Supplemental Information for

Task-based Functional Connectivity in Attention-Deficit/Hyperactivity Disorder: A Systematic Review

Investigations of functional connectivity in persisters/remitters, non-symptomatic siblings, and in relation to symptom severity

Studies identified in this systematic review comparing youth with ADHD with their non-symptomatic siblings tested inhibition-related functional connectivity differences (1,2), while studies comparing persisters and remitters focused on attention-related connectivity (3,4; Table 2 in the main text of the manuscript). Relative to non-symptomatic siblings, individuals with ADHD exhibited decreased connectivity of the striatum and increased connectivity of medial frontal/anterior cingulate and cerebellar regions. Additionally, both increases and decreases of connectivity have been observed across inferior and superior frontal cortex, middle temporal gyrus, sensorimotor, and parieto-occipital areas. Persistent ADHD was associated with lower connectivity of various fronto-striato-thalamic regions and greater connectivity of sensorimotor and middle frontal areas relative remitted ADHD. Across both comparison groups and both attention and inhibition paradigms, the right striatum emerged as a hub of decreased connectivity, while the precentral gyrus exhibited increased connectivity in ADHD compared to siblings/remitters. Finally, the right frontal pole showed both increases and decreases of functional connectivity in ADHD across all comparisons, with a 1:1 ratio.

One study tested the association between functional connectivity and symptom severity in adults with ADHD observing a negative relationship between ADHD severity and the connectivity between the left intraparietal sulcus and the right IFC, SFG, and postcentral gyrus (5).

Impact of sex, age, clinical presentations, and comorbities on the ADHD imaging literature

A number of patient-specific characteristics can impact on the findings emerging from the reviewed literature of task-based functional connectivity in ADHD. Sex differences, while prevalent in ADHD, are a relatively unexplored source of potential variability in the neuroimaging literature of ADHD. ADHD is more often diagnosed in males than females (6,7), which leads to a general male predominance of ADHD in both the clinic and in research (8). Sex differences have been observed in direct comparison studies of fMRI activation (9,10), as well as in a meta-regression analysis, showing that a greater number of females was related to greater hypoactivation in the left-hemispheric cerebellar and occipital regions during working memory (11). On the other hand, a well-powered study of inhibition-related brain activation and functional connectivity showed no sex differences in youth with ADHD (1,12). With regards to resting-state studies, mixed findings emerged, with some studies observing distinct sex-dependent patterns of connectivity in children (13) and adults (14) with ADHD, while others not observing differences (15). Although more research is needed to characterise sex differences in ADHD, the available literature indicates potential influence of sex on brain function in ADHD.

The imaging literature of ADHD is also dominated by paediatric cases, with considerably fewer studies focusing on adult ADHD. This was also reflected in the current review, with more than ⅔ of the included articles recruiting young people with ADHD. While ADHD was once considered a childhood disorder, it is now recognised that approximately 65-80% of childhood cases persist into adulthood (16–18) resulting in a prevalence of 2.5-5% of adults in the general population (19–24). Although there is evidence suggesting that adult ADHD is associated with similar neurofunctional deficits as paediatric ADHD (25–29), these abnormalities might be more pronounced in childhood (30–33). Nevertheless, further research is needed to better understand the neurofunctional developmental trajectories of ADHD.

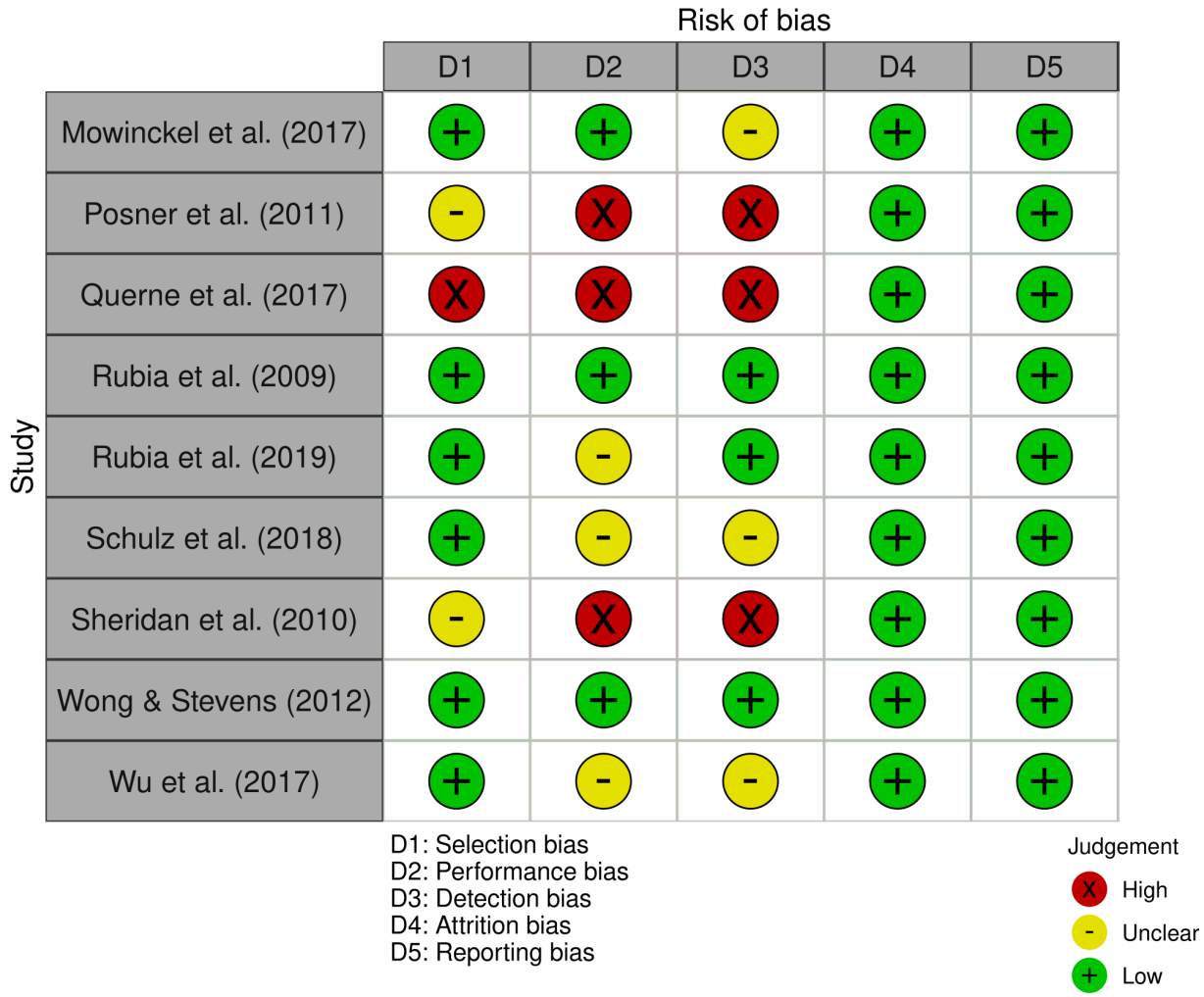
Interpretation of findings emerging from fMRI ADHD research is further complicated by the variability of clinical presentation and high degree of comorbidity with other psychiatric and neurodevelopmental conditions (34–37). Studies of ADHD neurocognition, including those reviewed here, tend to primarily recruit patients with combined or inattentive presentation. This is often motivated by greater clinical prevalence of those presentations (24) and the high susceptibility of fMRI techniques to motion artefacts. Excessive motion is a recognised issue in neuroimaging in ADHD. Exclusion due to inability to complete the protocol or excessive motion during scanning may theoretically bias the included groups to those with lower hyperactivity symptoms. Most studies included in the current review do not report ADHD presentation beyond average symptoms, with combined type making up the majority of those included (72% of participants across all reviewed studies). One larger study included in this review (N_{ADHD} = 185; $N_{control}$ = 124) reporting exclusions described 5% of the ADHD sample and 1% of the control sample being excluded due to excessive motion, but did not indicate if those excluded had a particular ADHD presentation (1). Overall, the bias towards samples with low hyperactivity symptoms in neuroimaging studies, combined with the evidence of distinct presentation-dependent cognitive (38–40) and neurofunctional (11,41–43) abnormalities urges caution in extrapolating findings from one clinical presentation to ADHD as a whole.

With regard to the impact of comorbidities, although there is evidence of disorder-specific inferior frontal cortex hypoactivation in ADHD relative to obsessive compulsive (31), autism spectrum (44), conduct (45), and paediatric bipolar disorders (46) during inhibition, this distinction is not as clear in other brain regions or during other cognitive functions. Shared deficits between ADHD and autism spectrum disorder have been observed in dorsolateral prefrontal, striato-thalamic, and superior parietal regions during sustained attention (47) and in the insula during response inhibition (44). Similarly, during sustained attention, both ADHD and obsessive-compulsive disorder were associated with hypoactivation in the insula, inferior frontal cortex, and cerebellum

(48). Shared dysfunction has also been observed during tasks of 'hot' executive functions in fronto-striatal regions compared to obsessive compulsive disorder (48,49) and in fronto-temporal regions relative to autism spectrum disorder (50). Consequently, although there is evidence that the disorder-specific neurofunctional impairment in ADHD may be context-dependent, with more distinct abnormalities during response inhibition, the wealth of evidence of shared dysfunction across other psychiatric disorders invites caution in the interpretation of results arising from comorbid populations.

These factors merely reflect the state and trend of the fMRI ADHD literature. Future studies will have to address these gaps by focusing on females with ADHD and by having a more comprehensive investigation of the development of ADHD through age. Finally, the impact of ADHD presentations and comorbidities will need more specific investigation to disentangle the general impairments observed in ADHD from the specific effects of the different presentations and comorbidities.

Cochrane risk of bias summary figure (51)



List of publications included in the systematic review:

1. Bedard, A.-C.V., Newcorn, J.H., Clerkin, S.M., Krone, B., Fan, J., Halperin, J.M., & Schulz, K.P. (2014). Reduced prefrontal efficiency for visuospatial working memory in attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 53*(9), 1020. https://doi.org/10.1016/j.jaac.2014.05.011

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