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Brain networks underlying inhibition in Traumatic Brain Injury and in Autism Spectrum Disorders

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BRAIN NETWORKS UNDERLYING INHIBITION IN TRAUMATIC BRAIN INJURY AND IN AUTISM SPECTRUM DISORDERS

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ABSTRACT

Autism Spectrum Disorder (ASD) and Traumatic Brain Injury (TBI) are clinical populations with social cognition difficulties, exhibited by deficits in controlling impulsive or perseverative behaviors. These difficulties have been attributed to executive functioning (EF) impairments, particularly for inhibition. Thus, understanding the neural bases of inhibition is preliminary to understanding EF impairments in populations like ASD and TBI. A coordinate-based meta-analysis of functional magnetic resonance imaging (fMRI) studies was used to identify the neural basis of response inhibition in neurotypical adults to compare with TBI and ASD. Inclusion criteria for studies required reported foci for adults (17+ years of age), reported on normal mapping, and used inhibition experiential tasks that revealed activations results. Five ASD and seven TBI studies met inclusion criteria, pooling fMRI data from 1431 neurotypical subjects, 145 TBI and 71 ASD subjects engaged in inhibition tasks, yielding 98 experiments in controls and 15 experiments (9 TBI) for contrast analyses. Brain regions found to be uniquely active in the ASD or TBI and in the Control groups were further analyzed using meta-analytic connectivity modeling (MACM) to determine whether differences in these regions were functionally relevant and associated with differing behavioral patterns. The MACM analyses included 480 neurotypical experiments (6820 subjects, 7008 foci) reporting activity in the left medial frontal gyrus region of interest and 809 experiments (11568 subjects, 11855 foci) reporting activity in the right medial frontal gyrus region of interest. Results provide evidence that the brain region involved to the greatest extent for response inhibition, the medial frontal cortex, is active in individuals with TBI, with ASD and Controls. However, the groups had differences in the peaks of activity in this region. Though subtle, these differences may indicate these clinical populations are relying more on top-down, higher-level cognitive processing to accomplish response inhibition than do neurotypical controls. Results support a hypothesis that those with ASD or TBI are engaging a smaller network of brain regions, with a higher proportion of activity in the frontal lobes, and therefore less efficient than that seen in the Controls. Given the heterogeneity of TBI and ASD demographics, and the variability of inhibition tasks used, these findings are speculative and require further study. This study provides support of concept for further research on functional imaging, attention, and inhibition.

INTRODUCTION

Executive functions (EF) are complex higher cognitive processes that are critical for directing and modulating thoughts and behaviors, involving widespread cortical and subcortical brain networks (Derrfuss et al., 2005; D. E. Nee et al., 2007). Adequate executive functions effectively manages performance in cognitive, behavioral, and social realms by adapting thoughts and actions to maintain novel or routine goal-directed tasks (Collette et al., 2005; Riggs et al., 2006). Tasks requiring executive functions can fall under personal, social, academic or professional routines, making EFs central to independence, productivity, and quality of life (Riggs et al., 2006).

Theorists divide executive functions into those that are domain-specific or domain-general divisions. Domain-specific processing, which may be considered "bottom-up" processing, is thought to be modulated by neural areas that process discrete sensory inputs (e.g., the visual cortex processes visual information) that then direct information to higher-level cortical areas (e.g. the prefrontal lobe)(A. Gazzaley et al., 2007). Domain-general, or top-down, processing maintains the integration of information across diverse mental processes (like the domain-specific sites) (Adam Gazzaley & D'Esposito, 2007; Adam Gazzaley & Nobre, 2012). This concept of top-down modulation, housed in the prefrontal cortex, is attributed to the idea of 'cognitive control' (Adam Gazzaley & D'Esposito, 2007), which refers to the successful integration of information from multiple brain regions via capabilities like attention, shifting, and inhibiting, to direct neural behavior (Adam Gazzaley & D'Esposito, 2007).

The collective ability to modulate information from domain-specific neural regions using various EFs is known by the synonymous terms 'executive function,' 'the common executive,' 'central executive,' or 'cognitive control' (Lenartowicz et al., 2010; MacDonald, 2008;

McKenna et al., 2017; Miyake & Friedman, 2012; Derek Evan Nee et al., 2013; Niendam et al., 2012). This term 'executive function,' or its synonyms, encompasses several sub-abilities of neural processing that have been discussed in the literature that include inhibition, shifting, updating, planning, flexibility, problem-solving, attentional abilities, working memory, planning, organizing, reasoning, categorization, etc. (Jurado & Rosselli, 2007). While it was first conceptualized in the 1970s, theories regarding the nature and role of executive functions continue to be developed(Baddeley & Hitch, 2004; Lezak, M.D, 1983; Miyake et al., 2000; Norman & Shallice, 1986; and many others).

In the 1970s, Alan Baddeley and his colleague, Graham Hitch, theorized a model of working memory, which is the ability to retrieve information from long-term and short-term memory stores to update (or refresh) that information. Working memory is a key postulated component in models of higher-cognitive functions. Baddeley and Hitch's (2002) working memory model was the first to introduce the 'central executive' (A. Baddeley, 1996, 2002; A. D. Baddeley & Logie, 1999). The 'central executive' was proposed as the principal component in a three-component model that integrates information held in the sub-component short-term memory buffers – i.e., 'phonological loop' and 'visuospatial sketchpad' – and links that information to long-term memory. These buffers are proposed temporary storage banks for verbal and visual information (received from domain-specific areas), that are needed to direct decisions in novel situations. Baddeley and Hitch (2002) initially defined the 'central executive' vaguely, as a component able to focus, switch, and divide attention. However, through continued case studies and the foundational work of other attentional control theorists like Norman and Shallice (1986), Baddeley realized a need to disassociate and deconstruct the aspects of the 'central executive' (A. Baddeley, 1996). Since Baddeley's initial work, the need to parse out and

define the sub-components of the 'central executive' have led to the continued development of EF frameworks and discussions about specific EFs.

A current framework of EF is the 'unity/diversity framework' (Jurado & Rosselli, 2007; Miyake et al., 2000; Miyake & Friedman, 2012). It states that while the unity of several EFs contribute to the overall ability of the common executive, the EF's performance and neural sites of activation can be distinguished from one another (Duncan et al., 1996; Miyake et al., 2000; Miyake & Friedman, 2012). That is, Miyake and colleagues (2000) hierarchically clustered three executive functions: inhibition, shifting, and updating (Miyake et al., 2000; Miyake & Friedman, 2012). Miyake et al. (2000) believed these three EFs to be foundational (i.e. necessary to perform other EFs, like planning). Inhibition, is the deliberate suppression of internal and automatic (response inhibition) or external (distractor inhibition) information that is not appropriate or necessary at a given time (Jurado & Rosselli, 2007; Miyake et al., 2000; Nee et al., 2013). Response inhibition is the focus of this research paper. Updating is the monitoring and maintenance of stimuli in memory, while also adding newer information to build upon previous stimuli (classic operation of working memory, Linden, 2007). Shifting is required to flexibly perform a task that requires alternating between different incoming stimuli or response demands (Collette et al., 2006; Derrfuss et al., 2005). Miyake et al. (2000) established this trichotomy by performing latent variable analysis on the behavioral performance of several EF-specific tasks in neurotypical individuals (e.g., the Flanker task for inhibition, the n-back test for updating, or the Color-Shape task for shifting). They found that correlations amongst specific executive functions demonstrated separability as well as an underlying unity consistent with a 'common executive,' that shared variance across all three (Miyake et al., 2000; Miyake & Friedman, 2012). Interestingly, while updating and shifting are separable from the common executive and

inhibition (i.e. showed variance in correlation), the correlation between the common executive and *inhibition* was nearly perfect (McKenna et al., 2017; Miyake & Friedman, 2012). These authors reasoned, then, that inhibition, being synonymous with the common executive, may be a more foundational to overall executive function than updating or shifting.

Further support for inhibition as a foundational EF comes from a developmental perspective as its underlying neural structure has been identified in early childhood (Garon et al., 2008; McKenna et al., 2017; D. E. Nee et al., 2007; Verbruggen & Logan, 2008). For example, Mckenna and colleagues (2017) report, using functional magnetic resonance imaging (fMRI) with children aged 6-18, report that neural activation for the common executive (or inhibition) is seen throughout the age span, but updating-specific and shifting-specific activation did not occur until age 12 years. This continues to suggest that, conceptually, inhibition is more foundational than other executive functions.

Neurophysiologically, executive functions engage large brain networks, involving several brain structures that are associated with organizing information for complex tasks. Brain networks are sets of brain regions that demonstrate coherent neural activity, both at rest and during tasks. The networks typically associated with EFs, include the (1) frontoparietal and (2) cingulo-opercular networks (Collette et al., 2006; McKenna et al., 2017; Nee et al., 2013; Niendam et al., 2012). Specific to inhibition in neurotypical individuals, as reported in a meta-analysis of over 100 inhibition experiments, regional activation is observed in the dorsolateral prefrontal cortex (DLPFC), the anterior cingulate cortex (ACC), the superior and inferior portions of the parietal lobes, and in subcortical structures (the thalamus, putamen, caudate, and cerebellar declive) (Niendam et al., 2012). Despite having broad atypical presentations, EF

performance on isolated tasks may be unimpaired (Chen et al., 2016; Hellyer et al., 2015; Johnston et al., 2019; Lopez et al., 2005; Rochat et al., 2013; Sharp et al., 2014; Wu et al., 2014).

Given the behavioral and neurophysiologic complexity of executive functions behaviorally and physiologically, it is not surprising that several clinical populations experience EF dysfunction when neurodevelopment is altered or the brain is damaged (Rasmussen et al., 2006). From a behavioral perspective, even neurotypical individuals demonstrate fluctuations in EFs as they develop and decline with advanced age (e.g., when completing a stop-signal paradigm, Williams et al., 1999). Behaviorally, EF dysfunction results in trouble staying on task, organizing and prioritizing daily activities, navigating the complexities of social situations, transitioning through known and unknown life situations, and building on previous knowledge to solve problems (Järbrink & Knapp, 2001; McCauley et al., 2013; Turkstra et al., 2001; Tyerman, 2012). For example, Autism Spectrum Disorder (ASD) and Traumatic Brain Injury (TBI) are two clinical populations with known executive functioning impairments that share remarkable similarities in the outward presentations of difficulties navigating social situations, relationships, and task organization (Alves et al., 1993; Hanten et al., 2011; Poon & Sidhu, 2017; Steel & Togher, 2019).

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder characterized by impairments in social communication and the presence of restricted or repetitive behaviors (American Psychiatric Association, 2013). It can be diagnosed as early as 18 months of age and has a current U.S. incidence of 1 in 59, with comparable global rates (CDC, 2019). Although ASD is often defined by primary impairments in understanding other's perspectives and initiating and maintaining friendships, there are also deficits in executive functioning. Insistency on routines, difficulties shifting between tasks or interests, and controlling impulsive comments

in conversations are behavioral examples of inhibition dysfunction in ASD (Craig et al., 2016; Geurts et al., 2014; Johnston et al., 2019; Lopez et al., 2005; Poon & Sidhu, 2017; Velasquez et al., 2017).

Neuroimaging findings have indicated that deficits in inhibition, working memory, and shifting in ASD are associated with hypoactivation in the dorsolateral prefrontal cortex and the anterior cingulate cortex (Geurts et al., 2014; Philip et al., 2012). Specifically, during inhibition-specific tasks like the Go/No-Go and the Flanker tasks, there is reduced activity in the parietal, insular, dorsolateral prefrontal, and the anterior cingulate cortices, regions that are typically engaged in attention-demanding tasks (Corbetta & Shulman, 2002; Dichter & Belger, 2007; Kana et al., 2007; Shafritz et al., 2015). However, with similar inhibition tasks in ASD, Schmitz et al. (2006) found hyperactivation activation in these areas (Schmitz et al., 2006). Therefore, whether ASD can be characterized by reduced or increased regional activity for inhibition, relative to age-matched controls during EF performance, is still unclear (Geurts et al., 2014).

Traumatic Brain Injury (TBI) is an umbrella term for acquired neurological injury that can be characterized by severity of trauma (mild to severe), the location of the trauma (focal or diffuse damage), and the nature of injury (open or closed head injury). In 2015, it was estimated between 3.2 and 5.3 million people were living with a TBI in the United States (CDC, 2015). Individuals with TBI face a wide range of challenges, with psychiatric, cognitive, biological, and behavioral realms impacted. The recovery from these challenges are influenced by a multitude of factors, including, age, quality of rehabilitation services, and site/severity of neural damage (Cremer et al., 2006; Zhou & Lui, 2013). EF dysfunction in TBI may be seen as a memory deficit, reduced error awareness, and emotional changes (Dikmen et al., 2010; McAvinue et al., 2005; Stuss et al., 1992). Inhibition dysfunction in individuals with TBI is often demonstrated by

impulsivity, distractibility, or the presence of perseverative comments and behaviors (Hellyer et al., 2015; Mayer et al., 2015; Rochat et al., 2013). For individuals with TBI, poorer EF is associated with lower rates of returning to work or academics (Crépeau & Scherzer, 1993; McAvinue et al., 2005).

Deficits in higher cognitive functions, particularly working memory and sustained attention, have been researched for TBI. There are known deficits in prefrontal and cingulate cortex during behavioral and cognitive assessments (Fontaine et al., 1999). Even when attention and inhibition performance (e.g., Stroop task) is equivalent to age-matched peers, brain activity differs with increased activation in right middle frontal gyrus, the medial frontal areas (including the anterior cingulate), the right dorsolateral prefrontal cortex, as well as the right superior and inferior parietal lobules in adolescents TBI (Tlustos et al., 2011). Smits et al. (2009) also found hyperactivation in adolescents with TBI compared to age-matched controls. For Smiths et al. (2009) these areas of hyperactivation during a Stroop task were in the right inferior and bilateral middle frontal gyrus, in the medial superior frontal gyrus (supplementary motor area), in the right inferior and superior parietal lobule and in the bilateral precuneus (Smits et al., 2009). Although these findings are limited to adolescents and the Stroop task, it appears that inhibition in TBI presents predominantly as hyperactivation of regions that are also active in controls.

In both populations, inhibition is critical to success in occupational, personal, and social endeavors. Although, research points to atypical inhibition in both ASD and TBI, the nature of this deficit between populations is very different. With ASD, a developmental disorder, impairments are typically explained by one of three theoretical models; the Theory of Mind Deficit, the Weak Central Coherence, and Executive Dysfunction Theory (Pellicano et al., 2006; Rajendran & Mitchell, 2007). Each theory focuses on a different deficit as the main contributor

to overall behavioral differences in ASD, whether that is (1) an inability to take the perspective of others, (2) an inability to see the whole gestalt of situations, or (3) executive dysfunction (Rajendran & Mitchell, 2007). Oznoff et al. (1991) first devised the Executive Dysfunction theory based on observations that there are similar atypical behaviors in ASD and TBI. Multiple theories, including the Executive Dysfunction Theory, on the nature of deficit in ASD are still being contended. Overall, given that ASD is heterogeneous in severity and presentation, it is important to study the individual and the big picture relationship between ASD and EFs.

TBI results in inhibition and EF dysfunction secondary to brain injury. Fontaine et al. (1999) found that closed head injury especially leads to neural damage of the white matter tracks and the frontal lobe. The diffuse damage to the brain may account for the lack the whole-brain integration that inhibition and cognitive control requires (Sharp et al., 2014). Typically, TBI disrupts processing across neural networks in the brain, given that the pathophysiology of the injury often includes diffuse axonal injury (Moreno-Lopez, Manktelow, Sahakian, Menon, & Stamatakis, 2017; Scheibel et al., 2003). Even at rest, individuals with TBI have widespread decreases in the neural oscillations that typically work in a coordinated manner to integrate information and reduced regional metabolism in prefrontal and cingulate cortices (Fontaine et al., 1999; Hellyer et al., 2015; Sharp et al., 2014).

While neuroimaging investigations of inhibition have been conducted in ASD and TBI, the results have not been compared between the two populations. Comparing developmental disorders with acquired disorders is the next step to uncover psychological premises of both populations (Rajendran & Mitchell, 2007). With inhibition, contributing foundationally to the overall common executive, delineating the neural signature of inhibition in both populations could provide several clinical benefits. First, learning about the neural nature of deficits is crucial

to creating interventions that are based on sound theory and that target core deficits. Second, identifying the neural sites of inhibition could act as a target for assessing neuroplasticity secondary to behavioral or medical interventions. Lastly, if the neural activation of inhibition in ASD and TBI is widely different, despite the fact that the presenting deficits are similar, then more information may be gained about variability of the brain networks underlying inhibition.

To identify the nature of the underlying *inhibition* sites and networks in TBI and ASD, this research will utilize coordinate-based meta-analytic tools to pool data across a large set of studies. The specific aims of this study are to identify the neural basis of inhibition in (1) TBI relative to healthy controls, (2) ASD relative to healthy controls, and (3) in TBI relative to ASD. The current hypothesis is that task-based activation for inhibition will be unique to each of the three populations. Given the lack of homogeneity in TBI and ASD demographics and the lack of focus on inhibition specifically in these populations, this topic is understudied. Providing a concise overview of current neural associations on this topic may help to address field gaps, direct future research, and support future intervention.

METHODS

Literature Search

PubMed, PsychINFO, Web of Science, and BrainMap were searched to obtain brain imaging studies of inhibition in adults with ASD and TBI. Studies were included if they (1) had adult participants (17 years+), (2) had functional magnetic resonance imaging studies with reported, significant activations, (3) reported whole brain voxel-wise analyses with coordinates reported in standardized space (Talairach and Tournoux or Montreal Neurological Institute [MNI] atlas systems), (4) were in English, and (5) were published in peer-reviewed journals.

Studies with only region of interest (ROI) effects were excluded to avoid bias about presupposed areas of inhibition activation.

In each database, the same terms were used to identify appropriate studies. See Figure 1 for full list of search terms. Pervasive Development Delay (PDD) and Asperger Syndrome were included terms as PDD was previously considered an umbrella category that included Autism, Asperger Syndrome, and PDD-Not Otherwise Specified (PDD-NOS), which were separate diagnoses from ASD that no longer exist under the most recent update to the *Diagnostic and Statistical Manual of Mental Disorder* – 5th edition (DSM-5). For this paper, 'inhibition' refers to the intentional suppression of information (i.e. response inhibition) (Miyake et al., 2000; Verbruggen & Logan, 2008). Inhibition tasks like the 'Go-No Go (GNG)', 'Stroop' and the 'Flanker,' were accepted for their relatively pure assessment of inhibition, as identified in previous studies comparing them to other EF-specific or complex EF tasks (e.g. Wisconsin Card Sort, antisaccades, or the Tower of London). Studies were combed to verify appropriateness of experiential task and meta-analytic inclusion criteria. Some studies claimed to test selective attention but were included because the task relied heavily on response inhibition. Figure 2 depicts the study evaluation process.

Studies that were not in the BrainMap database were coded using BrainMap Scribe 3.6 by the first author and checked by BrainMap reviewers for coding accuracy (P. T. Fox et al., 2005; P. T. Fox & Lancaster, 2002; Laird, Lancaster, et al., 2005; Vanasse et al., 2018). Once coded, experiments within the studies were reviewed for selection. Experiments are contrast analyses between two conditions that are performed by studies to reveal the targeted, residual activation effect. Experiments were included if the contrasted conditions revealed inhibition-related activation.

Five ASD studies and seven TBI studies met criteria (Figure 1), pooling data from *N*=145 TBI subjects and *N*=71 ASD subjects in 86 (44 TBI) experiments. Included contrasts analyses included 15 experiments (9 TBI) (Tables 2 and 3).

Control Data

To evaluate inhibition activation amongst control subjects, studies were used that were already in the BrainMap database. Control studies had to meet similar inclusion criteria as studies on ASD and TBI; i.e., reported foci for adults (17+ years of age), reported on normal mapping, and used inhibition experiential tasks that revealed activations (not deactivation) results. This yielded control data for 1431 subjects in 68 studies for 98 experiments meeting inclusion criteria.

Activation Likelihood Estimation (ALE) Technique and Meta-analytic Connectivity Modeling (MACM)

The primary statistical technique used was activation likelihood estimation (ALE), an accepted coordinate-based process that compares peak voxel-wise foci activations to determine regions or concentrations of above-chance activation from across multiple experiments (Eickhoff et al., 2009b; Laird, McMillan, et al., 2005). Reported foci from one study can be overlapped with foci from another study when, using ALE, they are represented as Gaussian functions (P. T. Fox & Lancaster, 2002, 2002; Laird, McMillan, et al., 2005). A Gaussian function is an algorithm used to analyze a continuous probability based on the normal distribution of linear equations. Treating foci as functions, instead of points, allows researchers to compute the union of probabilities at each voxel in a functional study. ALE assumes that the spatial distribution of foci within an experiment is fixed, allowing for random spatial association to be assessed between experiments. Multiple voxels that have significant convergence result in a whole brain

image, known as an ALE map. ALE maps are thresholded using a *p*-value or permutation-based Family-Wise Error (FWE). Once these maps are thresholded, a cluster analysis can be performed to distinguish final locations of significant clusters. GingerALE 3.0.2 software was used to distinguish foci ALE values and run cluster-analysis on thresholded ALE maps for initial and secondary contrasts (Eickhoff et al., 2009b, 2012; Turkeltaub et al., 2012). First-level analyses were completed to evaluate a population (ASD, TBI, Controls) with inhibition, then second-level analyses were completed to compare and contrast two first-level analyses. Result maps were overlaid on a high resolution MNI anatomical brain template using the image analysis viewer MANGO 4.1 software (http://ric.uthscsa.edu/mango/mango.html, Lancaster et al., 2010).

After the regions unique to each group were identified in the second-level analyses, meta-analytic connectivity modeling (MACM; (Eickhoff et al., 2009a; Robinson et al., 2009) was performed to inform study findings. This MACM analysis followed 4 steps, similar to those described above. First, regions of interest (ROI; made up of voxels) were created representing the unique clusters identified for each group in the ALE. These ROIs were used to separately search the BrainMap database for previous experiments in which activation was reported within that volume. The search was limited to studies coded for normal mapping, reporting activations only, and with normal, healthy subjects. Second, as in the ALE described above, those reported experiments are meta-analyzed to identify the *set* of regions most likely to be active when the volume of interest is active – these are called MACMs. These analyses were thresholded at $p < .001$ and corrected using the FWE correction at $p < .05$. Third, the two MACMs (for the lmedFG and the rmedFG ROIs) were then contrasted against one another to determine components of the MACMs that were specific to lmedFG (from the TBI&ASD ALE) versus rmedFG seeds (from the Controls ALE). Fourth, the union, or conjunction, indicating common activity across the two

MACMs was identified. Fifth, once these analyses were conducted, the meta-data for all of the experiments found in the MACM's first step was analyzed to determine the types of tasks used to elicit the neural activity – these data are characterized by behavioral domain and paradigm class. A behavioral domain is the area that an imaging task focused on. In Brainmap behavioral domains are divided into cognition, emotion, perception, interoperception, action, and pharmacology. Paradigm class refers to the type of experiment task used in the scanner, which is typically known by the same formal or informal name across researching groups (e.g., Stroop or Flanker).

RESULTS

First-Level Analyses, Within-group and Between-group ALEs

To investigate the differences between inhibition activation for individuals with ASD and TBI, a first-level analysis was conducted with a 0.01 *p*-value and no threshold correction (no FWE). This was to gain a simple overview of activation for each group without losing statistical power. For TBI, with 0.01 *p*-value uncorrected, there were three clusters. The largest cluster was in left and right middle frontal gyrus of the premotor cortex (peak at x= -8, y= 10, z= 52). All TBI clusters are in Table 4. For ASD, there were four clusters, with the largest cluster in the left anterior cingulate to cingulate gyrus (peak at x= 2, y= 32, z= 14). All ASD clusters are reported in Table 5.

Due to the small number of studies in both ASD and TBI groups, the populations of interest were combined (TBI&ASD) for the contrast analyses with controls. When correcting with a $p < 0.01$ cluster-level FWE and 0.05 *p*-value with 1000 permutations for initial-level analyses, the TBI&ASD combined group had one cluster, which was located in the bilateral anterior cingulate gyrus to the bilateral medial frontal gyrus (peak at x = -8, y =10 , z =52).

Cluster details are reported in Table 7. When correcting with a 0.01 cluster-level FWE and 0.001 *p*-value with 1000 permutations for initial-level analyses, the control group had seven clusters, with the largest extending from the right claustrum to medial frontal gyrus (peak at x =34, y =22, z =-6). The control group was analyzed at a $p < 0.001$ for presentation only, reported in Table 6. However, when contrast analyses were performed the same specifications were used on controls as on TBI&ASD ($p < 0.05$).

Second-level analyses – Contrast and Conjunction ALEs

To identify similarities and differences between the TBI&ASD and Controls, a contrast analysis was computed at a $p < 0.05$ with 10,000 permutations for TBI&ASD (first-level computed at 1000 permutations, 0.05 *p*-value and cluster-level of 0.01) and Controls (first-level computed at 1000 permutations, 0.05 *p*-value and cluster-level of 0.01). The common activity, or conjunction of the activation maps, was observed in the left medial frontal gyrus (peak at x = -8, y = 10, z = 52), extending into the right cingulate gyrus. The conjunction analysis is reported in Table 10.

In the contrast analyses, Controls had more activity than TBI&ASD in five clusters with the largest in the right middle frontal gyrus. Smaller clusters were located in the left thalamus to midbrain, left inferior frontal gyrus, the right cingulate gyrus to medial frontal gyrus, and the left sub-lobar claustrum (reported in Table 8). The Controls' peak activity in the medial frontal gyrus, similar to that of the TBI&ASD group, was in the right hemisphere (x = 6, y = 8, z = 50). TBI&ASD had more activity than Controls in one cluster in left medial frontal gyrus (peak at x = -10, y = 12, z = 52, reported in Table 9).

Meta-analytic connectivity mapping (MACM)

Given that the ALE analyses for both groups identified activity in the medial frontal gyrus, with a slight leftward peak in the TBI&ASD and a rightward peak in the Controls, MACMs were computed to determine whether these differences in peak activity were functionally relevant (i.e., connections to other brain regions differed for each of the peaks) and associated with differing behavioral patterns (i.e., activity was associated with different task types). The lmedFG volume of interest was reported in 480 BrainMap experiments (6820 subjects, contributing 7008 foci) and the rmedFG ROI was reported in 809 BrainMap experiments (11568 subjects, 11855 foci). These two peak ROIs demonstrated very similar MACMs (appendix Figures 4a and 5a) with a large set of regions commonly associated with both ROIs (Figure 5). However, they differed significantly with the lmedFG peak connecting with left inferior frontal and bilateral parietal regions (Figure 4, red) and the rmedFG peak connecting more with subcortical regions (thalamus, cerebellum). Reported MACM regions of activity reported in Tables 11 and 12.

Behavioral Domain and Paradigm Class analysis.

While the MACM maps were similar for each group and shared overlap with ALE results, the behavioral domain and paradigm class meta-data was significantly different between populations. The lmedFG peak ROI MACM (based on the peak from TBI&ASD ALE) was active during tasks categorized as cognition (working memory), execution of movement, and imagination with the following paradigm classes: task switching, counting/calculation, and word generation (Figure 6 and 8). In contrast, the rmedFG peak ROI MACM (based on the peak from Control ALE) was active for tasks categorized as execution of movement and perception (vision.motion) and the following paradigm classes: anti-saccades and saccades (Figure 7 and 8).

DISCUSSION

Coordinate-based meta-analysis of functional magnetic resonance imaging activation studies was used to attempt to identify the neural basis of inhibition in TBI relative to HC, ASD relative to HC, and in TBI relative to ASD. However due to the limited number of studies on inhibition-related fMRI activation in adults with TBI and ASD, the research question morphed to maintain statistical power to identify the neural basis underlying TBI and ASD combined (TBI&ASD) relative to HC.

Findings Related to Attentional Networks

Using activation likelihood estimation (ALE) as a first-level analysis across a large number of neurotypical adults indicated involvement of several brain regions when engaged in response inhibition tasks. These brain regions, which include areas in the frontal, temporal, and parietal regions, include the bulk of brain regions that also make up the ventral and dorsal attention networks (VAN and DAN) (blue activation in Figure 2). Uncorrected and corrected ALE maps revealed activation in the TBI and ASD groups was similar to Controls, but did not encompass as many of the VAN or DAN regions as the Controls (red and green activation in Figure 2). The latter may be relevant to the presence of disorder but may also be due to the lack of power/number of studies included in the patient population groups.

However, by conducting comparison and conjunction analyses, it became evident that the medial superior frontal and lateral inferior frontal regions were commonly active across groups. Group differences were only evident in the peak activation observed within the large cluster of activity in the medial prefrontal cortex. That is, results suggest that TBI&ASD activate the left medial frontal gyrus (BA 6) in response inhibition tasks to a greater extent than controls do. Controls have significant activity in the right medial frontal and cingulate gyri. The largest and

most common cluster of activity (cluster 1, Table 6) in the neurotypical sample was in the right inferior frontal gyrus (pars orbitalis), extending inferiorly into the anterior insula (claustrum) and superiorly into the precentral gyrus. Other regions active included the rostral anterior cingulate cortex, the left-sided homolog of cluster 1, along with regions consistent with the dorsal attention network (left and right inferior to superior parietal lobules).

Though the sample was small, a descriptive analysis can probe patterns of neural activation differences in TBI, ASD, and controls. Inhibition-related activity in the TBI group was distributed in the left medial frontal gyrus, the left precentral gyrus extending to the insula, and the right middle frontal gyrus (Table 4). Activity in the ASD group centered in the left anterior cingulate and cingulate gyrus, in the left superior and middle temporal gyrus to inferior parietal lobe, in the left frontal gyrus, and in the left cingulate gyrus (Table 5).

These fMRI ALE activation results suggest that the TBI&ASD group are using less whole-brain activation and using more frontal region activation than controls. These frontal and prefrontal regions are typically associated with higher-level cognitive functions and involved in top-down processing (Corbetta & Shulman, 2002; Adam Gazzaley & D'Esposito, 2007; Vossel et al., 2014). The controls are activating more diverse neural regions that replicate those found in the VAN and DAN. Controls, likewise, are using neural regions that are typically associated with lower-level functions involved in bottom-up processing (e.g. the thalamus, temporal, and parietal regions), in addition to using higher-level regions. This suggest that the primary finding of this study is that controls are using both top-down and bottom-up processing to complete response inhibition tasks, whereas the TBI&ASD groups are relying on apparent top-down processing. However, it is unclear from these findings if the TBI&ASD group is not using other neural regions associated with a VAN or DAN approach, or if they have an inability to use those

regions. Further research on TBI and ASD during inhibition is warranted to confirm these findings of top-down processing.

The MACM results further suggest this finding between impaired inhibition and reduced VAN and DAN activation through both a task connectivity analysis and a behavioral/paradigm class analysis. The lmedFG MACM, based on the TBI&ASD ALE results, revealed a network of activation observed in studies that looked at cognitive (working memory) and motor execution domains, as well as, task switching, calculation, and other higher-level cognitive tasks. This supports the idea that the TBI&ASD were using higher-level abilities, recognized as part of top-down processing, to complete response inhibition. In contrast, the rmedFG MACM, based on the Controls ALE results, revealed a network of activation also observed in studies limited to action or perceptual domains. This included tasks saccades/anti-saccades, passive viewing, repetition, as well as reward and GNG tasks. This supports the claim that the Controls are using a network to complete successful inhibition that is based on bottom-up processing as evidenced through lower-level cognitive tasks. Further research is needed to explore the functional connectivity of ASD and TBI as it relates to cognitive control and attention.

Previous functional connectivity studies in ASD and attention have similarly found hypoactivation of the VAN and DAN (Farrant & Uddin, 2016). Studies on TBI and functional connectivity between the VAN and DAN are minimal, with most functional connectivity studies focusing on the Default-Mode Network (DMN). If ASD and TBI were under-activating the VAN and DAN, then this would interrupt task performance for a multitude of daily activities, thus continued research on this idea is warranted.

In addition to these suggestive findings on the relationship between attention and domain specific or general processing, the following sections provide a more in-depth discussion of

individual neural regions as they relate to TBI&ASD, HC, TBI, and ASD findings. The following neural regions were areas of significant activation in the ALE analysis.

Right and Left Medial Frontal Gyrus

The TBI&ASD group had significant activation in the left medial frontal gyrus, whereas the controls had significant activation in the right. Some prior fMRI studies investigating mild TBI during the Stroop task performance found no group differences within the lateral or medial prefrontal cortex (Mayer et al., 2012). However, this current study looked at a range of severity for TBI, so it may be that these findings are severity-dependent. In fact, a fMRI and EEG study comparing TBI and HC found that reduced activation in the mid-dorsolateral prefrontal cortex for the TBI group was associated with increased severity of symptoms (Nadia Gosselin et al., 2011). Some studies of inhibition in TBI, found increased activation on the right, not the left, medial frontal gyrus however those studies were Stroop-specific (Smits et al., 2009; Tlustos et al., 2011). This current study included a range of inhibition tasks to look at inhibition in a broad sense and future task-dependent analyses should be completed to piece apart task-dependent results.

Sometimes, the medial frontal gyrus is associated with the supplementary motor area (SMA). Several other studies have found the SMA to be related to inhibitory control in neurotypical individuals (Chikara et al., 2018; Ko et al., 2016; Smits et al., 2009). According to the Connectomic Atlas of the Human Cerebrum (2018) the right medial frontal gyrus in neurotypical individuals, activation occurs during social interactions (Baker et al., 2018). Some of the included inhibition tasks for this study included faces as stimuli and some did not. Future research should include specific social stimuli when addressing the medial frontal are in TBI and

ASD to more clearly ascertain activation surrounding social information, attention, and inhibition.

Right Middle Frontal Gyrus

This study found increased activation in the right middle frontal gyrus for the TBI group, but not the ASD group. This aligns with previous research about hypoactivation in the right middle frontal gyrus for TBI (Smits et al., 2009; Tlustos et al., 2011). A study looking at the Go-No Go task using magnetoencephalography to identify timing and location of neural patterns for adolescents with ASD and controls, found that the ASD group recruited areas limited to the frontal cortex and the right middle frontal gyrus in the beginning of the inhibition task (Vara et al., 2014). This may suggest that the right middle frontal gyrus plays some role, although is not primary, in inhibition. Messel et al. (2019) paper found in healthy controls preforming a stop-signal task (like the GNG) that right middle frontal gyrus, along with the inferior parietal cortex, right inferior frontal gyrus and left anterior insula was activated in the go-trials, but not in the stop-trials (Messel et al., 2019). A stopping-specific pattern was only seen in the supplementary motor area, the anterior cingulate cortex, and the right anterior insula (Messel et al., 2019). Although both go- and stop-trials are necessary to complete the GNG task, this may suggest that the right middle frontal gyrus is a supplementary or preliminary, but not a primary area for inhibition. Further research must delineate the primary and supplementary areas of inhibition in order to compare the impact of disruption on clinical populations.

Bilateral Cingulate and Cingulate Gyrus

This study found that the ASD group had significant activation in the cingulate gyrus, in the rostral anterior and mid-cingulate regions, while the TBI did not. Unlike this study, previous GNG and Flanker tasks report hypoactivation of the anterior cingulate gyrus has been found for

ASD (Kana et al., 2007; Shafritz et al., 2008). Also unlike this study, Smits et al. (2009) found increased activation of the bilateral cingulate gyrus in mild TBI, but this study included several tasks that the Smits et al (2009) analysis did not. Using fMRI with neurotypical adults, the anterior cingulate has been shown to interact with the dorsolateral prefrontal cortices during error detection of EF tasks (Gehring & Knight, 2000; Kiehl et al., 2000). When assessing task performance in error detection and sustained attention for adults with TBI, McAvinue et al. (2005) found a correlated decrease in both (McAvinue et al., 2005). Further research is required to delineate the relationship between the cingulate and ASD, and to clarify decreased cingulate activation as it relates to attention or inhibition.

Left Superior and Middle Temporal Gyrus

This study found significant activation in the superior and middle temporal gyrus for the ASD group and not the TBI group. Temporal activation is not typically seen during inhibition tasks in control populations (Mayer et al., 2012). One study looking at healthy older adults (50+ years) found reduced activity in the left temporal gyrus during a set-shifting activity (which would require inhibition), as well as reduced functional connectivity in the prefrontal cortex (Gerrits et al., 2015). Otherwise, little else on the temporal gyrus has been reported for inhibition-based tasks. However, several studies have noticed that activation in the temporal lobe is important in comprehension of emotions, particularly emotions in music or faces (Adolphs et al., 2001; Gosselin et al., 2011). Some of the inhibition tasks included did have faces as a part of the task, particularly in the ASD group, which may explain this activation in the ASD group and not the TBI group.

Left Inferior Parietal Lobe

This study found significant activation in the left inferior parietal lobe for the ASD group and not for the TBI group. Similar to this study, Kennedy et al. (2006) found bilateral inferior parietal activation when individuals with ASD performed an fMRI Stroop task (Kennedy et al., 2006). In neurotypical adults EFs are considered to rely on the frontoparetial network (D. E. Nee et al., 2007; Niendam et al., 2012), so it is interesting that the TBI group did not have significant parietal activation in this study. Previous studies have found increased activation in the parietal lobes for working memory tasks in mild TBI when compared to healthy controls and this was speculated to be compensatory activation in the case of damaged frontal regions (Wu et al., 2014). A study by Caeyenberghs et al. (2013) looked at brain connectivity during complex executive function tasks for TBI and found that the hubs of activation were present in the parietal cortex, frontal cortex, and the basal ganglia (Caeyenberghs et al., 2013). However the network connectedness between hubs had decreased efficiency and decreased integration (Caeyenberghs et al., 2013). These findings point to the need for more delineation in future investigation to determine if inhibition for TBI is disrupted by decreased activation in the parietal regions, decreased connectedness with the parietal region, or both.

Limitations and Future Directions

There are limitations of note in this meta-analytic study. First, the comparison between TBI and ASD neuroimaging studies is complicated by the variability of the activation experiments used. Experiments were varying in their stimulus modality and task content. However, this effect was minimized by limiting tasks to those of inhibition experiments with inhibition-central contrast analysis (Table 3). Similarly, our study did not rely on a specific task

(focused only on activation associated with Stroop), thus limiting the ability to identify results being based on task-specific activation.

Second, TBI and ASD are heterogenous populations with great within-group variability and complexity. Unfortunately, this disrupts research in both populations. To accommodate this reality, a large range of severity of TBI and ASD subjects were included, despite meeting other inclusion criteria (specifically they had to be 17+, they had no psychological co-morbidities, and they had to have a formal diagnosis of TBI or ASD by an appropriate professional body). Future research would benefit from focusing on a range of severity within specific population groups.

Third, the limited number of available studies in ASD and TBI populations can reduce statistical power and allows for individual studies to bias results. Combining groups, thresholding, and correcting analyses was done to mitigate the question of statistical power. BrainMap's ALE statistic attempts to correct the influence of individual studies by having analyses not impacted based on the threshold of significance employed in each original study (Turkeltaub et al., 2012). However, overall this was considered a preliminary, non-exhaustive research project with a broad scope and the findings should be considered as such. The findings are suggestive of future potential directions only.

Despite these limitations, this project gives a direction for further research in three ways. First, this project links two populations that have similar presentations of impaired inhibition. Future research should continue to explore the commonalities and difference between TBI and ASD. Second, this paper provides supporting claims to present knowledge on inhibition in HC, along with additional insight to the neural underpinnings of inhibition in TBI and ASD. Future research should continue to explore the neural differences of TBI and ASD in order to develop accurate theories for each population, particularly in regard to the role of attention. Third, this

paper points to a neural area, the medial frontal gyrus, as an area for future, more extensive analysis. This neural finding may present as a target for future functional connectivity analysis on inhibition in TBI or ASD, or it may provide a neurological target to observe treatment effects with. Future examination of similar findings on the neural infrastructure of inhibition in TBI and ASD is warranted so that the limitations mentioned may be overcome and so questions about the relationship between neurology and behavior in executive dysfunction for TBI and ASD can continued to be answered.

TABLES AND FIGURES

Table 1. Term for literature search used.

Search Terms Used in Databases	
Functional Magnetic Resonance Imaging	fMRI functional magnetic resonance imaging fMRI brain imag* brain map*
Autism Spectrum Disorder	Autism Spectrum Disorder ASD Autis* High Functioning Autism HFA Pervasive Development Delay PDD Aspergers
Traumatic Brain Injury	TBI Traumatic Brain Injury Head Injury
Inhibition	Common Executive Inhibit* Stroop Go-No-Go Flanker
Adults	Adult*

Figure 1. Diagram of Article Exclusion

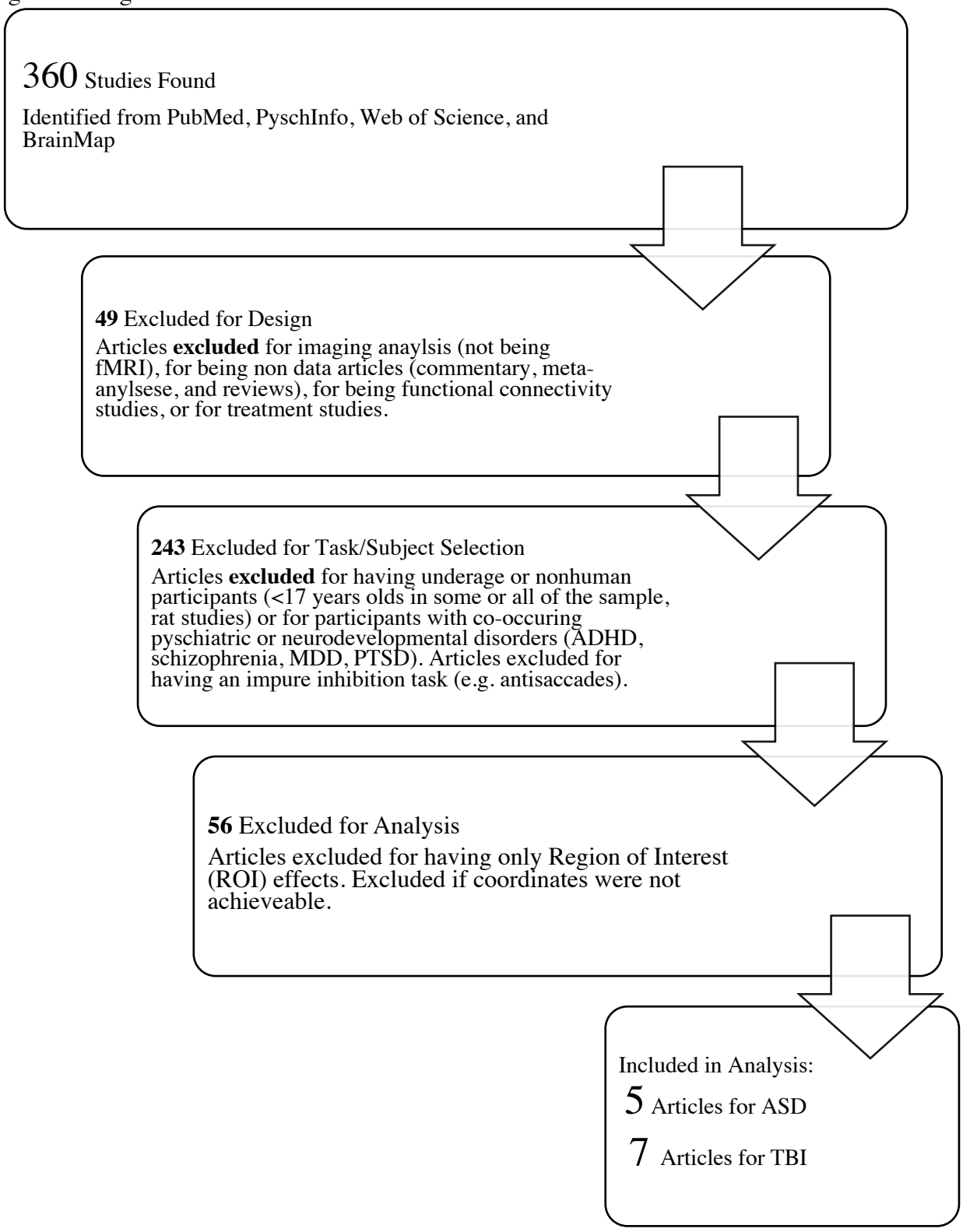


Table 2. Articles included in the study.

Study Year	**TBI Group**					**Control Criteria**	**Task Design**
	N (M:F)	**Mean Age**	**Diagnosis**	**Mean IQ and/or Edu in Years**	**Diagnostic Measures**		
Scheibel et al. (2007)	14 (11:3)	31.9 years (SD=24.0)	Moderate to severe TBI 9 MVA, 3 MA, 1 Fall, 1 Assault	Est. Preinjury (Barona) M= 98.4 (SD= 10.2) Edu= 13 (SD= 2.2)	GCS, number of brain lesions,	OI N= 10 (7:3), Age= 31.2 (SD=10.5)	Stimulus-response compatibility task; Red and Blue Arrows block-design
Scheibel et al. (2009)	30 (25:5); *__modTBI__: 9, __sTBI__: 8, __vsTBI__:13	__modTBI__: 46.32 (SD=7.29) __sTBI__: 22.46 (SD=3.99) __vsTBI__: 24.12 (SD=7.04)	Moderate to very severe TBI	Est. Preinjury (Barona) mTBI M= 101.89 (SD= 10.89), sTBI = 93.50 (SD=9.01), vsTBI = 99.08 (SD=9.45) Edu mTBI = 14.56 (SD= 2.74), sTBI = 13.5 (SD=2.51), vsTBI = 12.54 (SD=1.94)	Pre-resuscitation GCS	OI N=10 (7:3), Age= 30.8 (SD= 10.46)	Stimulus-response compatibility task; Red and Blue Arrows block-design
Scheibel et al. (2012)	15 (15:0)	28.73 (SD=5.97)	Mild TBI Explosive blast-related	Est. Preinjury (Barona) M= 103.27 (SD= 5.75) Edu= 13.8 (SD= 1.52)	PTA, injury with LOC less than 30 min,	Controls N=15 (14:1), Age= 30.93 (SD= 5.56)	Stimulus-response compatibility task; Red and Blue Arrows block-design
Sozda et al. (2011)	10 (6:4)	25.1 (SD=7.3)	Severe TBI 7 MVA, 2 MA, 1 Boating Accident	No IQ presented. Edu= 13.9 (SD=1.7)	GCS	Controls N=12, Age= 22.9 (SD=6.4)	Task switching cued-Stroop task

Sullivan et al. (2018)	17 (17:0)	31.7 (SD=6.8)	Mild TBI Explosive blast-related	No IQ presented. Edu= 14.7 (SD=1.6)	Verfaellie et al. (2013) Interview	Controls N=16 (14:2), Age= 33.1 (SD=5.6)	Flanker task
Terry et al. (2012)	22 (22:0)	20.3 (SD=1.17)	Mild TBI (2+ concussion)	No IQ presented. Edu= 14.3 (SD= 0.9)	Interview and questionnaire based on American Congress of Rehabilitation Medicine (ACRM) definition (1993)	Controls N=20 (20:0), Age=20.4 (SD=1.6)	Stroop color-word interference task
Ham et al. (2014)	48 (37:11)	35.7 (SD=10.9)	Probable to Moderate/Severe TBI grouped into low (N=18) and high (N=30)-performance monitoring groups Due to RTA, concussion, assault, or fall	No IQ or education presented	Mayo Classification (2007) which includes LOC, PTA, GCS, and neuroimaging.	Controls for fMRI task N= 25 (17:8), Age= 34.8 (SD=9.6)	Stop-signal task and Stop-change task
	ASD Group						
Fan et al. (2012)	12 (9:3)	30 (SD=6)	ASD and Asperger Syndrome (N=4)	Full scale IQ= 115 (SD= 14)	ADOS-G and ADI-R	N=12 (10:2), Age= 28 (SD=7)	Attention Network Test – Revised (ANT-R), which is a modified Flanker test.
Duerden et al. (2013)	16 (11:5)	27.2 (SD=5.3)	ASD	IQ= 111.89 (SD=13.71)	ADOS-G, ADI-R	N=17 (12:5), Age= 30.7 (SD=7.9)	Go/NoGo with emotional stimuli (happy or sad faces)

Velasquez et al. (2017)	19 (13:6)	25.84	ASD	IQ was above 80 for all participants.	ADOS and ADI-R	N=22 (16:6), Age= 29.03	Go/NoGo with faces and letters
Schmitz et al. (2006)	10 (10:0)	38 (SD=9)	High-functioning ASD, Asperger Syndrome	Full scale IQ= 105 (SD=14)	ADI and psychiatrist (DM) diagnosis with WHO ICD-10 criteria	N=12 (12:0), Age= 39 (SD=6)	Go/NoGo, Stroop, and SWITCH tasks
Dichter et al. (2008)	14 (13:1)	22.9 (5.2)	High-functioning ASD and Asperger's	Edu= 12.79 (SD= 2.01)	ADI-R and ADOS	N= 15 (14:1), Age= 23.2 (SD=5.7)	Flanker task modeled after ANT-R

Codes: IQ= Intelligence Quotient, GCS=Glasgow Coma Scale, TBI=Traumatic brain injury, OI= Orthopedic Injury, MVA= Motor Vehicle Accident, MA= Motorcycle Accident, LOC=loss of confusion, PTA= post-traumatic amnesia, ASD= Autism Spectrum Disorder, ADOS-G= Autism Diagnostic Observation Schedule- Generic, ADI-R= Autism Diagnostic Interview-Revised, RBS-R= Repetitive Behavior Scale-Revised

*divided TBI participants into three groups (9 moderate, 8 severe, 13 very severe TBI)

Table 3. Selected experiments for contrast.

Study Year	Task Design	Experiment(s) Contrast	Foci	Stimuli Mode	Response Mode
TBI					
Scheibel et al. (2007)	Stimulus-response compatibility task	Incompatible minus Compatible, TBI Patients	18	Visual	Finger Tap/Button Press
Scheibel et al. (2009)	Stimulus-response compatibility task	Incompatible minus Compatible: Orthopedic Injury Controls (OI) < Severe TBI Incompatible minus Compatible: OI < Very Severe TBI (vsTBI)	11 11	Visual	Finger Tap/Button Press
Scheibel et al. (2012)	Stimulus-response compatibility task	Incompatible > Compatible, TBI Group	3	Visual	Finger Tap/Button Press
Sozda et al. (2011)	Stroop task	Incorrect > Correct, sTBI	4	Visual	Finger Tap/Button Press
Sullivan et al. (2018)	Flanker task	Incongruent > Congruent, Mild Traumatic Brain Injury (mTBI)	4	Visual	Finger Tap/Button Press
Terry et al. (2012)	Stroop task	Stroop Incongruent > Congruent, mTBI	11	Visual	Finger Tap/Button Press
Ham et al. (2014)	Stop-signal task and Stop-change task	Low-Monitoring TBI (incorrect STOP>correct GO) > Controls (incorrect STOP>correct GO) High-Monitoring TBI (incorrect STOP > correct GO) > Control group (incorrect STOP > correct GO)	6 4	Visual	Finger Tap/Button Press
ASD					
Fan et al. (2012)	Flanker task modeled after ANT-R	Flanker (incongruent-congruent), HC > ASD	3	Visual	Finger Tap/Button Press
Duerden et al. (2013)	Go/NoGo	NoGo-Go, ASD only	7	Visual	Finger Tap/Button Press
Velasquez et al. (2017)	Go/NoGo	Face NoGo (ASD > HC) > Face Go (ASD > HC)	1	Visual	Finger Tap/Button Press
Schmitz et al. (2006)	Go/NoGo, Stroop, and SWITCH tasks	Go/No-Go, Autism Spectrum Disorder (ASD) Stroop, ASD	6 9	Visual	Finger Tap/Button Press
Dichter et al. (2008)	Flanker task modeled after ANT-R	High-arousal > Low-arousal pictures, Incongruent arrows, ASD Only	3	Visual	Finger Tap/Button Press

Table 4.
Uncorrected TBI only activations, $p < 0.01$, three clusters found.

Cluster #	x	y	z	ALE	Z	Label (Nearest Gray Matter within 5mm)	Voxels	Mm^3
1	**-8**	**10**	**52**	0.01230745	4.20	Left Medial Frontal Gyrus, BA 6	264	2112
2	**-46**	**16**	**6**	0.0097824	3.697	Left Precentral Gyrus and Left Insula, BA 44 and 13	172	1376
	-46	*10*	*0*	0.00896968	3.542			
3	**42**	**4**	**50**	0.00928279	3.603	Right Middle Frontal Gyrus, BA 6	142	1136
	32	*8*	*62*	0.00738903	3.102			

Table 5.
Uncorrected ASD only activations, $p < 0.01$, four clusters found.

Cluster #	x	y	z	ALE	Z	Label (Nearest Gray Matter within 5mm)	**Voxels**	Mm^3
1	**2**	**32**	**14**	0.00866706	4.0707	Left Anterior Cingulate and Cingulate Gyrus BA 24 and 32	548	4384
	2	*30*	*22*	0.00829139	3.9433			
	0	*28*	*32*	0.00740079	3.5924			
	0	*16*	*40*	0.00641351	3.228			
2	**-52**	**-50**	**18**	0.01166451	4.649	Left Superior Temporal Gyrus, Middle Temporal Gyrus, and Inferior Parietal Lobe BA 22, 39, and 40	406	3248
	-56	*-66*	*16*	0.00697456	3.3911			
	-50	*-46*	*32*	0.00647548	3.290			
3	**-34**	**26**	**-14**	0.00869964	4.077	Left Frontal Gyrus, BA 47	224	1792
4	**2**	**2**	**36**	0.00722067	3.512	Left Cingulate Gyrus, BA 24	199	1592
	-2	*-6*	*28*	0.00653593	3.303			

Table 6.
Corrected Control only activations, cluster-FWE at 0.01, $p < 0.001$ for presentation only, 1000 permutations.

Cluster #	x	y	z	ALE	Z	Label (Nearest Gray Matter within 5mm)	Voxels	Mm^3
1	**34**	**22**	**-6**	0.05808274	7.1622	Right Claustrum, Precentral Gyrus, Insula, and Inferior and Middle Frontal Gyrus, BA 9	2153	17224
	44	*10*	*34*	0.05365165	6.745			
	40	*18*	*0*	0.04915744	6.310			
	50	*26*	*26*	0.04601312	5.996			
	42	*38*	*20*	0.04329699	5.720			
	52	*18*	*18*	0.04043961	5.421			
2	**4**	**20**	**42**	0.07651953	8.791	Right Cingulate Gyrus and Medial Frontal Gyrus and Left Medial Frontal Gyrus, BA 32 and 6	1657	13256
	6	*32*	*26*	0.04254507	5.642			
	0	*6*	*50*	0.04209139	5.594			
	6	*2*	*58*	0.03224389	4.516			
3	**-34**	**18**	**4**	0.0535029	6.731	Left Insula, Precentral Gyrus, Inferior Frontal Gyrus, and Claustrum, BA 13, 9, 44,	1197	9576
	-42	*8*	*32*	0.04097684	5.478			
	-42	*14*	*-6*	0.03738919	5.094			
	-52	*10*	*18*	0.0352783	4.861			
	-32	*20*	*-8*	0.03403825	4.722			
	-52	*10*	*6*	0.03269097	4.568			

	-46	*20*	*28*	0.02617437	3.788			
4	**40**	**-46**	**48**	0.04242817	5.630	Right Inferior Parietal Lobule, Angular Gyrus, and Precuneus, BA 40, 38, 7	676	5408
	36	*-56*	*44*	0.04235259	5.621			
	24	*-68*	*48*	0.03602877	4.944			
	14	*-68*	*52*	0.03572682	4.911			
5	**-42**	**-46**	**50**	0.04870766	6.2658925	Left Inferior Parietal Lobule and Angular Gyrus, BA 40, 39	422	3376
	-34	*-56*	*42*	0.02848489	4.0725317			
6	**56**	**-44**	**38**	0.03474468	4.8007	Right Inferior Parietal Lobule, BA 40	160	1280
7	**-24**	**-66**	**48**	0.03304441	4.6083403	Left Superior Parietal Lobule, BA 7		

Table 7.
Corrected TBI&ASD combined group activations, cluster-FWE 0.01, $p < 0.05$ for presentation only, 1000 permutations.

Cluster #	x	y	z	ALE	Z	Label (Nearest Gray Matter within 5mm)
1	**-8**	**10**	**52**	0.01232191	4.028	Left Medial Frontal Gyrus, Cingulate Gyrus, and Anterior Cingulate. BA 6, 32, 24, 8, 9
	14	*24*	*28*	0.00953422	3.469	
	-4	*26*	*30*	0.00947309	3.458	
	2	*32*	*14*	0.00866722	3.292	
	2	*30*	*22*	0.00836742	3.226	
	12	*24*	*46*	0.00731156	2.920	
	2	*2*	*36*	0.00722185	2.893	
	-12	*34*	*30*	0.00656421	2.683	
	-2	*-6*	*28*	0.00653645	2.678	
	0	*16*	*40*	0.00644578	2.647	

Table 8.
Controls > TBI&ASD, contrast set to $p < 0.05$ and 10,000 permutations.

Cluster #	x	y	z	Z	Label (Nearest Gray Matter within 5mm)
1	**26**	**46**	**32**	3.353	Right Superior and Middle Frontal Gyrus, Precentral Gyrus and Insula, BA 9, 8, 6, 44, 13
	40	*42*	*34*	3.121	
	32	*25*	*23*	0	
	33	*44*	*34*	2.968	
	44	*22*	*26*	0	
	44	*36*	*36*	2.929	
	38	*10*	*26*	2.878	
	18	*52*	*22*	2.706	
	43.3	*20*	*34.7*	2.697	
	56	*12*	*10*	2.370	
	48	*12*	*10*	2.301	
	18	*56*	*16*	2.254	
2	**-2**	**-18**	**-2**	2.370	Left Thalamus and Medial Nucleus, Left and Right Red Nucleus of Midbrain
	-6	*-20*	*-4*	2.264	
	-10	*-21*	*-2*	2.254	
	8	*-20*	*-4*	2.209	
	-3	*-25*	*-2*	2.149	
	-16	*-22*	*0*	2.115	
	-10	*-14*	*4*	2.081	
3	**-50**	**6**	**20**	2.820	Left Inferior Frontal Gyrus and Precentral Gyrus, BA 9
	-56	*8*	*22*	2.652	
	-36	*12*	*24*	2.556	
	-36	*14*	*30*	2.086	
4	**6**	**8**	**50**	2.636	Right Medial Frontal Gyrus and Cingulate Gyrus, BA 6, 24
	14	*8*	*46*	2.217	
	4	*-6*	*56*	2.167	
5	**-26**	**24**	**8**	2.706	Left Claustrum and Insula, BA 13
	-30	*26*	*8*	2.678	

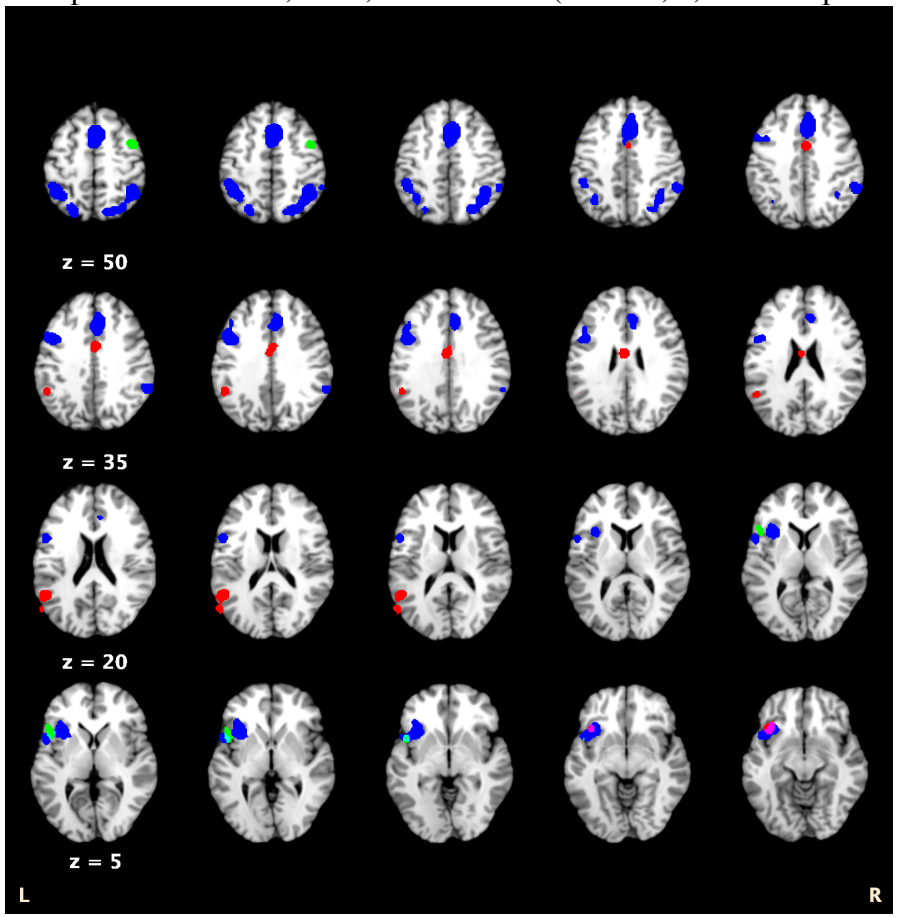
Table 9.
TBI&ASD > Controls, contrast set to $p < 0.05$ and 10,000 permutations.

Cluster #	x	y	z	Z	Label (Nearest Gray Matter within 5mm)
1	**-10**	**12**	**52**	3.719	Left Medial Frontal Gyrus, BA 6
	-14	*10*	*51*	3.291	

Table 10.
TBI&ASD conjunction Controls, contrast set to $p < 0.05$ and 10,000 permutations.

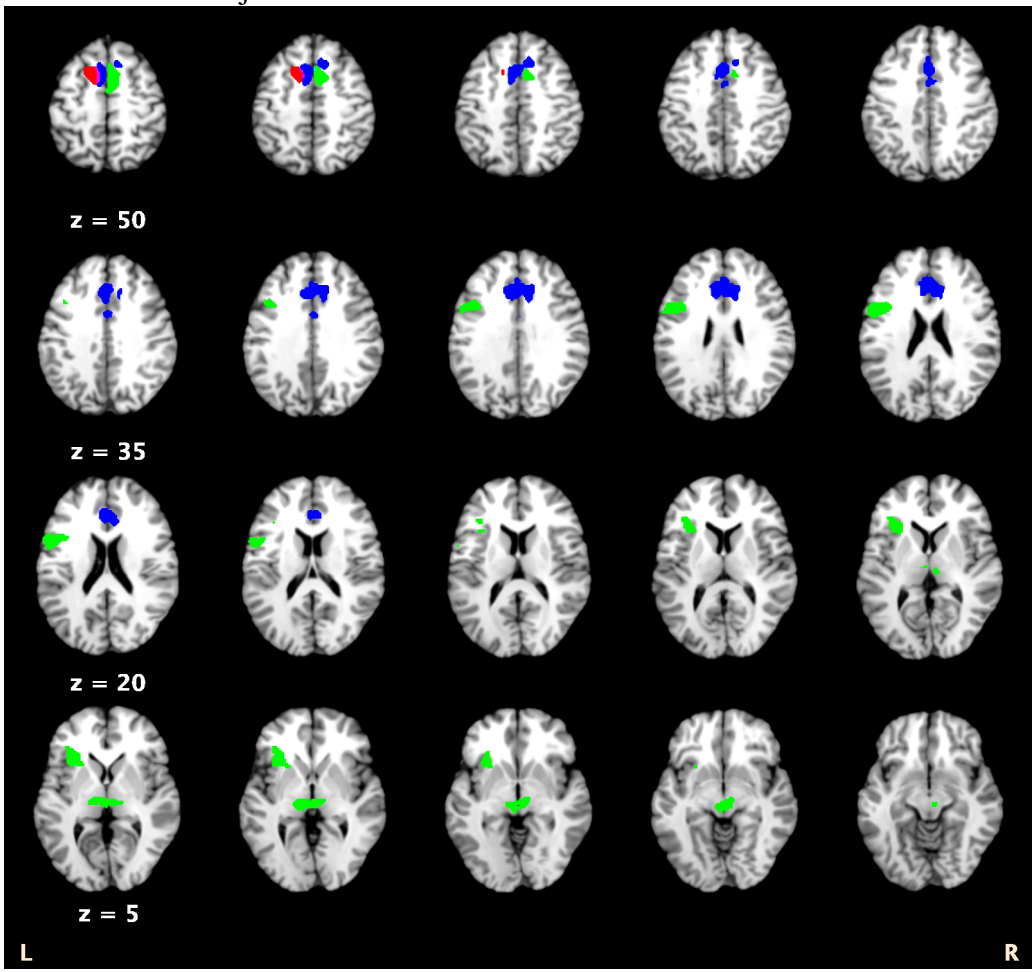
Cluster #	x	y	z	ALE	Label (Nearest Gray Matter within 5mm)
1	**-8**	**10**	**52**	0.012	Left and Right Medial Frontal Gyrus, Right and Left Cingulate Gyrus, BA 6, 8, 32, 24
	14	*24*	*28*	0.010	
	-4	*26*	*30*	0.009	
	2	*30*	*22*	0.008	
	12	*24*	*46*	0.007	
	2	*2*	*34*	0.007	
	0	*16*	*40*	0.006	

Figure 2.
Group results for TBI, ASD, and Controls (Table 4, 5, and 6 respectively) from first-level analysis.



Red = ASD only, Green = TBI only, Blue = Controls only, Pink = overlap between ASD and Controls, Turquoise = overlap between TBI and Controls

Figure 3.
Contrasts and Conjunction results.



Red = Unique to ASD&TBI, Green= Unique to Controls, Blue= Conjunction

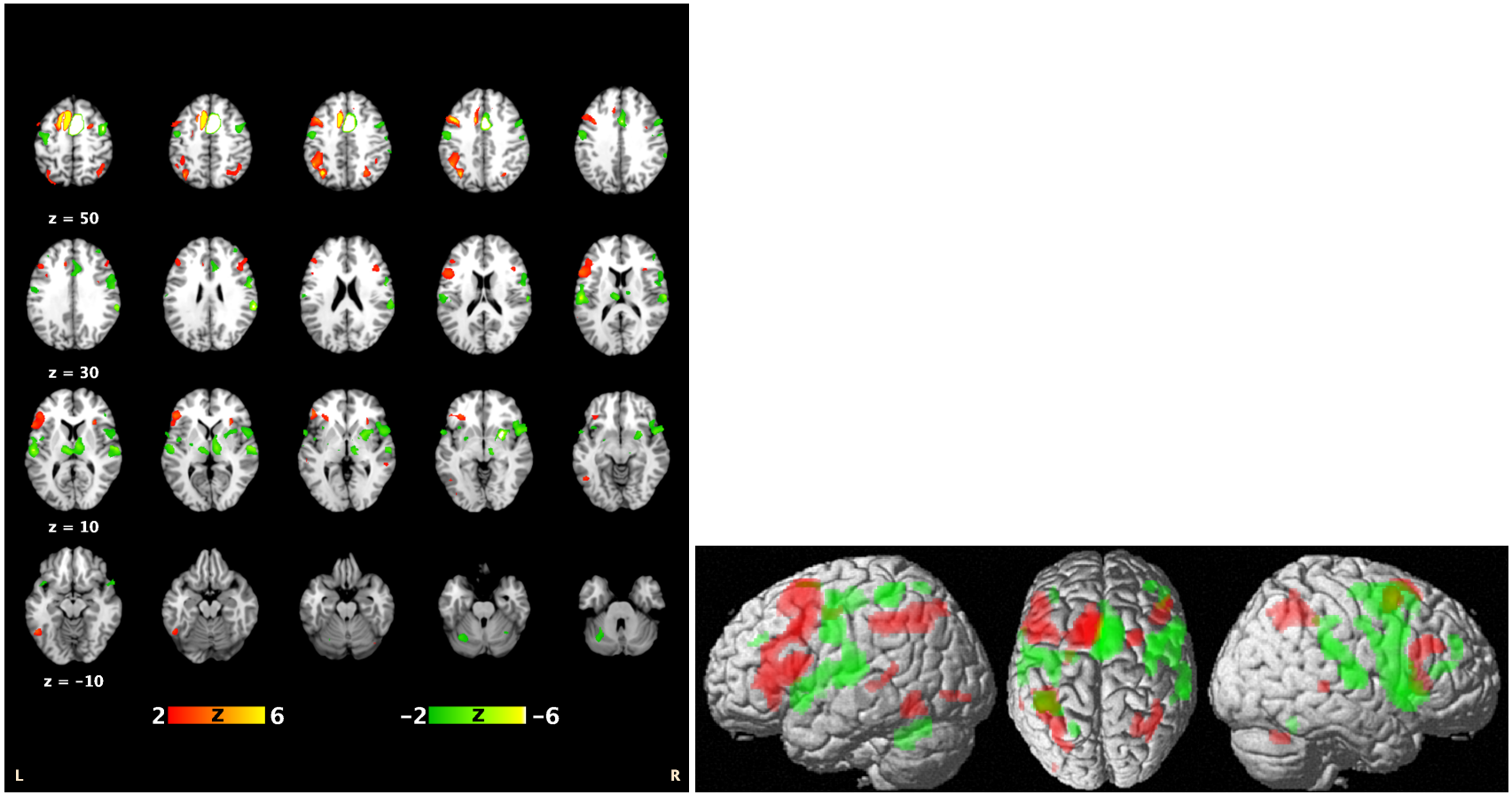
Table 11.
MACM results for related regions of activation in TBI&ASD.

Cluster #	x	y	z	ALE	Z	Label (Nearest Gray Matter within 5mm)
1	**-10**	**12**	**52**	0.19186415	20.21	Left Medial Frontal Gyrus, BA 6
2	**34**	**18**	**0**	0.04190031	6.862	Right Claustrum
3	**6**	**16**	**48**	0.03442418	5.967	Right Medial Frontal Gyrus, BA 32
4	**20**	**-66**	**48**	0.03582437	6.140	Right Precuneus, BA 7

Table 12.
MACM for related regions of activation in Controls.

Cluster#	x	y	z	ALE	Z	Label (Nearest Gray Matter within 5mm)
1	**6**	**10**	**52**	0.3565536	28.105	Right Medial Frontal Gyrus, BA 6
2	**38**	**22**	**-6**	0.07500941	8.293	Right Insula, BA 13
2	*46*	*16*	*-4*	0.07019604	7.873	
3	**-32**	**22**	**2**	0.06869555	7.741	Left Insula, BA 13
3	*-42*	*16*	*-6*	0.05143267	6.129	
4	**50**	**8**	**26**	0.06092195	7.034	Right Frontal and Precentral Gyrus, BA 9 and 6
4	*52*	*6*	*36*	0.0584417	6.803	
5	**-12**	**-18**	**6**	0.07760286	8.516	Left Thalamus
6	**10**	**-16**	**8**	0.07484397	8.279	Right Thalamus, Medial Dorsal Nucleus
7	**38**	**-2**	**50**	0.0577578	6.739	Right Precentral and Middle Frontal Gyrus, BA 6
7	*28*	*-2*	*52*	0.04922725	5.911	
8	**6**	**26**	**32**	0.05026608	6.015	Right Cingulate Gyrus, BA 32, and Left Cingulate Gyrus, BA 24
8	*-2*	*16*	*32*	0.04760294	5.747	
9	**-44**	**4**	**30**	0.05304677	6.288	Left Precentral Gyrus, BA 6

Figure 4.
Contrast MACM results.



Red = ASD+TBI, Green = Controls

Figure 5.
Conjunction MACM results.

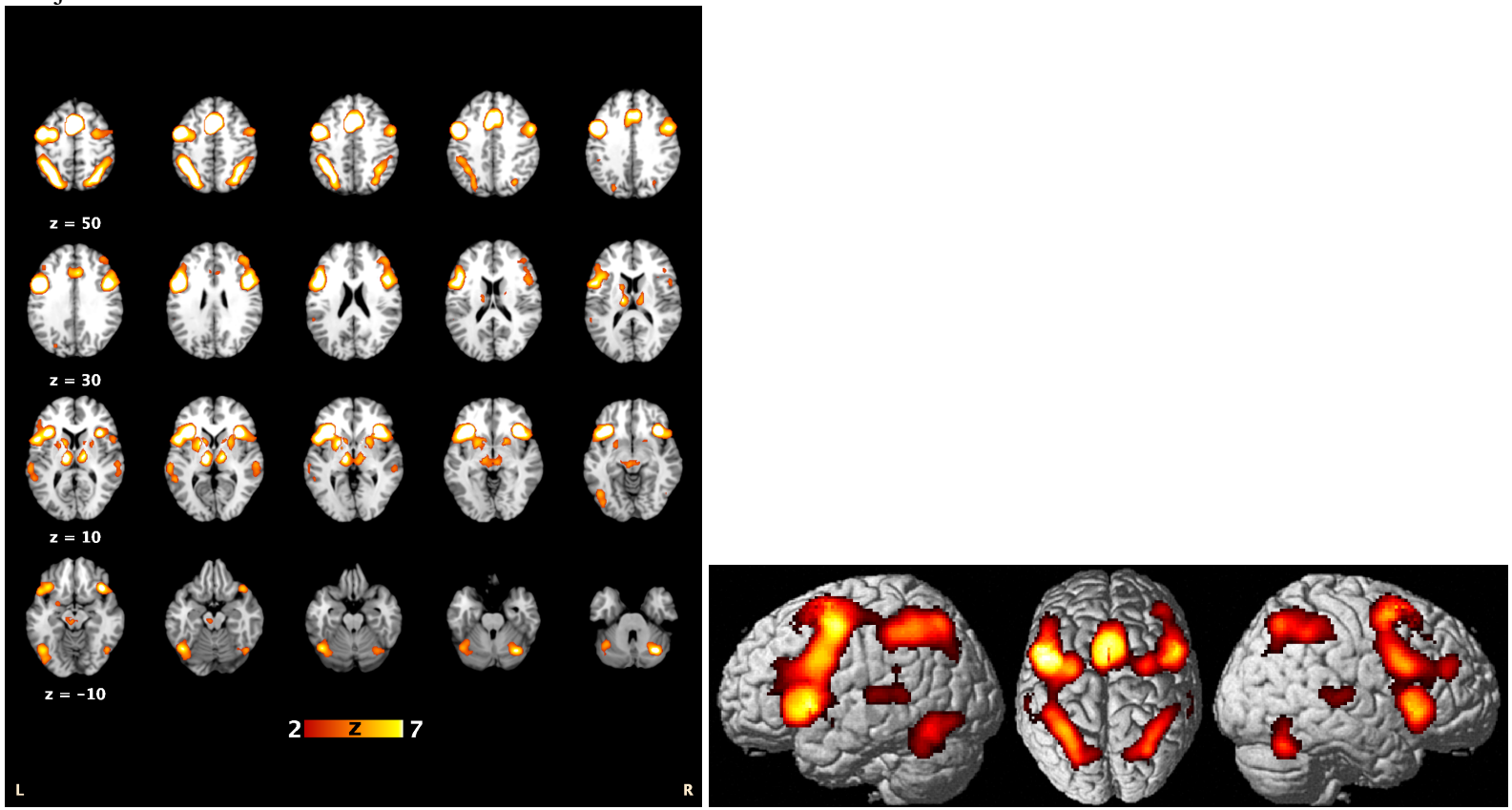
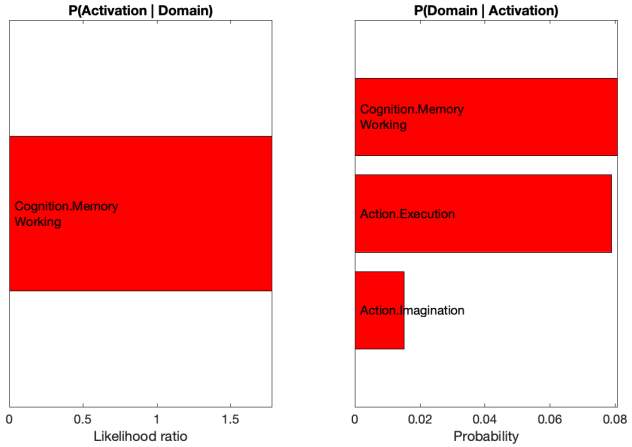


Figure 6.
MACM results depicting what types of tasks are activating the VOIs in TBI&ASD.



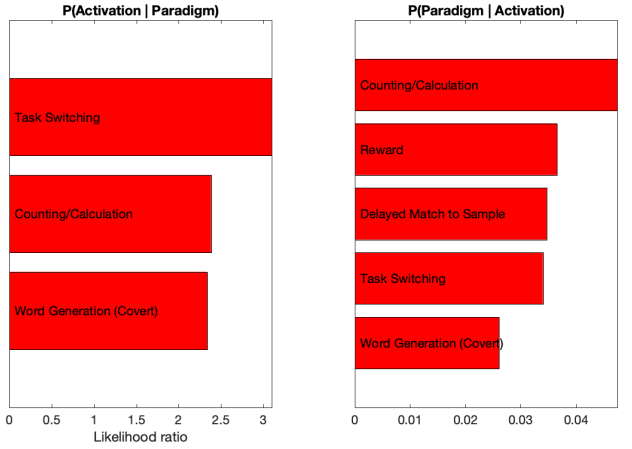


Figure 7.
MACM results depicting what types of tasks are activating the VOIs in Controls.

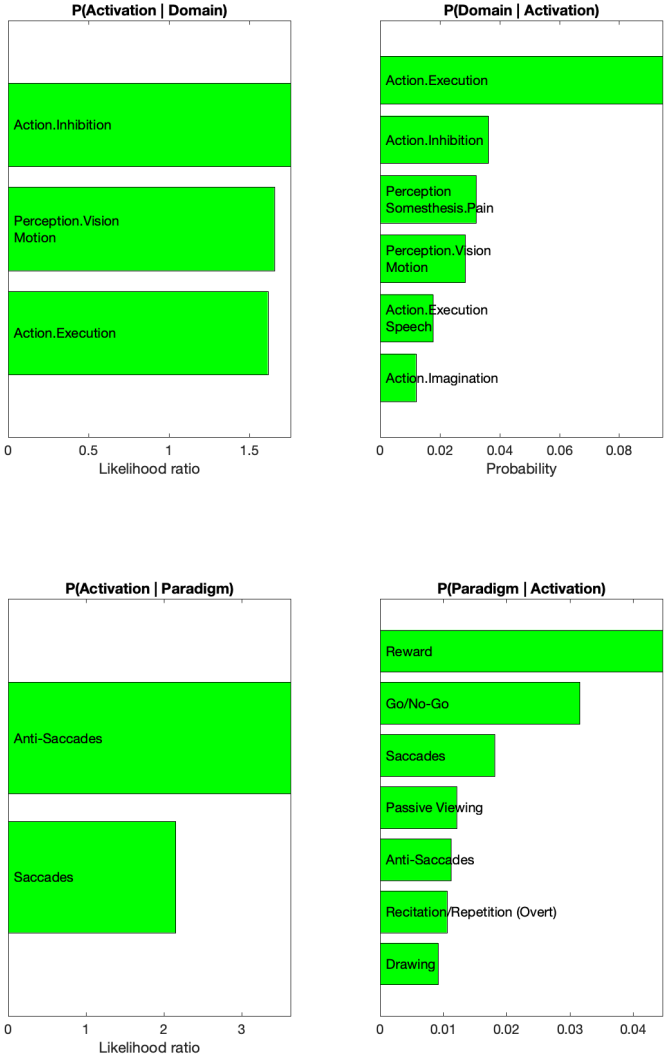


Figure 8.
MACM results depicting the contrast of task activation in ASD&TBI and Controls.

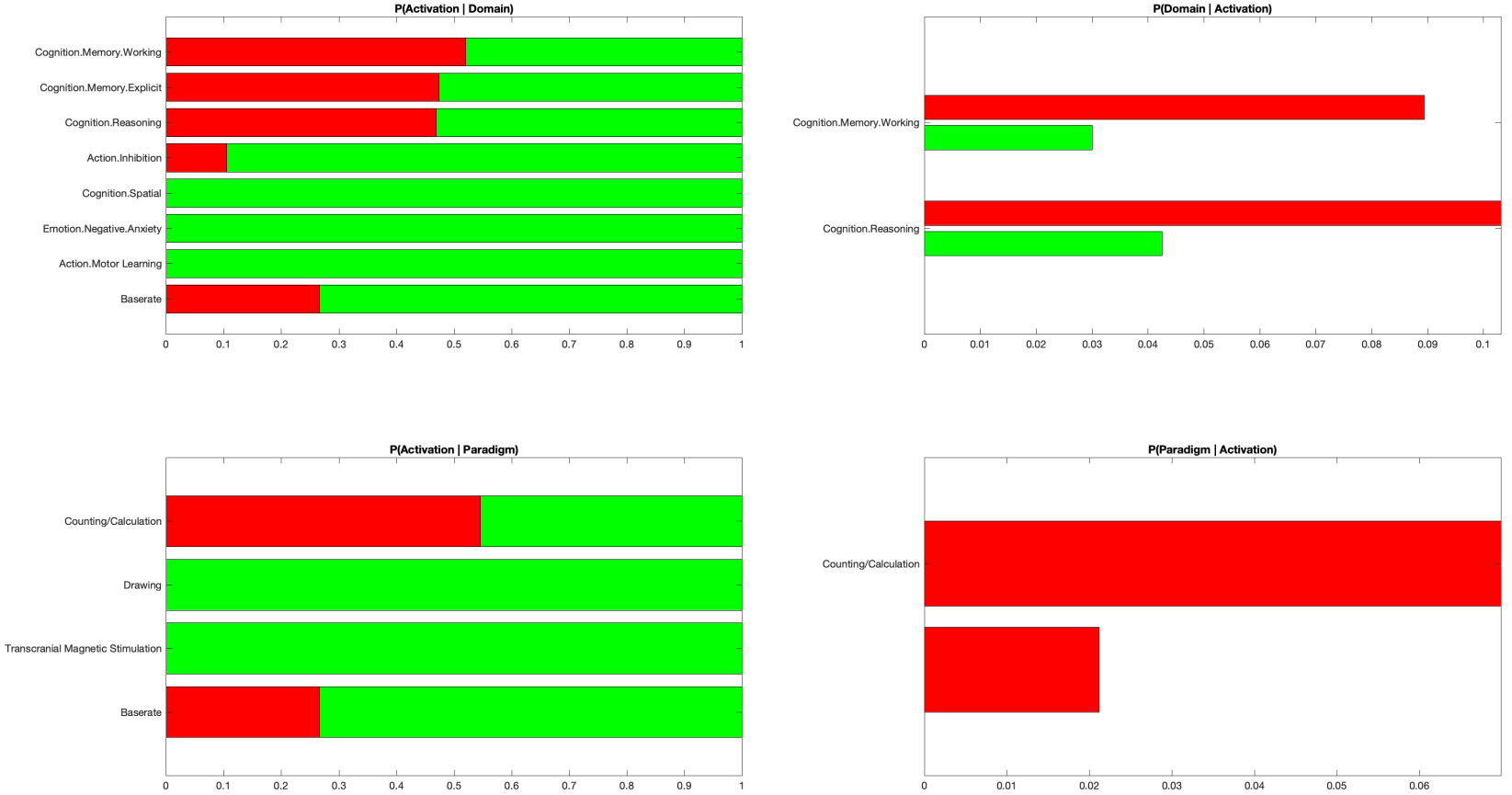


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Figure 1a.
Controls > ASD+TBI, contrast set to $p < 0.05$, 10,000 permutations.

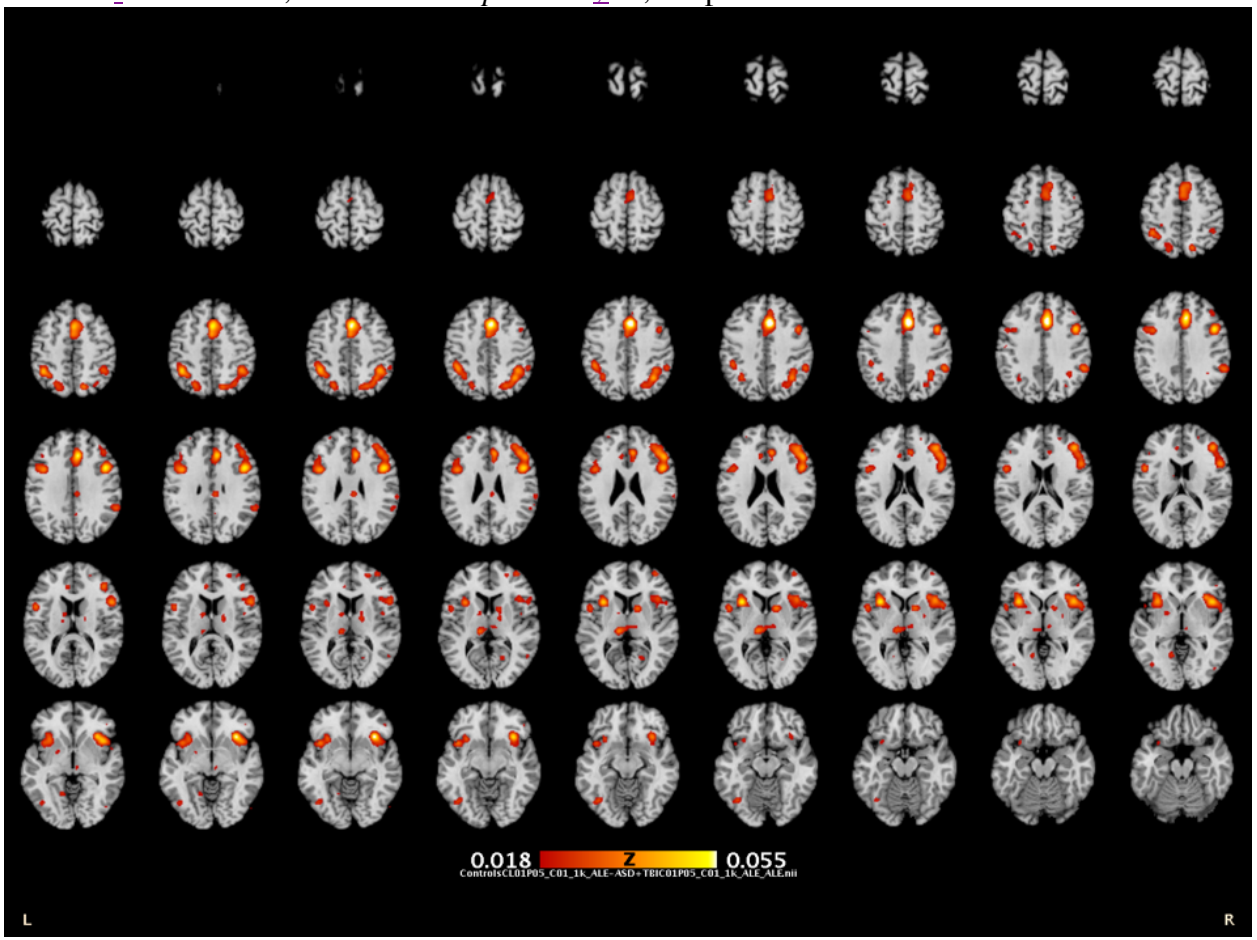


Figure 2a.
ASD+TBI > Controls, contrast set to $p < 0.05$ and 10,000 permutations.

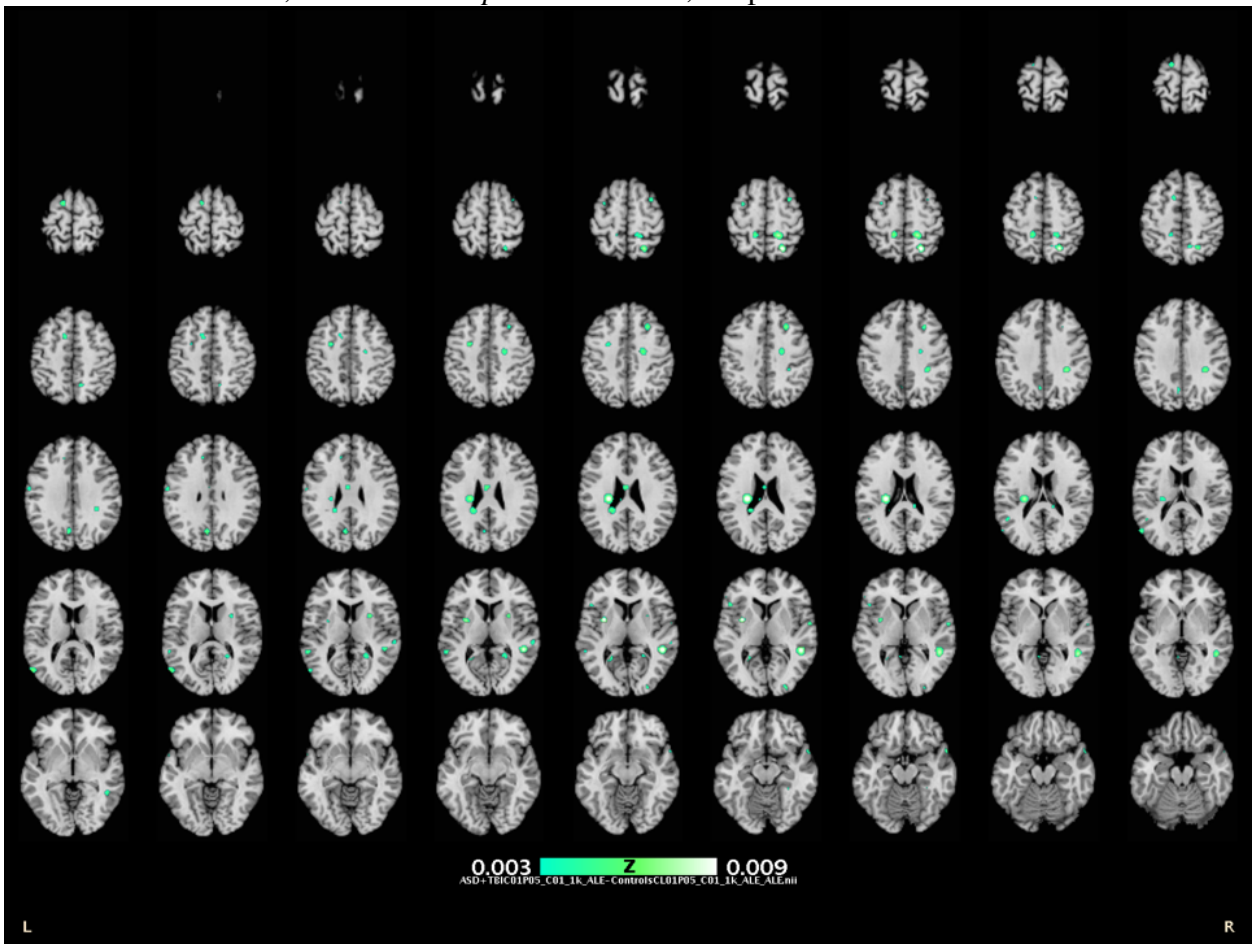


Figure 3a.
ASD+TBI conjunction Controls, contrast set to $p < 0.05$ and 10,000 permutations.

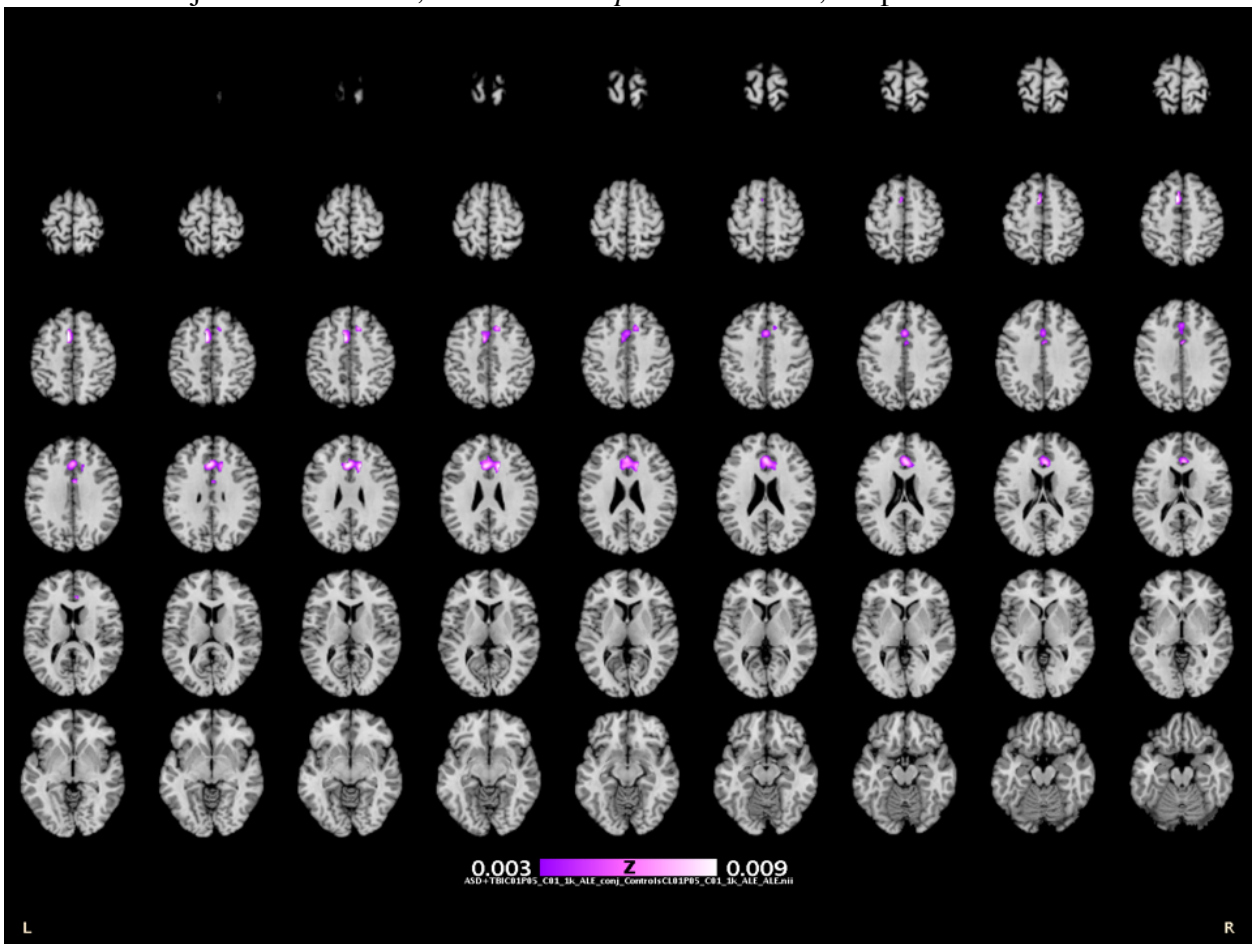
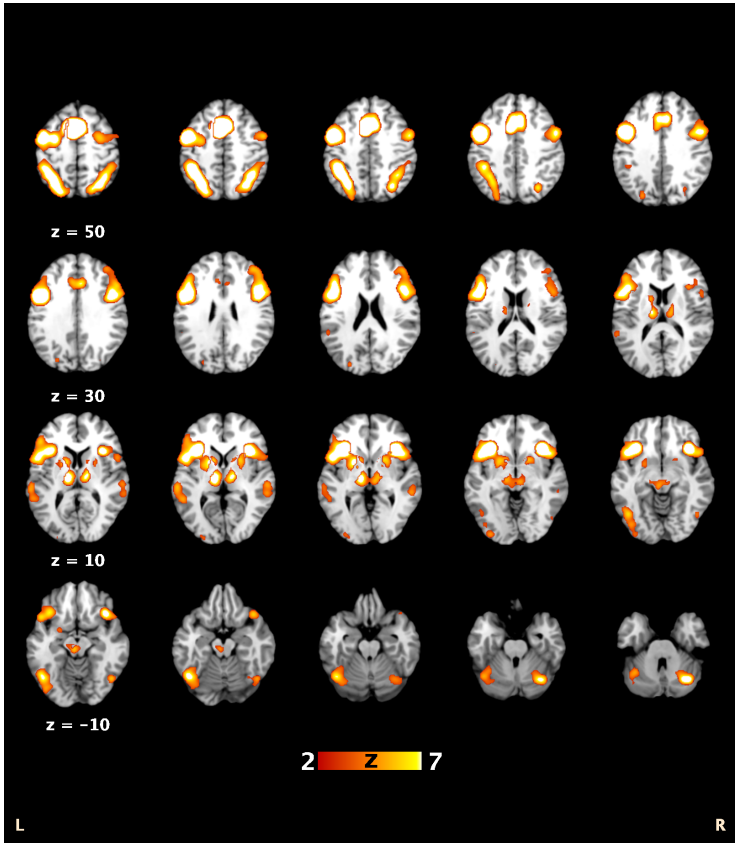


Figure 4a.
MACM results for task connectivity in the ASD&TBI group.



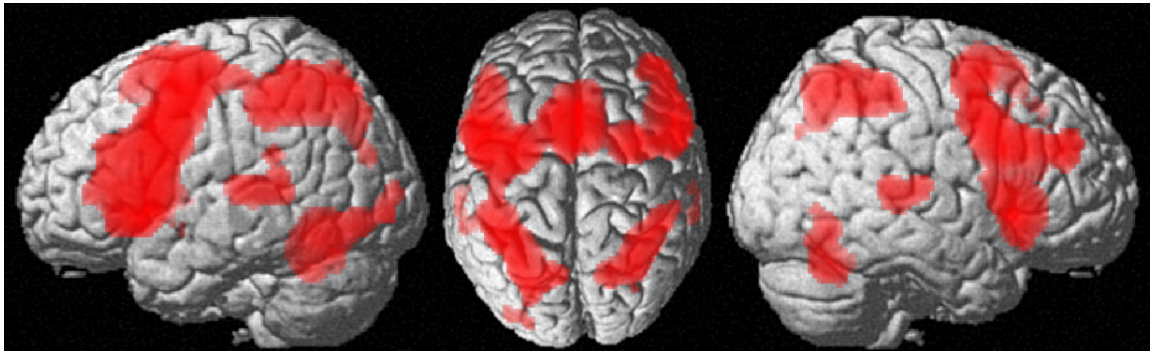


Figure 5a.
MACM results for task connectivity in the Control group.