to cortical nodes of the ToM network but also by damage to connections between the nodes (depicted in the bottom panel of Fig. 8). The present results therefore suggest that acquired ToM dysfunction can be usefully explored in future research as a disconnection syndrome. By this view, deficits could be caused by damage to underlying white-matter axons connecting core mentalising regions of the cortex, irrespective of, or supplementary to lesion location (Bartolomeo et al., 2007; Das, Calhoun, & Malhi, 2012; Philippi, Mehta, Grabowski, Adolphs, & Rudrauf, 2009; Wang & Olson, 2018).

To date, the disconnection syndrome hypothesis has gained some support. For example, Herbet et al. (2014) analysed the degree of disconnection in the underlying white-matter pathways in a sample of patients with diffuse low-grade gliomas (DLGG; a rare type of brain tumor that migrates along subcortical white-matter connections). The results showed that RMET deficits in this group were mainly associated with damage to fronto-temporal white-matter connectivity. Specifically, mentalising ability, as operationalised by performance on the RMET, was significantly correlated with the degree of disconnection in the arcuate fasciculus. In a separate study, Philippi et al. (2009) showed that damage to association white-matter tracts disrupts facial affect recognition in patients with stable focal lesions. In particular, damage to the right inferior fronto-occipital fasciculus (IFOF) was associated with impairment in this domain. Consistent with Herbet et al. (2014), our results also implicated the arcuate fasciculus; however, in the present study we also provided evidence for a more widespread pattern of white-matter disruption linked to RMET performance. One possible reason for this discrepancy may be the limited locations of the lesions in the Philippi et al. (2009) study (given that DLGGs migrate preferentially along associative white-matter pathways). As an illustration, no patients had posterior lesions, which limited the spatial scope of their analysis. It is possible that a larger sample of patients with lesions more representative of the whole brain would lead to more converging findings between these two different sets of analyses.

The current results need to be interpreted in the context of several possible limitations. First, some authors have suggested that in the acute phase of a syndrome, mechanisms such as hypometabolism and diaschisis can cause profound

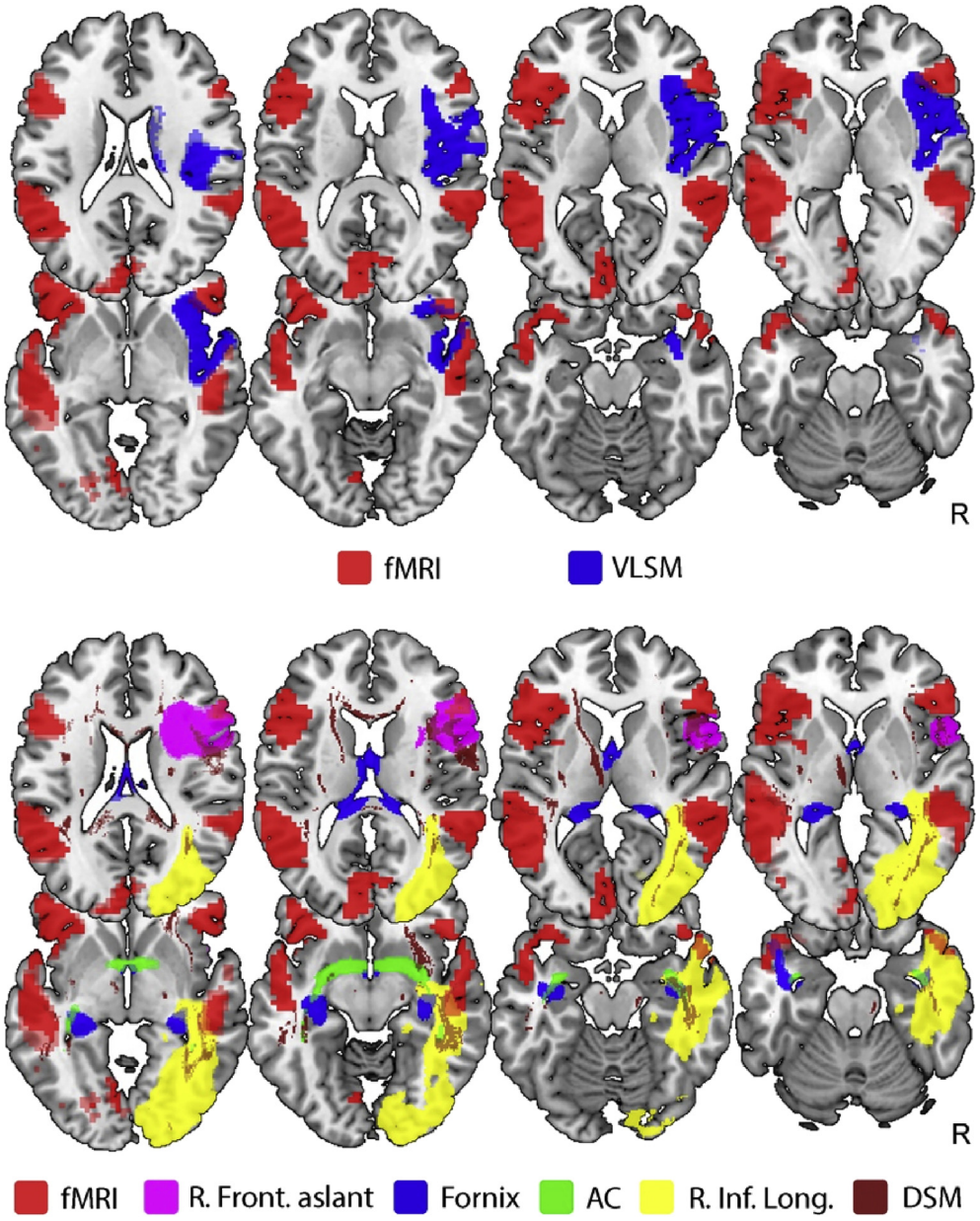

Fig. 8 – Schematic representation of fMRI together with PLSM (top), and TSA and DSM (bottom) results overlayed on the MRIcron ch2bet template. As can be clearly seen, both the right FAT (which connects the lesion sites associated with poor ToM performance to the "core" ToM region in the dmPFC), the fornix and anterior commissure, AC (which connects the two cerebral hemispheres and hence the functional regions in both sides of the brain) and the right ILF (which connects occipital with temporal regions), help explain why specific lesion sites associated with ToM disability only partially overlapped with the functional network observed during the fMRI experiment. Results are displayed on the ch2bet template in MRIcroGL. Shown, from left to right, top to bottom, slices 20, 14, 8, 2, −4, −10, −16, −22 (MNI Z-coordinates). R, right.

neurocognitive dysfunction that affects large brain areas (Thiebaut de Schotten et al., 2014). Such dysfunction, it is argued, often spontaneously alleviates when tested a short period later. This is unlikely to be a significant issue in the present investigation because previous studies have demonstrated that ToM deficits do not readily dissipate after stroke and often persist many months (and years) post-onset (Balaban et al., 2016; Happé et al., 1999; Yeh & Tsai, 2014). Second, as noted in the introduction, LSM analyses are inherently limited by the variability of patients' lesion sites, such that a region cannot be identified as a critical site if none of the included patients have lesions to that area. Lesion sites in stroke patients are not uniformly distributed throughout the brain and are highly dependent upon vascular territory (Kimberg et al, 2007; Rorden et al., 2007). As a result, it is possible that other regions critical for ToM ability were not identified in our PLSM and TSA/DSM analyses. Third, while we found widespread damage to white-matter tracts involved in RMET performance, we cannot conclude that these are the only pathways potentially compromised. Damage to the IFOF,

for example, showed significant effects on the recognition of facial emotions (Philippi et al., 2009). In the same study, Philippi et al. (2009) reported similar results in inferior and superior longitudinal tracts from simple linear regressions (SLR) when no other tracts were used as covariates and suggested that shared variance between tract regressors, coupled with issues with scant sampling, makes it very possible that the absence of effects for a number of tracts in their multiple regression analysis could be false negatives. In contrast, it is possible that using the full set of spatial coordinates for each tract in isolated analysis may exaggerate its contributions (Philippi et al., 2009).

As noted previously, we chose the RMET because it imposes minimal demands on other cognitive processes such as language and executive functioning. However, considering the findings reported in this study, it is also important to acknowledge that processes not specific to ToM, such as impaired face or visuo-spatial processing may have affected stroke patients' performance on the RMET. We think impaired face processing is unlikely because the fusiform gyrus, the most common region associated with face processing in neuroimaging studies (Kanwisher & Yovel, 2006), was for the most part spared in our sample (with a lesion overlap of 1 in neighbouring areas; see Fig. 2, slice −16). However, future studies investigating ToM with the RMET, should include a face recognition test to more definitively exclude this possibility. Regarding visuo-spatial processing, it is important to note that Toba et al. (2017) tested stroke patients in a set of visuo-spatial attention tasks and found similar brain areas as those critical for the present study (IFG and TPJ) being responsible for the poorer performance on these kinds of tasks. The Bells task we administered to test for neglect is more broadly a tool for visuo-spatial perceptual deficits, so we think it is unlikely that patients in our study were clinically impaired in this regard (patients evincing neglect were excluded).

Regardless of these potential limitations, the present study provides important new evidence into the neural basis of ToM, and eye-based mentalising in particular, offering converging evidence of two integrated sub-networks of mentalising abilities (Herbet et al., 2014; Keysers & Gazzola, 2007). The results also have immediate and direct practical implications. First, they show that patients with damage to the regions identified through PLSM can be regarded as most likely to experience deficits in their ability to read complex emotions in others' faces, facilitating early diagnosis and treatment. The fact that over a third of the stroke patients in our sample showed an abnormal score on the RMET indicates that ToM impairment is a major problem in this group, and consequently, early intervention of this type is critical. Second, the tract-wise analysis suggests that post-stroke ToM impairments can sometimes be attributable to damaged white-matter pathways, as opposed to direct damage to the ToM network itself. In future research, it will be useful to apply this approach to larger samples of patients to determine whether the functional changes involved in ToM impairment are consistent across patients, and whether there are other white-matter tracts in which integrity is predictive of ToM ability (e.g., Herbet et al., 2014). In addition, it is important to also repeat this technique with other validated measures of ToM processing, so that our understanding of the types of lesions that underlie ToM impairment can be generalised more broadly. This approach would also determine whether certain types of lesions are associated with impairments to different facets of ToM ability.

In conclusion, the current study provides evidence that regions of both ST and TT networks are involved in understanding other people's facial expressions and that damage to these networks, either directly or through disconnection of white-matter pathways that connect these areas, are key determinants of impairments in this critical function.

Open practices

The study in this article earned Open Materials and Open Data badges for transparent practices. Materials and data for the study are available at https://osf.io/ys8ct/wiki/home/.

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