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## **The devil is in the detail: exploring the intrinsic neural mechanisms that link attention-deficit/hyperactivity disorder symptomatology to ongoing cognition.**

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# Psychological Medicine

## The Devil is in the Detail: Exploring the Intrinsic Neural Mechanisms that Link Attention-Deficit/Hyperactivity Disorder Symptomatology to Ongoing Cognition --Manuscript Draft--

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<b>Manuscript Region of Origin:</b>	UNITED KINGDOM
<b>Abstract:</b>	<p>Background: Attention-deficit/hyperactivity disorder (ADHD) is a developmental condition that profoundly affects quality of life. Although mounting evidence now suggests uncontrolled mind-wandering as a core aspect of the attentional problems associated with ADHD, the neural mechanisms underpinning this deficit remains unclear. To that extent, competing views argue for i) excessive generation of task-unrelated mental content, or ii) deficiency in the control of task-relevant cognition.</p> <p>Methods: In a cross-sectional investigation of a large neurotypical cohort (n = 184), we examined alterations in the intrinsic brain functional connectivity architecture of the default mode (DMN) and frontoparietal (FPN) networks during resting state functional magnetic resonance imaging (rs-fMRI) in relation to ADHD symptomatology, which could potentially underlie changes in ongoing thought within variable environmental contexts.</p> <p>Results: The results illustrated that ADHD symptoms were linked to lower levels of detail in ongoing thought while the participants made more difficult, memory-based decisions. Moreover, greater ADHD scores were associated with lower levels of connectivity between the DMN and right motor cortex, and between the FPN and right ventral visual cortex. Finally, a combination of high levels of ADHD symptomatology with reduced FPN connectivity to the visual cortex was associated with reduced levels of detail in thought.</p> <p>Conclusions: The results of our study suggest that the frequent mind-wandering observed in ADHD may be an indirect consequence of the deficient control of ongoing cognition in response to increasing environmental demands, and that this may partly arise from dysfunctions in the intrinsic organisation of the FPN at rest.</p>

1 **The Devil is in the Detail: Exploring the [Intrinsic](#) Neural Mechanisms that Link**  
2 **Attention-Deficit/Hyperactivity Disorder Symptomatology to Ongoing**  
3 **Cognition ~~at Rest~~**

4  
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25 **Abstract**

26

27 **Background:** Attention-deficit/hyperactivity disorder (ADHD) is a developmental condition  
28 that profoundly affects quality of life. Although mounting evidence now suggests  
29 uncontrolled mind-wandering as a core aspect of the attentional problems associated with  
30 ADHD, the neural mechanisms underpinning this deficit remains unclear. To that extent,  
31 competing views argue for i) excessive generation of task-unrelated mental content, or ii)  
32 deficiency in the control of task-relevant cognition.

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34 **Methods:** In a cross-sectional investigation of a large neurotypical cohort (n = 184), we  
35 examined alterations in the intrinsic brain functional connectivity architecture of the default  
36 mode (DMN) and frontoparietal (FPN) networks during resting state functional magnetic  
37 resonance imaging (rs-fMRI) in relation to ADHD symptomatology, which could potentially  
38 underlie changes in ongoing thought within variable environmental contexts.

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40 **Results:** The results illustrated that ADHD symptoms were linked to lower levels of detail in  
41 ongoing thought while the participants made more difficult, memory-based decisions.  
42 Moreover, greater ADHD scores were associated with lower levels of connectivity between  
43 the DMN and right motor cortex, and between the FPN and right ventral visual cortex.  
44 Finally, a combination of high levels of ADHD symptomology with reduced FPN connectivity  
45 to the visual cortex was associated with reduced levels of detail in thought.

46

47 **Conclusions:** The results of our study suggest that the frequent mind-wandering observed in  
48 ADHD may be an indirect consequence of the deficient control of ongoing cognition in  
49 response to increasing environmental demands, and that this may partly arise from  
50 dysfunctions in the intrinsic organisation of the FPN at rest.

51

52 **Keywords:** attention-deficit/hyperactivity disorder, default mode network, frontoparietal  
53 network, functional connectivity, mind-wandering, ongoing thought.

54

55

## 56 Introduction

57 Attention-deficit/hyperactivity disorder (ADHD) is a childhood onset developmental  
58 disorder with profound psychosocial consequences (Barkley and Fischer, 2010, Kieling *et al.*,  
59 2010) that often persist into adulthood (Faraone, 2007). In addition to the observed deficits  
60 in cognitive performance (Banaschewski *et al.*, 2012, Kofler *et al.*, 2013, McLean *et al.*,  
61 2004), it is commonly associated with a constellation of symptoms that include emotional  
62 lability (Skirrow *et al.*, 2009), dyslexia (Germano *et al.*, 2010) and mental health problems  
63 such as depression, anxiety, addiction and substance use disorders (Fayyad *et al.*, 2007).

64 One common feature of ADHD symptomatology is an elevated tendency for  
65 attentional lapses and reports of uncontrolled mind-wandering, i.e. periods when attention  
66 has shifted away from the current task goals. Both inside and outside the laboratory,  
67 individuals with ADHD characterise their mind-wandering experiences as excessively  
68 frequent, spontaneous and unintentional (Franklin *et al.*, 2014, Seli *et al.*, 2015), and  
69 describe their ongoing cognition as “thoughts that are constantly on the go, flitting from  
70 one topic to another, and multiple thoughts that appear at the same time” (Mowlem *et al.*,  
71 2016). Although converging evidence highlights frequent mind-wandering as a core aspect  
72 of ADHD symptomatology, the neural mechanisms that underlie this deficit remain unclear.

73 Contemporary accounts suggest that mind-wandering is a heterogeneous state that  
74 is not the product of a single mental process, but rather one that emerges from a  
75 *component process architecture* in which certain aspects of mental experience are produced  
76 by the combination of specific elements of cognition (Seli *et al.*, 2018, Smallwood, 2013,  
77 Smallwood and Schooler, 2015). For example, during off-task thought, attention is often  
78 focused on mental content generated from internal memory stores. Consequently,  
79 individuals, who retrieve information from memory more efficiently, engage in more off-  
80 task thought (Poerio *et al.*, 2017, Smallwood *et al.*, 2011). One possibility, therefore, is that  
81 uncontrollable mind-wandering associated with ADHD symptomatology results from  
82 excessive tendencies to self-generate mental content from memory.

83 In addition to being beneficial for psychological functions that require creativity  
84 (Baird *et al.*, 2012) and planning (Medea *et al.*, 2016), such excessive generation of off-task  
85 thought can also have negative consequences, chiefly because it can lead to errors in task  
86 performance (Smallwood *et al.*, 2008). Accordingly, neurotypical individuals tend to reduce

87 off-task experiences and increase task-related thoughts when performing more attention  
88 demanding tasks - a process known as *context regulation* (Smallwood and Andrews-Hanna,  
89 2013) linked to executive control (Bernhardt *et al.*, 2014, Kane *et al.*, 2007, McVay and Kane,  
90 2009, Mrazek *et al.*, 2012, Smallwood *et al.*, 2013b). An alternative perspective, therefore, is  
91 that alterations in patterns of ongoing thought emerge in ADHD because of problems in  
92 implementing a form of controlled cognition that is appropriate to the specific task context.

93 In relation to these competing views, recent advances in functional neuroimaging  
94 have provided the opportunity to evaluate changes in cognition that is linked to ADHD from  
95 a mechanistic perspective. For example, the default mode network (DMN) has been shown  
96 to reduce its activity under demanding contexts (Mazoyer *et al.*, 2001, Shulman *et al.*, 1997),  
97 and to increase activity during lapses in attention (Eichele *et al.*, 2008). Individuals with  
98 ADHD, however, are reported to lack such task-evoked activity dynamics – a pattern often  
99 taken as evidence of excessive self-generation of mental contents (Liddle *et al.*, 2011). In  
100 parallel, deficits in executive control (Barkley, 1997), and the dysregulation of associated  
101 neural systems such as the frontoparietal network (FPN) (Cortese *et al.*, 2012), are both  
102 well-documented elements of ADHD.

103 Based on this evidence, the current study aimed to compare and contrast the role of  
104 excessive generation of off-task thoughts and impaired context regulation in deficits of  
105 ongoing thought with respect to ADHD symptomatology, and to understand whether  
106 perturbation in either the connectivity of the DMN or the FPN at rest underpin these  
107 problems. For that purpose, we recruited a set of neurotypical participants who completed  
108 (i) a battery of questionnaires, including a well-established measure of ADHD, (ii) a  
109 laboratory-based thought sampling method measuring ongoing cognition, and (iii) a resting  
110 state functional magnetic resonance imaging (rs-fMRI) scan, which provided a measure of  
111 intrinsic neural organisation. A critical element of our design was that the thought sampling  
112 method used a behavioural paradigm that alternated between conditions that encouraged  
113 participants to restrict their thoughts to task focused information, and those that were  
114 more conducive to off-task thoughts (Smallwood *et al.*, 2009, Teasdale *et al.*, 1993). This  
115 paradigm, therefore, provided the opportunity to index both context regulation (i.e. the  
116 ability to increase task-relevant cognition when a task is demanding) and self-generation  
117 (i.e. the amount of off-task thought produced throughout the task as a whole) accounts of  
118 mind-wandering, allowing us to compare these views in relation to ADHD symptomatology.

## 119 **Methods**

### 120 **Participants**

121 Ethical approval for this study was obtained from the Department of Psychology and  
122 York Neuroimaging Centre, University of York ethics committees. All participants gave  
123 informed consent prior to taking part in the experimental assessments. A total of 226  
124 healthy, native English-speaker, right-handed participants were recruited subsequent to the  
125 study screening based on the following exclusion criteria: history of psychiatric or  
126 neurological illness, severe claustrophobia, anticipated pregnancy or drug use that could  
127 alter cognitive functioning. Out of this cohort, 184 participants fully completed the  
128 laboratory-based thought sampling and ADHD symptomatology questionnaire and were  
129 included in the initial analysis (mean = 20.13, SD = 2.24, range = 18-31, 121/63 female to  
130 male ratio).

131 Subsequently, all of these participants were scanned with a nine minutes long rs-  
132 fMRI during wakeful rest. A strict motion correction procedure (described in detail below)  
133 was utilised, which resulted in the further exclusion of nine participants, whereas three  
134 participants were removed due to problems associated with fMRI scanning. The average age  
135 for the final cohort of 172 participants suitable for the fMRI data analysis was 20.12 (SD =  
136 2.28, range = 18-31) with a 113/59 female to male ratio.

137

### 138 **Thought Sampling Method**

139 The participants' ongoing cognition was measured in a 30-minutes long behavioural  
140 paradigm that alternated between blocks of 0-Back and 1-Back conditions that manipulated  
141 working memory load (Fig. 1a). Non-target trials in both the 0-Back and 1-Back conditions  
142 were identical, consisting of black shapes (circles, squares or triangles) separated by a line,  
143 the colour of which signified whether the condition was 0-Back or 1-Back (mean  
144 presentation duration = 1050 ms, 200 ms jitter), counterbalanced across individuals. The  
145 non-target trials were followed by the presentation of a black fixation cross (mean  
146 presentation duration = 1530 ms, 130 ms jitter), and presented in runs of between 2 and 8  
147 trials with a mean of 5 trials after which a target trial or a multidimensional experience  
148 sampling (MDES) probe was presented. In either the 0-Back or 1-Back non-target trials,  
149 participants were not required to make a behavioural response.



150 During the target trials, participants were required to make a response, which  
151 differed depending on the task condition. In the 0-Back condition, the target trial was a pair  
152 of coloured shapes presented on either side of a coloured line with a probe shape in the  
153 centre of the screen. Participants had to press a button to indicate whether the central  
154 shape matched the shape on the left or right-hand side of the screen. In this condition,  
155 there was no need to retain the details of the non-target trials since the response trials  
156 could be completed based on the information on the screen, releasing working memory  
157 from task relevant information (i.e. easy perceptual decisions).

158 In the 1-Back condition, the target trial consisted of two coloured question marks  
159 presented on either side of a coloured line with a probe shape in the centre of the screen.  
160 Participants had to indicate using a button press whether the central shape matched either  
161 the shape on the left or right side of the screen on the previous (non-target) trial. Thus, in  
162 this condition, participants had to maintain the visuo-spatial array in working memory for  
163 each trial and use this information appropriately in the target trials (i.e. more difficult,  
164 memory-based decisions). This task is presented schematically in Figure 1a.

165 The contents of ongoing thought during this N-Back task was measured using MDES.  
166 On each occasion that the participants were asked about their thoughts, they rated their  
167 answers to the 13 questions presented in Table 1 using a 4-point Likert scale that ranged  
168 from 0 to 1. Participants always rated their level of task-focus first and then described their  
169 thoughts at the moment before the probe on a further 12 questions. MDES probes occurred  
170 on a quasi-random basis to minimise the likelihood that participants could anticipate the  
171 occurrence of a probe. At the moment of target presentation, there was 20% chance of a  
172 MDES probe instead of a target with a maximum of one probe per condition.

173 For the purpose of analyses, the ratings on the 13 MDES questions were  
174 decomposed into distinct patterns of thought that described the underlying structure of the  
175 participants responses. Following prior studies (Konishi *et al.*, 2017, Medea *et al.*, 2016,  
176 Ruby *et al.*, 2013a, Ruby *et al.*, 2013b, Smallwood *et al.*, 2016) we concatenated the  
177 responses of each participant at each probe and in each task into a single matrix and  
178 employed a principal component analysis (PCA) for factor reduction with Varimax rotation  
179 using SPSS (Version 23) (<https://www.ibm.com/products/spss-statistics>). We selected a  
180 total of four components based on the scree plot illustrated in Figure S1.

181

## 182 **ADHD Symptomatology Assessment**

183 With the aim of determining individual variability on the ADHD symptomatology of  
184 this neurotypical cohort, we administered the widely-used and validated Adult ADHD Self-  
185 Report Scale (ASRS-v1.1) (Kessler *et al.*, 2005, Kessler *et al.*, 2007). ASRS includes 18  
186 questions that reflect the main criteria for a DSM-IV-TR based ADHD diagnosis. Previous  
187 research has indicated that six out of the 18 questions were most predictive of an ADHD  
188 diagnosis (Gray *et al.*, 2014, Kessler *et al.*, 2005, Kessler *et al.*, 2007), constituting the Part A  
189 of this scale. Average self-reported responses on this subscale of ASRS was thus utilised in  
190 our subsequent analyses aimed at investigating the link between ADHD symptomatology,  
191 ongoing thoughts and neural organisation at rest.

192 In addition, based on recent reports suggesting a close link between ADHD  
193 symptomatology, depression and dyslexia (Fayyad *et al.*, 2007, Germano *et al.*, 2010,  
194 Skirrow *et al.*, 2009), we have also employed measures of these co-morbid symptoms to be  
195 removed as nuisance variables in our analyses. For depression, we used the Center for  
196 Epidemiologic Studies Depression Scale (Radloff, 1977); whereas for dyslexia the Dyslexia  
197 Adult Checklist (DAC) was utilised (Smythe and Everatt, 2001). The correlation between  
198 these measures and ADHD scores are provided in the Supplementary Material (Fig. S2).

199

## 200 **MRI Data Acquisition**

201 All MRI data acquisition was carried out at the York Neuroimaging Centre, York with  
202 a 3T GE HDx Excite MRI scanner using an eight-channel phased array head coil. Following a  
203 T1-weighted structural scan with 3D fast spoiled gradient echo (TR = 7.8 s, TE = minimum  
204 full, flip angle= 20°, matrix size = 256 x 256, 176 slices, voxel size = 1.13 x 1.13 x 1 mm<sup>3</sup>), a  
205 nine-minute resting state fMRI scan was carried out using single-shot 2D gradient-echo-  
206 planar imaging. The parameters for this sequence were as follows: TR = 3000 ms, TE =  
207 minimum full, flip angle = 90°, matrix size = 64 x 64, 60 slices, voxel size = 3 x 3 x 3 mm<sup>3</sup>, 180  
208 volumes. During resting state scanning, the participants were asked to focus on a fixation  
209 cross in the middle of the screen.

210

## 211 **MRI Data Preprocessing**

212 All preprocessing steps for the MRI data were carried out using the SPM software  
213 package (Version 12.0) (<http://www.fil.ion.ucl.ac.uk/spm/>) based on the MATLAB platform

214 (Version 16.a) (<https://uk.mathworks.com/products/matlab.html>). After removing the first  
215 three functional volumes to account for the magnetisation equilibrium, the remaining data  
216 was first corrected for motion using six degrees of freedom (x, y, z translations and  
217 rotations), and adjusted for differences in slice-time. Subsequently, the high-resolution  
218 structural image was co-registered to the mean functional image via rigid-body  
219 transformation, segmented into grey/white matter and cerebrospinal fluid probability  
220 maps, and were spatially normalized to the Montreal Neurological Institute (MNI) space  
221 alongside with all functional volumes using the segmented images and *a priori* templates.  
222 This indirect procedure utilizes the unified segmentation–normalization framework, which  
223 combines tissue segmentation, bias correction, and spatial normalization in a single unified  
224 model (Ashburner and Friston, 2005). Finally, all the functional images were smoothed using  
225 an 8 mm full width at half maximum (FWHM) Gaussian kernel.

226

### 227 **Functional Connectivity Analysis**

228 MRI data denoising procedures and the subsequent seed-based functional  
229 connectivity analyses were carried out using the *Conn* functional connectivity toolbox  
230 (Version 17.f) (<https://www.nitrc.org/projects/conn>) (Whitfield-Gabrieli and Nieto-  
231 Castanon, 2012). With the goal of ensuring that motion and other artefacts did not  
232 confound our data, we first employed an extensive motion-correction procedure and  
233 denoising steps, comparable to those reported in the literature (Ciric *et al.*, 2017). In  
234 addition to the removal of six realignment parameters and their second-order derivatives  
235 using the general linear model (GLM) (Friston *et al.*, 1996), a linear detrending term was  
236 applied as well as the CompCor method that removed five principal components of the  
237 signal from white matter and cerebrospinal fluid (Behzadi *et al.*, 2007). Moreover, the  
238 volumes affected by motion were identified and scrubbed based on the conservative  
239 settings of motion greater than 0.5 mm and global signal change larger than  $z = 3$ . A total of  
240 nine participants, who had more than 15% of their data affected by motion was excluded  
241 from the analysis (Power *et al.*, 2014). The distribution of average and maximum framewise  
242 displacement and global blood oxygen level dependent (BOLD) signal change, as well as the  
243 percentage of invalid scans in the final cohort utilised in this study are provided in Figure S3.  
244 Though recent reports suggest the ability of global signal regression to account for head  
245 motion, it is also known to introduce spurious anti-correlations, and thus was not utilised in

246 our analysis (Saad *et al.*, 2012). Finally, a band-pass filter between 0.009 Hz and 0.08 Hz was  
247 employed in order to focus on low frequency fluctuations (Fox *et al.*, 2005).

248 Following this procedure, we performed two separate seed-based functional  
249 connectivity analyses based on two regions of interest (ROIs) that were selected from the  
250 Yeo 7-Network parcellation scheme (Yeo *et al.*, 2011), namely the frontoparietal and default  
251 mode networks. For each participant, average BOLD signal from the binarised seed ROIs  
252 described above were correlated with time courses from the rest of the brain with the aim  
253 of obtaining individual connectivity maps. Group-level inferences on positive and negative  
254 connectivity of the chosen seed ROIs were made based on one-sample t-tests. Further linear  
255 regressions with FPN as well as DMN connectivity were performed with ADHD  
256 symptomatology as the variable of interest, while correcting for dyslexia, depression and  
257 the percentage of invalid scans based on the motion scrubbing procedure. All reported  
258 clusters were corrected for multiple comparisons using the Family-Wise Error (FWE)  
259 detection technique at the .05 level of significance (uncorrected at the voxel-level, .001 level  
260 of significance). Beta values representing connectivity of the clusters and the chosen seed  
261 ROIs that significantly explained individual variability in ADHD symptomatology, were then  
262 extracted for each participant for subsequent statistical analyses.

263

## 264 **Statistical Analysis**

265 We performed three main analyses to test the relationships between ADHD  
266 symptomatology, patterns of ongoing thought and their potential neural mechanisms. First,  
267 using a mixed Analysis of Variance (ANOVA) we examined the relationship between patterns  
268 of ongoing thought in the two tasks and variation in ADHD symptomatology with the aim of  
269 determining if their relationships support either the excessive self-generation, or the  
270 impaired context regulation accounts of ADHD, while correcting for depression and dyslexia.  
271 Second, we used linear regressions in seed-based functional connectivity analyses to  
272 identify how the intrinsic neural organisation varies with natural variation in ADHD  
273 symptomatology. For this, we included co-morbid depression, dyslexia scores and subject  
274 motion inside the scanner as nuisance variables. Finally, we examined whether patterns of  
275 shared variance in association between patterns of neural function and ongoing thought  
276 linked to ADHD using connectivity values (beta weights) obtained from the seed-based  
277 analysis and component scores from thought sampling during specific task contexts. In this

278 analysis, we repeated the mixed ANOVA from the first step of our analysis, additionally  
279 including the neural changes identified through our functional connectivity analysis as  
280 covariates. This last step allowed us to identify potential neural mechanisms that underpin  
281 ADHD related changes in patterns of ongoing thought.

282

283

## 284 Results

285 Our first analysis examined the relationship between ADHD and patterns of ongoing  
286 thought recorded in the laboratory session (Fig. 1a). Following a decomposition of the  
287 thought sampling data (Fig. 1b) we conducted a series of repeated measure ANCOVAs. In  
288 these models, while the dependent measure was the scores for each component of  
289 thought, the within participant factor was the task context (0-Back/1-Back) and the between  
290 participants factor was ADHD scores (correcting for depression and dyslexia). These analyses  
291 first revealed three components of thought that varied across the task conditions:  
292 “Detailed” ( $F_{(1,182)} = 9.24, p = .0027$ ), “Off-Task” ( $F_{(1,182)} = 4.98, p = .027$ ), and “Modality-  
293 Specific (Images/Words)” ( $F_{(1,182)} = 5.27, p = .023$ ) thoughts. “Emotion+” did not vary across  
294 the task conditions. In the 1-Back, thoughts were more detailed ( $M = .11, 95\% \text{ CI } [-.208,$   
295  $.002]$ ) than in the 0-Back condition ( $M = -.07, 95\% \text{ CI } [.028, -.17]$ ). Off-Task thoughts were  
296 more prominent in the 0-Back ( $M = .14, 95\% \text{ CI } [.237, .04]$ ) than in the 1-Back condition ( $M$   
297  $= -.15, 95\% \text{ CI } [-.057, -.246]$ ). Finally, thoughts were less in the form of words in the 1-Back  
298 ( $M = -.06, 95\% \text{ CI } [.037, -.175]$ ) than in the 0-Back condition ( $M = .07, 95\% \text{ CI } [.170, .06]$ ).

299 We also identified an ADHD by N-Back task condition interaction for the “Detailed”  
300 component ( $F_{(1, 182)} = 6.82, p = .0098$ ) of the reported thoughts. This interaction indicated  
301 that greater ADHD scores were linked to a smaller difference in the level of thought details  
302 reported in the 1-Back than the 0-Back task condition [Pearson  $r = -.19, p = .0046$ ] (Fig. 1c).  
303 Increasing levels of ADHD, therefore, were associated with reports of less detailed  
304 experiences in the more demanding 1-Back condition.

305 Our next analysis explored the association between brain functional connectivity at  
306 rest and levels of ADHD symptomology within our sample. After generating spatial maps for  
307 each individual that described the associations at the whole brain level for each of the two  
308 networks that formed the focus of our investigation (i.e. FPN and DMN) (Fig. 2a-b), we  
309 conducted two group level regressions. In these analyses we included mean centred ADHD  
310 scores as a between participant variable of interest, while controlling for potential  
311 confounds such as depression, dyslexia and the percentage of motion-based invalid scans.

312 These analyses revealed two differences. Higher ADHD scores were linked to  
313 reduced correlation between the FPN and a region of right lingual gyrus (visual cortex). In  
314 addition, higher ADHD scores were associated with reduced correlation between the DMN

315 and a region of right pre/post central gyrus (motor cortex) (Fig. 2c). Increasing levels of  
316 ADHD within our sample, therefore, were linked to reduced correlation between  
317 transmodal association cortices (DMN, FPN) and unimodal sensorimotor cortices.

318 Thus far we have identified the correlates of ADHD symptomology with both  
319 patterns of ongoing thought and neural organisation. Our final analyses assessed whether  
320 these parallel relationships were statistically related. For that purpose, we examined  
321 whether the beta weights describing the patterns of neural coupling were linked to  
322 variations in the level of “Detailed” thoughts reported by this cohort, either in terms of  
323 overall levels of thought, or in terms of how they were expressed in each N-Back task  
324 condition. We addressed this question by conducting a repeated ANCOVA in which the  
325 dependent variable was the PCA loading describing “Detailed” thoughts. The within  
326 participant factor was the task condition (i.e. 0/1-Back). The beta weights derived from both  
327 functional connectivity analyses, as well as the ADHD scores, were entered as between-  
328 participant variables. We also included depression, dyslexia and composite motion scores as  
329 covariates of no interest. In these analyses we modelled the main effects for each variable,  
330 as well as the two-way interactions between the DMN and FPN beta weights with the ADHD  
331 symptoms. This revealed a main effect of the FPN connectivity with respect to overall levels  
332 of Detail [ $F_{(1, 170)} = 7.03, p = .0088$ ] as well as an ADHD and FPN connectivity interaction [ $F_{(1, 170)} = 5.78, p = .017$ ]. This analysis suggests that FPN connectivity with the right ventral visual  
334 cortex was linked to more detailed thoughts [Pearson  $r = .34, p = .0015$ ] (Fig. 3a), and this  
335 association was present only for individuals that scored low on ADHD symptomatology,  
336 while no significant association was found for individuals that scored high on ADHD  
337 symptomatology [Pearson  $r = -.031, p = .78$ ] (Fig. 3b).

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## 342 **Discussion**

343 Our study set out to understand the relationship between individual variability in  
344 ADHD symptomology and patterns of ongoing thought in a neurotypical population,  
345 focusing on its link to the functional connectivity of two large-scale brain networks at rest –  
346 the frontoparietal and default mode networks (FPN and DMN, respectively). Our  
347 behavioural analysis demonstrated that ADHD symptoms were linked to the level of detail  
348 reported in the participants' patterns of ongoing thought during the more demanding 1-  
349 Back condition of the working memory task used in our study. In neural terms, we found  
350 that the intrinsic architecture of both the frontoparietal and default mode networks varied  
351 with ADHD symptomology, in both cases showing reduced correlation with regions in the  
352 unimodal sensorimotor cortices. In particular, higher scores on ADHD were linked to  
353 reduced correlation between the FPN and a region of the right ventral visual cortex, while  
354 the DMN showed reduced correlation with a region of the right motor cortex. Importantly,  
355 only the connectivity of the FPN was linked to changes in the level of detail in ongoing  
356 thought for individuals with generally low ADHD symptoms. Overall, our results are  
357 consistent with the hypothesis that ADHD may be linked to deficient adjustment of  
358 cognition in line with increasing demands imposed by the environment and that this may  
359 partly arise from dysfunctions in the intrinsic organisation of the brain at rest.

360 Behaviourally, ADHD symptomatology was linked to reduced detail in ongoing  
361 thought when participants were actively engaged in the rehearsal of information in working  
362 memory. As maintaining a detailed visual representation of task relevant stimuli is an  
363 integral part of the 1-Back condition of our task (Owen *et al.*, 2005), this pattern of data  
364 suggests that ADHD symptoms are linked to deficits in maintaining detailed task  
365 representations in working memory. Importantly, this association with ADHD was specific to  
366 the more difficult 1-Back task, a pattern consistent with difficulties in regulating ongoing  
367 cognition in line with the demands of a specific task context. Notably, in our data we found  
368 no evidence that problems in ADHD are associated with increased levels of off-task thinking,  
369 which is one common definition of mind-wandering (Christoff *et al.*, 2016). Together these  
370 observations suggest that ADHD may not simply be associated with excessively thinking  
371 about matters unrelated to the here and now, but also to problems associated with the  
372 maintenance of detailed cognitive representations of an ongoing task.



373 In neural terms, we found that FPN connectivity with visual cortex was reduced in  
374 participants with higher ADHD scores and this was associated with lower levels of detailed  
375 cognition. This result suggests that patterns of ongoing thought linked to ADHD are partly  
376 related to the intrinsic architecture of FPN connectivity. Such an interpretation is consistent  
377 with evidence showing that the FPN plays a general role across a variety of demanding  
378 cognitive tasks (Cole *et al.*, 2013, Duncan, 2010). We note, however, that the influence of  
379 this network on the changes of ongoing thought linked to ADHD symptoms might also  
380 depend on other variables. Behaviourally, the associations between ADHD scores and  
381 detailed thoughts were limited to the more difficult 1-Back task condition, while the  
382 interaction with the brain was related to lower levels of detail in general. It is possible that  
383 this discrepancy arises due to the influence of other variables, such as levels of motivation.  
384 In neurotypical individuals, ongoing thought tends to be more deliberately focused on the  
385 task when task demands are high and this effect is partly dependent on the individuals' level  
386 of motivation (Seli *et al.*, 2018). It is possible, therefore, that the variation in levels of  
387 motivation to focus on the task in the non-demanding 0-Back condition, and, in particular in  
388 individuals that score low in ADHD symptoms, may explain why neural processes linked to  
389 ADHD were related to lower levels of detail in general, rather than in a task specific manner.

390 Contemporary accounts of spontaneous thought have argued that individuals with  
391 ADHD are unable to suppress internally-oriented cognition that is supported by the DMN  
392 (Andrews-Hanna *et al.*, 2014, Christoff *et al.*, 2016). Our analysis using MDES found no  
393 evidence that ADHD was linked to greater off-task thought. Moreover, while high levels of  
394 ADHD were linked to low levels of connectivity between the DMN and motor cortex, unlike  
395 the neural activity in the FPN, this connection showed no relationship with changes in  
396 detailed thought that were associated with ADHD scores. These results suggest that instead  
397 of problems in suppressing internally-oriented cognition related to over activity within the  
398 DMN, experiential differences in ADHD may be, at least in part, mediated by problems in  
399 maintaining detailed task representations. As is made explicit in executive failure views of  
400 mind-wandering (McVay and Kane, 2009), the inability to sustain attention on task relevant  
401 information, could indirectly produce periods of elevated off-task thought since individuals  
402 would spend less time focused on the task in hand (Smallwood *et al.*, 2013a).

403 More generally, recent studies suggest that the DMN might carry out a role that  
404 extends beyond that of internally-oriented cognition (Vatansever *et al.*, 2018). For example,

405 recent work has demonstrated that the DMN can make an important contribution to  
406 externally-oriented tasks, especially when behaviour is guided by representations gained  
407 from memory (Konishi *et al.*, 2015, Murphy *et al.*, 2017, Vatansever *et al.*, 2016a, b,  
408 Vatansever *et al.*, 2015, Vatansever *et al.*, 2017). Thus, it is possible that the absence of a  
409 relationship between the DMN and patterns of ongoing thought linked to ADHD emerges  
410 because of the task in which we assessed ongoing cognition. Plausibly, this relationship may  
411 emerge more readily in the context of a task requiring greater DMN engagement such as  
412 reading (Regev *et al.*, 2018, Smallwood *et al.*, 2013a) or during unconstrained states of rest  
413 (Castellanos *et al.*, 2008).

414         Alternatively, it is possible that the role of the DMN in ongoing cognition is more  
415 transient and is therefore undetectable using our cross-sectional design in a neurotypical  
416 cohort. [Notably, however, in a recent online experience sampling study we were able to](#)  
417 [predict patterns of off-task thought in regions of attention and sensorimotor cortex \(Sormaz](#)  
418 [et al., 2018\) while connectivity between the ventral attention network with motor cortex](#)  
419 [predicted the ability to regulate the occurrence of off-task thought \(Turnbull et al., 2018\).](#)  
420 Future cognitive research, therefore, may be able to provide valuable empirical evidence on  
421 the brain basis of patterns of ongoing thought, by measuring neural function in individuals  
422 with ADHD concurrently with experience sampling. Such studies could help determine  
423 whether activity within the DMN, or other large-scale brain networks, varies with the level  
424 of ADHD symptoms during mind-wandering. Nonetheless, in the absence of new data, our  
425 study suggests that in the context of a working memory task, (i) ADHD related changes in  
426 ongoing thought are more parsimoniously explained by changes in the intrinsic architecture  
427 of the FPN, rather than the DMN, and (ii) do not reflect the inability to suppress off-task  
428 thought, but reflect problems in maintaining detailed task representations.

429         More generally, the results of both our functional connectivity analyses highlight  
430 changes in connectivity linked to ADHD that reflect reduced communication between  
431 regions of the transmodal cortex (DMN and FPN) with aspects of cortex linked to more  
432 specialised unimodal functions (visual and motor cortices). Current views of both ongoing  
433 thought (Baird *et al.*, 2014, Kam *et al.*, 2011, Seli, 2016, Smallwood *et al.*, 2008) and ADHD  
434 (Ghanizadeh, 2011) highlight patterns of sensorimotor decoupling as an important feature.  
435 Both of these literatures suggest that a general problem in ADHD may emerge from an  
436 exacerbation in the decoupling between transmodal and unimodal cortical regions. It is

437 important to note, however, that the process of sensorimotor decoupling is most effectively  
438 measured when indices of neural function are assessed online during task performance  
439 (Baird *et al.*, 2014). Nonetheless, it is intriguing that neural patterns associated with ADHD  
440 show patterns of connectivity that are consistent with a reduction in neural communication  
441 between aspects of unimodal cortex that support task performance in a direct manner (i.e.  
442 perception and action) and those that play a more general supervisory role. Future research  
443 into deficits linking ADHD and ongoing thought, may wish to explore the coupling between  
444 regions of unimodal and transmodal cortex online during task performance, perhaps using  
445 an electrophysiological neuroimaging method that is more suited to assessing momentary  
446 changes in the dynamics of neural function (Fox *et al.*, 2018, Vidaurre *et al.*, 2016).

447 We also consider the implications of our results for the occurrence and management  
448 of ADHD symptoms in the real world. Our study provides complementary neural and  
449 subjective markers that, if replicated within a clinical population, would provide an  
450 important metric for assessing the efficacy of both psychological and pharmacological  
451 interventions for individuals with this disorder. For example, psychological interventions,  
452 such as mindfulness training (Mitchell *et al.*, 2015), and drug interventions (Turner *et al.*,  
453 2005) have both shown promise in reducing ADHD symptomatology. Based on our results,  
454 studies combining experience sampling with measures of neural function may provide  
455 important insight into the specific neurocognitive changes that underlie the effectiveness of  
456 such interventions. In addition, given mounting evidence on the genetic basis of ADHD (Mick  
457 and Faraone, 2008, Pironti *et al.*, 2014), population studies that examine experiential and  
458 neural differences that emerge in this cohort may provide unique insight into the link  
459 between genes, behaviour and cognition.

460 There are a number of limitations that should be considered when interpreting the  
461 results of this study. We examined levels of ADHD symptomatology in a group of  
462 neurotypical, healthy undergraduate students, rather than in a clinical population. While it  
463 is reasonably common to examine differences in ADHD in the normal population as a  
464 proximal measure for a clinical population (van Dongen *et al.*, 2015), it is possible that some  
465 of the relationships we identified in our current study may vary in clinical populations for  
466 whom symptoms are likely to be more extreme. In addition, as outlined earlier, our study  
467 used a cross-sectional design in which differences in functional connectivity at rest was used  
468 to explain patterns in ongoing cognition measured outside of the scanner in a behavioural

469 laboratory. While this approach provides important evidence on how neural architecture  
470 can relate to the manner in which cognition unfolds during tasks, it is possible that certain  
471 aspects of the relationships described in our study would vary if neural function was  
472 measured during task performance. Such limitations notwithstanding, our study suggests  
473 that patterns of ADHD symptomatology are linked to problems in maintaining detailed  
474 representations during a working memory task and that this pattern is partially accounted  
475 for by associated changes in the coupling between regions of cortex important in  
476 demanding tasks and those linked to visual processing.

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478

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489

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491

492 **Ethical Standards:** The authors assert that all procedures contributing to this work comply  
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494 experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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731

732

733 **Tables and Figure Captions**

734

735 **Table 1.** Multidimensional Experience Sampling (MDES) questions that were presented  
 736 during the N-Back task. Participants rated their ongoing thoughts on a 4-point Likert scale  
 737 ranging from 0 to 1.

<b>Names</b>	<b>Questions</b>	<b>0</b>	<b>1</b>
Task	My thoughts were focused on the task I was performing.	Not at all	Completely
Future	My thoughts involved future events.	Not at all	Completely
Past	My thoughts involved past events.	Not at all	Completely
Self	My thoughts involved myself.	Not at all	Completely
Other	My thoughts involved other people.	Not at all	Completely
Emotion	The content of my thoughts was:	Negative	Positive
Words	My thoughts were in the form of words.	Not at all	Completely
Images	My thoughts were in the form of images.	Not at all	Completely
Evolving	My thoughts tended to evolve in a series of steps.	Not at all	Completely
Habit	This thought has recurrent themes similar to those I have had before.	Not at all	Completely
Detailed	My thoughts were detailed and specific.	Not at all	Completely
Vivid	My thoughts were vivid as if I was there.	Not at all	Completely
Deliberate	My thoughts were:	Spontaneous	Deliberate

738

739

740

741 **Figure 1. Thought sampling procedures and the association between individual variability**  
742 **in thought structures and ADHD symptomatology.** (a) A thought sampling procedure was  
743 employed during an N-Back paradigm, in which the participants altered between 0-Back (i.e.  
744 easy perceptual decisions) and 1-Back (i.e. more difficult, memory-based decisions)  
745 conditions (Konishi *et al.*, 2015). During the thought probes, participants had to rate their  
746 thoughts using a 4-point Likert Scale from 0 (not at all) to 1 (completely) based on a set of  
747 mind-wandering questions. (b) The participants' ratings were then decomposed into distinct  
748 dimensions of thought using principal component analysis (PCA) and Varimax rotation in  
749 order to achieve interpretable results. (c) Individual variation on the identified thought  
750 structures were used as explanatory variables in a linear regression assessing their relation  
751 to ADHD scores. Out of the four components, the difference in the participants' detailed  
752 thoughts between the 1-Back and 0-Back versions of the N-Back task was negatively related  
753 to ADHD scores.

754

755 **Figure 2. Association between differential brain connectivity patterns and ADHD**  
756 **symptomatology.** (a) Two binarized masks representing the frontoparietal (FPN) and default  
757 mode networks (DMN) from the Yeo 7-Network parcellation scheme were used as regions  
758 on interest (ROI) in seed-based functional connectivity analyses. (b) Group-level statistical  
759 maps were created that represent the functional connectivity patterns of the chosen FPN  
760 and DMN seeds. (c) Whole-brain linear regression analyses revealed that both FPN  
761 connectivity to the right lingual gyrus (visual cortex) and DMN connectivity to the right  
762 pre/post central gyrus (motor) were negatively related to the ADHD scores. All results were  
763 corrected for depression, dyslexia and the percentage of invalid scans due to motion, and  
764 the reported clusters were multiple comparison corrected using Family Wise Error (FWE)  
765 correction at the .05 significance level (0.001 uncorrected at the voxel level).

766

767 **Figure 3. The link between detailed thoughts and task context in individuals who scored**  
768 **low and high in ADHD scores.** The participants were first divided in to low and high ADHD  
769 groups based on the median scores on the ADHD scale. (a) Participants who scored low on  
770 the ADHD scale showed a significant relationship between overall detailed thoughts in both  
771 the 0-Back and 1-Back conditions of the N-Back task. In this group, greater connectivity  
772 between the FPN with the right ventral visual cortex correlated with greater detailed

773 thoughts reported across both conditions of the task ( $r = .34$ ,  $p = .0015$ ). (b) However, those  
774 who scored high on the ADHD scale did not show a significant relationship between detailed  
775 patterns of thought and FPN connectivity to the right ventral visual cortex ( $r = -.031$ ,  $p = .78$ ).  
776



1 **The Devil is in the Detail: Exploring the Intrinsic Neural Mechanisms that Link**  
2 **Attention-Deficit/Hyperactivity Disorder Symptomatology to Ongoing**  
3 **Cognition**

4  
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24 **Word Count:** 5,225

25 **Abstract**

26

27 **Background:** Attention-deficit/hyperactivity disorder (ADHD) is a developmental condition  
28 that profoundly affects quality of life. Although mounting evidence now suggests  
29 uncontrolled mind-wandering as a core aspect of the attentional problems associated with  
30 ADHD, the neural mechanisms underpinning this deficit remains unclear. To that extent,  
31 competing views argue for i) excessive generation of task-unrelated mental content, or ii)  
32 deficiency in the control of task-relevant cognition.

33

34 **Methods:** In a cross-sectional investigation of a large neurotypical cohort (n = 184), we  
35 examined alterations in the intrinsic brain functional connectivity architecture of the default  
36 mode (DMN) and frontoparietal (FPN) networks during resting state functional magnetic  
37 resonance imaging (rs-fMRI) in relation to ADHD symptomatology, which could potentially  
38 underlie changes in ongoing thought within variable environmental contexts.

39

40 **Results:** The results illustrated that ADHD symptoms were linked to lower levels of detail in  
41 ongoing thought while the participants made more difficult, memory-based decisions.  
42 Moreover, greater ADHD scores were associated with lower levels of connectivity between  
43 the DMN and right motor cortex, and between the FPN and right ventral visual cortex. Finally,  
44 a combination of high levels of ADHD symptomology with reduced FPN connectivity to the  
45 visual cortex was associated with reduced levels of detail in thought.

46

47 **Conclusions:** The results of our study suggest that the frequent mind-wandering observed in  
48 ADHD may be an indirect consequence of the deficient control of ongoing cognition in  
49 response to increasing environmental demands, and that this may partly arise from  
50 dysfunctions in the intrinsic organisation of the FPN at rest.

51

52 **Keywords:** attention-deficit/hyperactivity disorder, default mode network, frontoparietal  
53 network, functional connectivity, mind-wandering, ongoing thought.

54

55

## 56 Introduction

57 Attention-deficit/hyperactivity disorder (ADHD) is a childhood onset developmental  
58 disorder with profound psychosocial consequences (Barkley and Fischer, 2010, Kieling *et al.*,  
59 2010) that often persist into adulthood (Faraone, 2007). In addition to the observed deficits  
60 in cognitive performance (Banaschewski *et al.*, 2012, Kofler *et al.*, 2013, McLean *et al.*, 2004),  
61 it is commonly associated with a constellation of symptoms that include emotional lability  
62 (Skirrow *et al.*, 2009), dyslexia (Germano *et al.*, 2010) and mental health problems such as  
63 depression, anxiety, addiction and substance use disorders (Fayyad *et al.*, 2007).

64 One common feature of ADHD symptomatology is an elevated tendency for  
65 attentional lapses and reports of uncontrolled mind-wandering, i.e. periods when attention  
66 has shifted away from the current task goals. Both inside and outside the laboratory,  
67 individuals with ADHD characterise their mind-wandering experiences as excessively  
68 frequent, spontaneous and unintentional (Franklin *et al.*, 2014, Seli *et al.*, 2015), and describe  
69 their ongoing cognition as “thoughts that are constantly on the go, flitting from one topic to  
70 another, and multiple thoughts that appear at the same time” (Mowlem *et al.*, 2016).  
71 Although converging evidence highlights frequent mind-wandering as a core aspect of ADHD  
72 symptomatology, the neural mechanisms that underlie this deficit remain unclear.

73 Contemporary accounts suggest that mind-wandering is a heterogeneous state that is  
74 not the product of a single mental process, but rather one that emerges from a *component*  
75 *process architecture* in which certain aspects of mental experience are produced by the  
76 combination of specific elements of cognition (Seli *et al.*, 2018, Smallwood, 2013, Smallwood  
77 and Schooler, 2015). For example, during off-task thought, attention is often focused on  
78 mental content generated from internal memory stores. Consequently, individuals, who  
79 retrieve information from memory more efficiently, engage in more off-task thought (Poerio  
80 *et al.*, 2017, Smallwood *et al.*, 2011). One possibility, therefore, is that uncontrollable mind-  
81 wandering associated with ADHD symptomatology results from excessive tendencies to self-  
82 generate mental content from memory.

83 In addition to being beneficial for psychological functions that require creativity (Baird  
84 *et al.*, 2012) and planning (Medea *et al.*, 2016), such excessive generation of off-task thought  
85 can also have negative consequences, chiefly because it can lead to errors in task  
86 performance (Smallwood *et al.*, 2008). Accordingly, neurotypical individuals tend to reduce

87 off-task experiences and increase task-related thoughts when performing more attention  
88 demanding tasks - a process known as *context regulation* (Smallwood and Andrews-Hanna,  
89 2013) linked to executive control (Bernhardt *et al.*, 2014, Kane *et al.*, 2007, McVay and Kane,  
90 2009, Mrazek *et al.*, 2012, Smallwood *et al.*, 2013b). An alternative perspective, therefore, is  
91 that alterations in patterns of ongoing thought emerge in ADHD because of problems in  
92 implementing a form of controlled cognition that is appropriate to the specific task context.

93 In relation to these competing views, recent advances in functional neuroimaging  
94 have provided the opportunity to evaluate changes in cognition that is linked to ADHD from  
95 a mechanistic perspective. For example, the default mode network (DMN) has been shown  
96 to reduce its activity under demanding contexts (Mazoyer *et al.*, 2001, Shulman *et al.*, 1997),  
97 and to increase activity during lapses in attention (Eichele *et al.*, 2008). Individuals with ADHD,  
98 however, are reported to lack such task-evoked activity dynamics – a pattern often taken as  
99 evidence of excessive self-generation of mental contents (Liddle *et al.*, 2011). In parallel,  
100 deficits in executive control (Barkley, 1997), and the dysregulation of associated neural  
101 systems such as the frontoparietal network (FPN) (Cortese *et al.*, 2012), are both well-  
102 documented elements of ADHD.

103 Based on this evidence, the current study aimed to compare and contrast the role of  
104 excessive generation of off-task thoughts and impaired context regulation in deficits of  
105 ongoing thought with respect to ADHD symptomatology, and to understand whether  
106 perturbation in either the connectivity of the DMN or the FPN at rest underpin these  
107 problems. For that purpose, we recruited a set of neurotypical participants who completed  
108 (i) a battery of questionnaires, including a well-established measure of ADHD, (ii) a laboratory-  
109 based thought sampling method measuring ongoing cognition, and (iii) a resting state  
110 functional magnetic resonance imaging (rs-fMRI) scan, which provided a measure of intrinsic  
111 neural organisation. A critical element of our design was that the thought sampling method  
112 used a behavioural paradigm that alternated between conditions that encouraged  
113 participants to restrict their thoughts to task focused information, and those that were more  
114 conducive to off-task thoughts (Smallwood *et al.*, 2009, Teasdale *et al.*, 1993). This paradigm,  
115 therefore, provided the opportunity to index both context regulation (i.e. the ability to  
116 increase task-relevant cognition when a task is demanding) and self-generation (i.e. the  
117 amount of off-task thought produced throughout the task as a whole) accounts of mind-  
118 wandering, allowing us to compare these views in relation to ADHD symptomatology.

## 119 **Methods**

### 120 **Participants**

121 Ethical approval for this study was obtained from the Department of Psychology and  
122 York Neuroimaging Centre, University of York ethics committees. All participants gave  
123 informed consent prior to taking part in the experimental assessments. A total of 226 healthy,  
124 native English-speaker, right-handed participants were recruited subsequent to the study  
125 screening based on the following exclusion criteria: history of psychiatric or neurological  
126 illness, severe claustrophobia, anticipated pregnancy or drug use that could alter cognitive  
127 functioning. Out of this cohort, 184 participants fully completed the laboratory-based thought  
128 sampling and ADHD symptomatology questionnaire and were included in the initial analysis  
129 (mean = 20.13, SD = 2.24, range = 18-31, 121/63 female to male ratio).

130 Subsequently, all of these participants were scanned with a nine minutes long rs-fMRI  
131 during wakeful rest. A strict motion correction procedure (described in detail below) was  
132 utilised, which resulted in the further exclusion of nine participants, whereas three  
133 participants were removed due to problems associated with fMRI scanning. The average age  
134 for the final cohort of 172 participants suitable for the fMRI data analysis was 20.12 (SD =  
135 2.28, range = 18-31) with a 113/59 female to male ratio.

136

### 137 **Thought Sampling Method**

138 The participants' ongoing cognition was measured in a 30-minutes long behavioural  
139 paradigm that alternated between blocks of 0-Back and 1-Back conditions that manipulated  
140 working memory load (Fig. 1a). Non-target trials in both the 0-Back and 1-Back conditions  
141 were identical, consisting of black shapes (circles, squares or triangles) separated by a line,  
142 the colour of which signified whether the condition was 0-Back or 1-Back (mean presentation  
143 duration = 1050 ms, 200 ms jitter), counterbalanced across individuals. The non-target trials  
144 were followed by the presentation of a black fixation cross (mean presentation duration =  
145 1530 ms, 130 ms jitter), and presented in runs of between 2 and 8 trials with a mean of 5  
146 trials after which a target trial or a multidimensional experience sampling (MDES) probe was  
147 presented. In either the 0-Back or 1-Back non-target trials, participants were not required to  
148 make a behavioural response.

149 During the target trials, participants were required to make a response, which differed  
150 depending on the task condition. In the 0-Back condition, the target trial was a pair of  
151 coloured shapes presented on either side of a coloured line with a probe shape in the centre  
152 of the screen. Participants had to press a button to indicate whether the central shape  
153 matched the shape on the left or right-hand side of the screen. In this condition, there was  
154 no need to retain the details of the non-target trials since the response trials could be  
155 completed based on the information on the screen, releasing working memory from task  
156 relevant information (i.e. easy perceptual decisions).

157 In the 1-Back condition, the target trial consisted of two coloured question marks  
158 presented on either side of a coloured line with a probe shape in the centre of the screen.  
159 Participants had to indicate using a button press whether the central shape matched either  
160 the shape on the left or right side of the screen on the previous (non-target) trial. Thus, in this  
161 condition, participants had to maintain the visuo-spatial array in working memory for each  
162 trial and use this information appropriately in the target trials (i.e. more difficult, memory-  
163 based decisions). This task is presented schematically in Figure 1*a*.

164 The contents of ongoing thought during this N-Back task was measured using MDES.  
165 On each occasion that the participants were asked about their thoughts, they rated their  
166 answers to the 13 questions presented in Table 1 using a 4-point Likert scale that ranged from  
167 0 to 1. Participants always rated their level of task-focus first and then described their  
168 thoughts at the moment before the probe on a further 12 questions. MDES probes occurred  
169 on a quasi-random basis to minimise the likelihood that participants could anticipate the  
170 occurrence of a probe. At the moment of target presentation, there was 20% chance of a  
171 MDES probe instead of a target with a maximum of one probe per condition.

172 For the purpose of analyses, the ratings on the 13 MDES questions were decomposed  
173 into distinct patterns of thought that described the underlying structure of the participants  
174 responses. Following prior studies (Konishi *et al.*, 2017, Medea *et al.*, 2016, Ruby *et al.*, 2013*a*,  
175 Ruby *et al.*, 2013*b*, Smallwood *et al.*, 2016) we concatenated the responses of each  
176 participant at each probe and in each task into a single matrix and employed a principal  
177 component analysis (PCA) for factor reduction with Varimax rotation using SPSS (Version 23)  
178 (<https://www.ibm.com/products/spss-statistics>). We selected a total of four components  
179 based on the scree plot illustrated in Figure S1.

180

## 181 **ADHD Symptomatology Assessment**

182 With the aim of determining individual variability on the ADHD symptomatology of  
183 this neurotypical cohort, we administered the widely-used and validated Adult ADHD Self-  
184 Report Scale (ASRS-v1.1) (Kessler *et al.*, 2005, Kessler *et al.*, 2007). ASRS includes 18 questions  
185 that reflect the main criteria for a DSM-IV-TR based ADHD diagnosis. Previous research has  
186 indicated that six out of the 18 questions were most predictive of an ADHD diagnosis (Gray *et*  
187 *al.*, 2014, Kessler *et al.*, 2005, Kessler *et al.*, 2007), constituting the Part A of this scale. Average  
188 self-reported responses on this subscale of ASRS was thus utilised in our subsequent analyses  
189 aimed at investigating the link between ADHD symptomatology, ongoing thoughts and neural  
190 organisation at rest.

191 In addition, based on recent reports suggesting a close link between ADHD  
192 symptomatology, depression and dyslexia (Fayyad *et al.*, 2007, Germano *et al.*, 2010, Skirrow  
193 *et al.*, 2009), we have also employed measures of these co-morbid symptoms to be removed  
194 as nuisance variables in our analyses. For depression, we used the Center for Epidemiologic  
195 Studies Depression Scale (Radloff, 1977); whereas for dyslexia the Dyslexia Adult Checklist  
196 (DAC) was utilised (Smythe and Everatt, 2001). The correlation between these measures and  
197 ADHD scores are provided in the Supplementary Material (Fig. S2).

198

## 199 **MRI Data Acquisition**

200 All MRI data acquisition was carried out at the York Neuroimaging Centre, York with a  
201 3T GE HDx Excite MRI scanner using an eight-channel phased array head coil. Following a T1-  
202 weighted structural scan with 3D fast spoiled gradient echo (TR = 7.8 s, TE = minimum full, flip  
203 angle= 20°, matrix size = 256 x 256, 176 slices, voxel size = 1.13 x 1.13 x 1 mm<sup>3</sup>), a nine-minute  
204 resting state fMRI scan was carried out using single-shot 2D gradient-echo-planar imaging.  
205 The parameters for this sequence were as follows: TR = 3000 ms, TE = minimum full, flip angle  
206 = 90°, matrix size = 64 x 64, 60 slices, voxel size = 3 x 3 x 3 mm<sup>3</sup>, 180 volumes. During resting  
207 state scanning, the participants were asked to focus on a fixation cross in the middle of the  
208 screen.

209

## 210 **MRI Data Preprocessing**

211 All preprocessing steps for the MRI data were carried out using the SPM software  
212 package (Version 12.0) (<http://www.fil.ion.ucl.ac.uk/spm/>) based on the MATLAB platform

213 (Version 16.a) (<https://uk.mathworks.com/products/matlab.html>). After removing the first  
214 three functional volumes to account for the magnetisation equilibrium, the remaining data  
215 was first corrected for motion using six degrees of freedom (x, y, z translations and rotations),  
216 and adjusted for differences in slice-time. Subsequently, the high-resolution structural image  
217 was co-registered to the mean functional image via rigid-body transformation, segmented  
218 into grey/white matter and cerebrospinal fluid probability maps, and were spatially  
219 normalized to the Montreal Neurological Institute (MNI) space alongside with all functional  
220 volumes using the segmented images and *a priori* templates. This indirect procedure utilizes  
221 the unified segmentation–normalization framework, which combines tissue segmentation,  
222 bias correction, and spatial normalization in a single unified model (Ashburner and Friston,  
223 2005). Finally, all the functional images were smoothed using an 8 mm full width at half  
224 maximum (FWHM) Gaussian kernel.

225

## 226 **Functional Connectivity Analysis**

227 MRI data denoising procedures and the subsequent seed-based functional  
228 connectivity analyses were carried out using the *Conn* functional connectivity toolbox  
229 (Version 17.f) (<https://www.nitrc.org/projects/conn>) (Whitfield-Gabrieli and Nieto-Castanon,  
230 2012). With the goal of ensuring that motion and other artefacts did not confound our data,  
231 we first employed an extensive motion-correction procedure and denoising steps,  
232 comparable to those reported in the literature (Ciric *et al.*, 2017). In addition to the removal  
233 of six realignment parameters and their second-order derivatives using the general linear  
234 model (GLM) (Friston *et al.*, 1996), a linear detrending term was applied as well as the  
235 CompCor method that removed five principal components of the signal from white matter  
236 and cerebrospinal fluid (Behzadi *et al.*, 2007). Moreover, the volumes affected by motion  
237 were identified and scrubbed based on the conservative settings of motion greater than 0.5  
238 mm and global signal change larger than  $z = 3$ . A total of nine participants, who had more than  
239 15% of their data affected by motion was excluded from the analysis (Power *et al.*, 2014). The  
240 distribution of average and maximum framewise displacement and global blood oxygen level  
241 dependent (BOLD) signal change, as well as the percentage of invalid scans in the final cohort  
242 utilised in this study are provided in Figure S3. Though recent reports suggest the ability of  
243 global signal regression to account for head motion, it is also known to introduce spurious  
244 anti-correlations, and thus was not utilised in our analysis (Saad *et al.*, 2012). Finally, a band-



245 pass filter between 0.009 Hz and 0.08 Hz was employed in order to focus on low frequency  
246 fluctuations (Fox *et al.*, 2005).

247 Following this procedure, we performed two separate seed-based functional  
248 connectivity analyses based on two regions of interest (ROIs) that were selected from the Yeo  
249 7-Network parcellation scheme (Yeo *et al.*, 2011), namely the frontoparietal and default  
250 mode networks. For each participant, average BOLD signal from the binarised seed ROIs  
251 described above were correlated with time courses from the rest of the brain with the aim of  
252 obtaining individual connectivity maps. Group-level inferences on positive and negative  
253 connectivity of the chosen seed ROIs were made based on one-sample t-tests. Further linear  
254 regressions with FPN as well as DMN connectivity were performed with ADHD  
255 symptomatology as the variable of interest, while correcting for dyslexia, depression and the  
256 percentage of invalid scans based on the motion scrubbing procedure. All reported clusters  
257 were corrected for multiple comparisons using the Family-Wise Error (FWE) detection  
258 technique at the .05 level of significance (uncorrected at the voxel-level, .001 level of  
259 significance). Beta values representing connectivity of the clusters and the chosen seed ROIs  
260 that significantly explained individual variability in ADHD symptomatology, were then  
261 extracted for each participant for subsequent statistical analyses.

262

### 263 **Statistical Analysis**

264 We performed three main analyses to test the relationships between ADHD  
265 symptomatology, patterns of ongoing thought and their potential neural mechanisms. First,  
266 using a mixed Analysis of Variance (ANOVA) we examined the relationship between patterns  
267 of ongoing thought in the two tasks and variation in ADHD symptomatology with the aim of  
268 determining if their relationships support either the excessive self-generation, or the  
269 impaired context regulation accounts of ADHD, while correcting for depression and dyslexia.  
270 Second, we used linear regressions in seed-based functional connectivity analyses to identify  
271 how the intrinsic neural organisation varies with natural variation in ADHD symptomatology.  
272 For this, we included co-morbid depression, dyslexia scores and subject motion inside the  
273 scanner as nuisance variables. Finally, we examined whether patterns of shared variance in  
274 association between patterns of neural function and ongoing thought linked to ADHD using  
275 connectivity values (beta weights) obtained from the seed-based analysis and component  
276 scores from thought sampling during specific task contexts. In this analysis, we repeated the

277 mixed ANOVA from the first step of our analysis, additionally including the neural changes  
278 identified through our functional connectivity analysis as covariates. This last step allowed us  
279 to identify potential neural mechanisms that underpin ADHD related changes in patterns of  
280 ongoing thought.

281

282

## 283 Results

284 Our first analysis examined the relationship between ADHD and patterns of ongoing  
285 thought recorded in the laboratory session (Fig. 1a). Following a decomposition of the  
286 thought sampling data (Fig. 1b) we conducted a series of repeated measure ANCOVAs. In  
287 these models, while the dependent measure was the scores for each component of thought,  
288 the within participant factor was the task context (0-Back/1-Back) and the between  
289 participants factor was ADHD scores (correcting for depression and dyslexia). These analyses  
290 first revealed three components of thought that varied across the task conditions: “Detailed”  
291 ( $F_{(1,182)} = 9.24, p = .0027$ ), “Off-Task” ( $F_{(1,182)} = 4.98, p = .027$ ), and “Modality-Specific  
292 (Images/Words)” ( $F_{(1,182)} = 5.27, p = .023$ ) thoughts. “Emotion+” did not vary across the task  
293 conditions. In the 1-Back, thoughts were more detailed ( $M = .11, 95\% \text{ CI } [-.208, .002]$ ) than  
294 in the 0-Back condition ( $M = -.07, 95\% \text{ CI } [.028, -.17]$ ). Off-Task thoughts were more prominent  
295 in the 0-Back ( $M = .14, 95\% \text{ CI } [.237, .04]$ ) than in the 1-Back condition ( $M = -.15, 95\% \text{ CI } [-$   
296  $.057, -.246]$ ). Finally, thoughts were less in the form of words in the 1-Back ( $M = -.06, 95\% \text{ CI } [-$   
297  $.037, -.175]$ ) than in the 0-Back condition ( $M = .07, 95\% \text{ CI } [.170, .06]$ ).

298 We also identified an ADHD by N-Back task condition interaction for the “Detailed”  
299 component ( $F_{(1,182)} = 6.82, p = .0098$ ) of the reported thoughts. This interaction indicated that  
300 greater ADHD scores were linked to a smaller difference in the level of thought details  
301 reported in the 1-Back than the 0-Back task condition [Pearson  $r = -.19, p = .0046$ ] (Fig. 1c).  
302 Increasing levels of ADHD, therefore, were associated with reports of less detailed  
303 experiences in the more demanding 1-Back condition.

304 Our next analysis explored the association between brain functional connectivity at  
305 rest and levels of ADHD symptomology within our sample. After generating spatial maps for  
306 each individual that described the associations at the whole brain level for each of the two  
307 networks that formed the focus of our investigation (i.e. FPN and DMN) (Fig. 2a-b), we  
308 conducted two group level regressions. In these analyses we included mean centred ADHD  
309 scores as a between participant variable of interest, while controlling for potential confounds  
310 such as depression, dyslexia and the percentage of motion-based invalid scans.

311 These analyses revealed two differences. Higher ADHD scores were linked to reduced  
312 correlation between the FPN and a region of right lingual gyrus (visual cortex). In addition,  
313 higher ADHD scores were associated with reduced correlation between the DMN and a region

314 of right pre/post central gyrus (motor cortex) (Fig. 2c). Increasing levels of ADHD within our  
315 sample, therefore, were linked to reduced correlation between transmodal association  
316 cortices (DMN, FPN) and unimodal sensorimotor cortices.

317 Thus far we have identified the correlates of ADHD symptomology with both patterns  
318 of ongoing thought and neural organisation. Our final analyses assessed whether these  
319 parallel relationships were statistically related. For that purpose, we examined whether the  
320 beta weights describing the patterns of neural coupling were linked to variations in the level  
321 of “Detailed” thoughts reported by this cohort, either in terms of overall levels of thought, or  
322 in terms of how they were expressed in each N-Back task condition. We addressed this  
323 question by conducting a repeated ANCOVA in which the dependent variable was the PCA  
324 loading describing “Detailed” thoughts. The within participant factor was the task condition  
325 (i.e. 0/1-Back). The beta weights derived from both functional connectivity analyses, as well  
326 as the ADHD scores, were entered as between-participant variables. We also included  
327 depression, dyslexia and composite motion scores as covariates of no interest. In these  
328 analyses we modelled the main effects for each variable, as well as the two-way interactions  
329 between the DMN and FPN beta weights with the ADHD symptoms. This revealed a main  
330 effect of the FPN connectivity with respect to overall levels of Detail [ $F_{(1, 170)} = 7.03, p = .0088$ ]  
331 as well as an ADHD and FPN connectivity interaction [ $F_{(1, 170)} = 5.78, p = .017$ ]. This analysis  
332 suggests that FPN connectivity with the right ventral visual cortex was linked to more detailed  
333 thoughts [Pearson  $r = .34, p = .0015$ ] (Fig. 3a), and this association was present only for  
334 individuals that scored low on ADHD symptomatology, while no significant association was  
335 found for individuals that scored high on ADHD symptomatology [Pearson  $r = -.031, p = .78$ ]  
336 (Fig. 3b).

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## 341 **Discussion**

342           Our study set out to understand the relationship between individual variability in  
343 ADHD symptomology and patterns of ongoing thought in a neurotypical population, focusing  
344 on its link to the functional connectivity of two large-scale brain networks at rest – the  
345 frontoparietal and default mode networks (FPN and DMN, respectively). Our behavioural  
346 analysis demonstrated that ADHD symptoms were linked to the level of detail reported in the  
347 participants' patterns of ongoing thought during the more demanding 1-Back condition of the  
348 working memory task used in our study. In neural terms, we found that the intrinsic  
349 architecture of both the frontoparietal and default mode networks varied with ADHD  
350 symptomology, in both cases showing reduced correlation with regions in the unimodal  
351 sensorimotor cortices. In particular, higher scores on ADHD were linked to reduced  
352 correlation between the FPN and a region of the right ventral visual cortex, while the DMN  
353 showed reduced correlation with a region of the right motor cortex. Importantly, only the  
354 connectivity of the FPN was linked to changes in the level of detail in ongoing thought for  
355 individuals with generally low ADHD symptoms. Overall, our results are consistent with the  
356 hypothesis that ADHD may be linked to deficient adjustment of cognition in line with  
357 increasing demands imposed by the environment and that this may partly arise from  
358 dysfunctions in the intrinsic organisation of the brain at rest.

359           Behaviourally, ADHD symptomatology was linked to reduced detail in ongoing thought  
360 when participants were actively engaged in the rehearsal of information in working memory.  
361 As maintaining a detailed visual representation of task relevant stimuli is an integral part of  
362 the 1-Back condition of our task (Owen *et al.*, 2005), this pattern of data suggests that ADHD  
363 symptoms are linked to deficits in maintaining detailed task representations in working  
364 memory. Importantly, this association with ADHD was specific to the more difficult 1-Back  
365 task, a pattern consistent with difficulties in regulating ongoing cognition in line with the  
366 demands of a specific task context. Notably, in our data we found no evidence that problems  
367 in ADHD are associated with increased levels of off-task thinking, which is one common  
368 definition of mind-wandering (Christoff *et al.*, 2016). Together these observations suggest  
369 that ADHD may not simply be associated with excessively thinking about matters unrelated  
370 to the here and now, but also to problems associated with the maintenance of detailed  
371 cognitive representations of an ongoing task.

372 In neural terms, we found that FPN connectivity with visual cortex was reduced in  
373 participants with higher ADHD scores and this was associated with lower levels of detailed  
374 cognition. This result suggests that patterns of ongoing thought linked to ADHD are partly  
375 related to the intrinsic architecture of FPN connectivity. Such an interpretation is consistent  
376 with evidence showing that the FPN plays a general role across a variety of demanding  
377 cognitive tasks (Cole *et al.*, 2013, Duncan, 2010). We note, however, that the influence of this  
378 network on the changes of ongoing thought linked to ADHD symptoms might also depend on  
379 other variables. Behaviourally, the associations between ADHD scores and detailed thoughts  
380 were limited to the more difficult 1-Back task condition, while the interaction with the brain  
381 was related to lower levels of detail in general. It is possible that this discrepancy arises due  
382 to the influence of other variables, such as levels of motivation. In neurotypical individuals,  
383 ongoing thought tends to be more deliberately focused on the task when task demands are  
384 high and this effect is partly dependent on the individuals' level of motivation (Seli *et al.*,  
385 2018). It is possible, therefore, that the variation in levels of motivation to focus on the task  
386 in the non-demanding 0-Back condition, and, in particular in individuals that score low in  
387 ADHD symptoms, may explain why neural processes linked to ADHD were related to lower  
388 levels of detail in general, rather than in a task specific manner.

389 Contemporary accounts of spontaneous thought have argued that individuals with  
390 ADHD are unable to suppress internally-oriented cognition that is supported by the DMN  
391 (Andrews-Hanna *et al.*, 2014, Christoff *et al.*, 2016). Our analysis using MDES found no  
392 evidence that ADHD was linked to greater off-task thought. Moreover, while high levels of  
393 ADHD were linked to low levels of connectivity between the DMN and motor cortex, unlike  
394 the neural activity in the FPN, this connection showed no relationship with changes in detailed  
395 thought that were associated with ADHD scores. These results suggest that instead of  
396 problems in suppressing internally-oriented cognition related to over activity within the DMN,  
397 experiential differences in ADHD may be, at least in part, mediated by problems in  
398 maintaining detailed task representations. As is made explicit in executive failure views of  
399 mind-wandering (McVay and Kane, 2009), the inability to sustain attention on task relevant  
400 information, could indirectly produce periods of elevated off-task thought since individuals  
401 would spend less time focused on the task in hand (Smallwood *et al.*, 2013a).

402 More generally, recent studies suggest that the DMN might carry out a role that  
403 extends beyond that of internally-oriented cognition (Vatansever *et al.*, 2018). For example,

404 recent work has demonstrated that the DMN can make an important contribution to  
405 externally-oriented tasks, especially when behaviour is guided by representations gained  
406 from memory (Konishi *et al.*, 2015, Murphy *et al.*, 2017, Vatansever *et al.*, 2016a, b,  
407 Vatansever *et al.*, 2015, Vatansever *et al.*, 2017). Thus, it is possible that the absence of a  
408 relationship between the DMN and patterns of ongoing thought linked to ADHD emerges  
409 because of the task in which we assessed ongoing cognition. Plausibly, this relationship may  
410 emerge more readily in the context of a task requiring greater DMN engagement such as  
411 reading (Regev *et al.*, 2018, Smallwood *et al.*, 2013a) or during unconstrained states of rest  
412 (Castellanos *et al.*, 2008).

413         Alternatively, it is possible that the role of the DMN in ongoing cognition is more  
414 transient and is therefore undetectable using our cross-sectional design in a neurotypical  
415 cohort. Notably, however, in a recent online experience sampling study we were able to  
416 predict patterns of off-task thought in regions of attention and sensorimotor cortex (Sormaz  
417 *et al.*, 2018) while connectivity between the ventral attention network with motor cortex  
418 predicted the ability to regulate the occurrence of off-task thought (Turnbull *et al.*, 2018).  
419 Future cognitive research, therefore, may be able to provide valuable empirical evidence on  
420 the brain basis of patterns of ongoing thought, by measuring neural function in individuals  
421 with ADHD concurrently with experience sampling. Such studies could help determine  
422 whether activity within the DMN, or other large-scale brain networks, varies with the level of  
423 ADHD symptoms during mind-wandering. Nonetheless, in the absence of new data, our study  
424 suggests that in the context of a working memory task, (i) ADHD related changes in ongoing  
425 thought are more parsimoniously explained by changes in the intrinsic architecture of the  
426 FPN, rather than the DMN, and (ii) do not reflect the inability to suppress off-task thought,  
427 but reflect problems in maintaining detailed task representations.

428         More generally, the results of both our functional connectivity analyses highlight  
429 changes in connectivity linked to ADHD that reflect reduced communication between regions  
430 of the transmodal cortex (DMN and FPN) with aspects of cortex linked to more specialised  
431 unimodal functions (visual and motor cortices). Current views of both ongoing thought (Baird  
432 *et al.*, 2014, Kam *et al.*, 2011, Seli, 2016, Smallwood *et al.*, 2008) and ADHD (Ghanizadeh,  
433 2011) highlight patterns of sensorimotor decoupling as an important feature. Both of these  
434 literatures suggest that a general problem in ADHD may emerge from an exacerbation in the  
435 decoupling between transmodal and unimodal cortical regions. It is important to note,

436 however, that the process of sensorimotor decoupling is most effectively measured when  
437 indices of neural function are assessed online during task performance (Baird *et al.*, 2014).  
438 Nonetheless, it is intriguing that neural patterns associated with ADHD show patterns of  
439 connectivity that are consistent with a reduction in neural communication between aspects  
440 of unimodal cortex that support task performance in a direct manner (i.e. perception and  
441 action) and those that play a more general supervisory role. Future research into deficits  
442 linking ADHD and ongoing thought, may wish to explore the coupling between regions of  
443 unimodal and transmodal cortex online during task performance, perhaps using an  
444 electrophysiological neuroimaging method that is more suited to assessing momentary  
445 changes in the dynamics of neural function (Fox *et al.*, 2018, Vidaurre *et al.*, 2016).

446 We also consider the implications of our results for the occurrence and management  
447 of ADHD symptoms in the real world. Our study provides complementary neural and  
448 subjective markers that, if replicated within a clinical population, would provide an important  
449 metric for assessing the efficacy of both psychological and pharmacological interventions for  
450 individuals with this disorder. For example, psychological interventions, such as mindfulness  
451 training (Mitchell *et al.*, 2015), and drug interventions (Turner *et al.*, 2005) have both shown  
452 promise in reducing ADHD symptomatology. Based on our results, studies combining  
453 experience sampling with measures of neural function may provide important insight into the  
454 specific neurocognitive changes that underlie the effectiveness of such interventions. In  
455 addition, given mounting evidence on the genetic basis of ADHD (Mick and Faraone, 2008,  
456 Pironti *et al.*, 2014), population studies that examine experiential and neural differences that  
457 emerge in this cohort may provide unique insight into the link between genes, behaviour and  
458 cognition.

459 There are a number of limitations that should be considered when interpreting the  
460 results of this study. We examined levels of ADHD symptomatology in a group of neurotypical,  
461 healthy undergraduate students, rather than in a clinical population. While it is reasonably  
462 common to examine differences in ADHD in the normal population as a proximal measure for  
463 a clinical population (van Dongen *et al.*, 2015), it is possible that some of the relationships we  
464 identified in our current study may vary in clinical populations for whom symptoms are likely  
465 to be more extreme. In addition, as outlined earlier, our study used a cross-sectional design  
466 in which differences in functional connectivity at rest was used to explain patterns in ongoing  
467 cognition measured outside of the scanner in a behavioural laboratory. While this approach



468 provides important evidence on how neural architecture can relate to the manner in which  
469 cognition unfolds during tasks, it is possible that certain aspects of the relationships described  
470 in our study would vary if neural function was measured during task performance. Such  
471 limitations notwithstanding, our study suggests that patterns of ADHD symptomatology are  
472 linked to problems in maintaining detailed representations during a working memory task  
473 and that this pattern is partially accounted for by associated changes in the coupling between  
474 regions of cortex important in demanding tasks and those linked to visual processing.

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487

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489

490 **Ethical Standards:** The authors assert that all procedures contributing to this work comply  
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492 experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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723

724

725 **Tables and Figure Captions**

726

727 **Table 1.** Multidimensional Experience Sampling (MDES) questions that were presented during  
 728 the N-Back task. Participants rated their ongoing thoughts on a 4-point Likert scale ranging  
 729 from 0 to 1.

<b>Names</b>	<b>Questions</b>	<b>0</b>	<b>1</b>
Task	My thoughts were focused on the task I was performing.	Not at all	Completely
Future	My thoughts involved future events.	Not at all	Completely
Past	My thoughts involved past events.	Not at all	Completely
Self	My thoughts involved myself.	Not at all	Completely
Other	My thoughts involved other people.	Not at all	Completely
Emotion	The content of my thoughts was:	Negative	Positive
Words	My thoughts were in the form of words.	Not at all	Completely
Images	My thoughts were in the form of images.	Not at all	Completely
Evolving	My thoughts tended to evolve in a series of steps.	Not at all	Completely
Habit	This thought has recurrent themes similar to those I have had before.	Not at all	Completely
Detailed	My thoughts were detailed and specific.	Not at all	Completely
Vivid	My thoughts were vivid as if I was there.	Not at all	Completely
Deliberate	My thoughts were:	Spontaneous	Deliberate

730

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732

733 **Figure 1. Thought sampling procedures and the association between individual variability**  
734 **in thought structures and ADHD symptomatology.** (a) A thought sampling procedure was  
735 employed during an N-Back paradigm, in which the participants altered between 0-Back (i.e.  
736 easy perceptual decisions) and 1-Back (i.e. more difficult, memory-based decisions)  
737 conditions (Konishi *et al.*, 2015). During the thought probes, participants had to rate their  
738 thoughts using a 4-point Likert Scale from 0 (not at all) to 1 (completely) based on a set of  
739 mind-wandering questions. (b) The participants' ratings were then decomposed into distinct  
740 dimensions of thought using principal component analysis (PCA) and Varimax rotation in  
741 order to achieve interpretable results. (c) Individual variation on the identified thought  
742 structures were used as explanatory variables in a linear regression assessing their relation to  
743 ADHD scores. Out of the four components, the difference in the participants' detailed  
744 thoughts between the 1-Back and 0-Back versions of the N-Back task was negatively related  
745 to ADHD scores.

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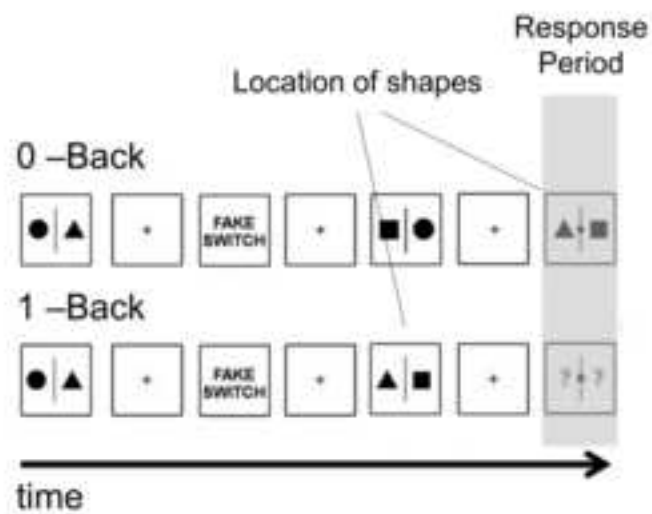
747 **Figure 2. Association between differential brain connectivity patterns and ADHD**  
748 **symptomatology.** (a) Two binarized masks representing the frontoparietal (FPN) and default  
749 mode networks (DMN) from the Yeo 7-Network parcellation scheme were used as regions on  
750 interest (ROI) in seed-based functional connectivity analyses. (b) Group-level statistical maps  
751 were created that represent the functional connectivity patterns of the chosen FPN and DMN  
752 seeds. (c) Whole-brain linear regression analyses revealed that both FPN connectivity to the  
753 right lingual gyrus (visual cortex) and DMN connectivity to the right pre/post central gyrus  
754 (motor) were negatively related to the ADHD scores. All results were corrected for  
755 depression, dyslexia and the percentage of invalid scans due to motion, and the reported  
756 clusters were multiple comparison corrected using Family Wise Error (FWE) correction at the  
757 .05 significance level (0.001 uncorrected at the voxel level).

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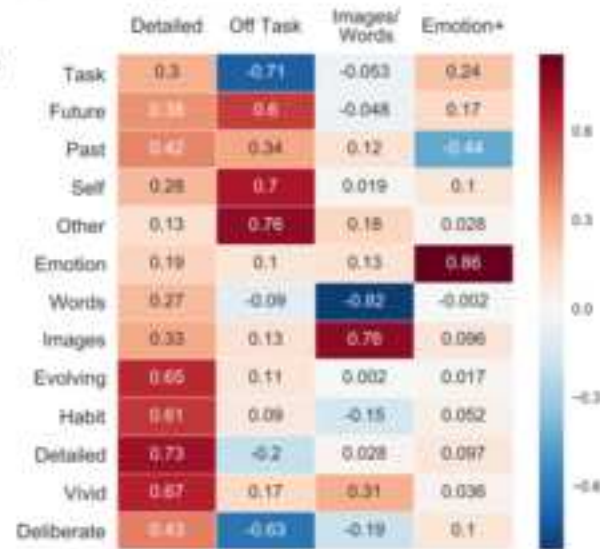
759 **Figure 3. The link between detailed thoughts and task context in individuals who scored low**  
760 **and high in ADHD scores.** The participants were first divided in to low and high ADHD groups  
761 based on the median scores on the ADHD scale. (a) Participants who scored low on the ADHD  
762 scale showed a significant relationship between overall detailed thoughts in both the 0-Back  
763 and 1-Back conditions of the N-Back task. In this group, greater connectivity between the FPN  
764 with the right ventral visual cortex correlated with greater detailed thoughts reported across

765 both conditions of the task ( $r = .34$ ,  $p = .0015$ ). (*b*) However, those who scored high on the  
766 ADHD scale did not show a significant relationship between detailed patterns of thought and  
767 FPN connectivity to the right ventral visual cortex ( $r = -.031$ ,  $p = .78$ ).  
768

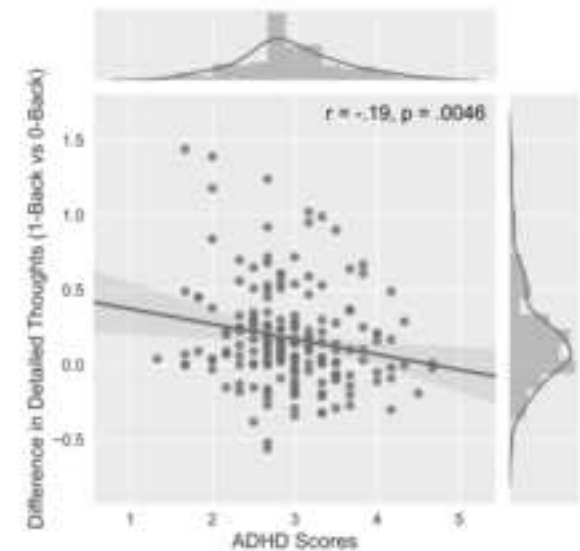
(a) Thought Sampling



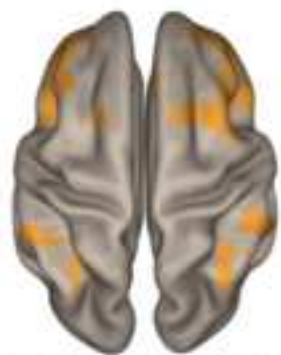
(b) Thought Structures



(c) Association of Thoughts with ADHD



**(a) Seed Region of Interest**

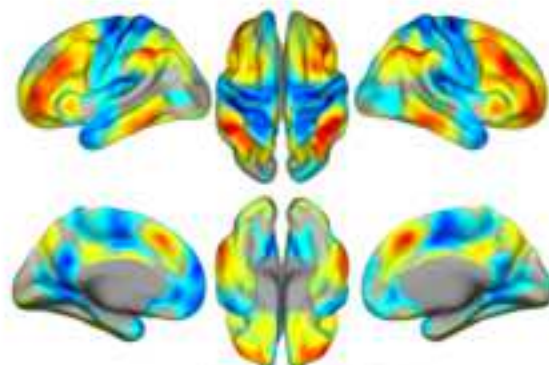


Yeo7 (FPN) Parcellation

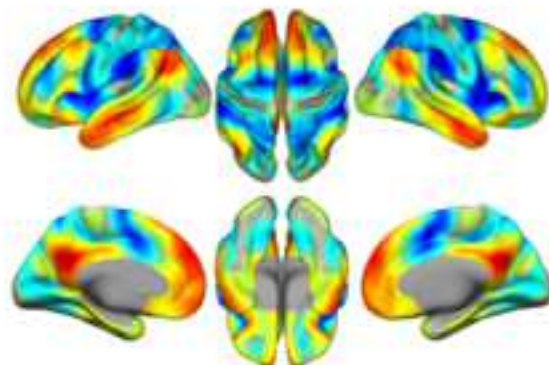


Yeo7 (DMN) Parcellation

**(b) Functional Connectivity**



54.56 T-score -22.54

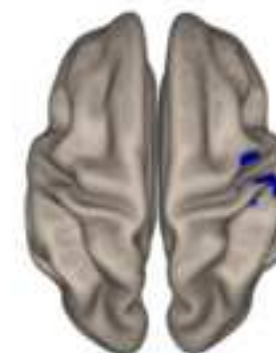


65.69 T-score -30.57

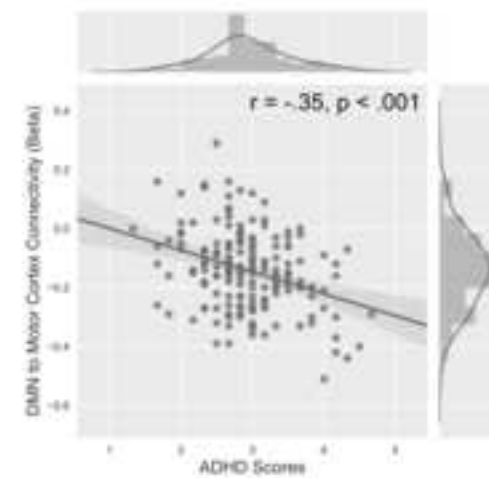
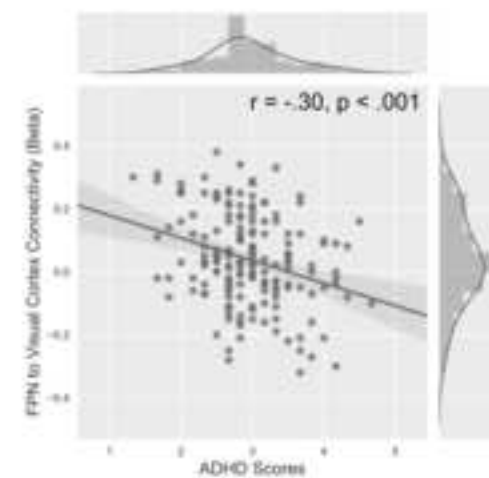
**(c) Association with ADHD Scores**

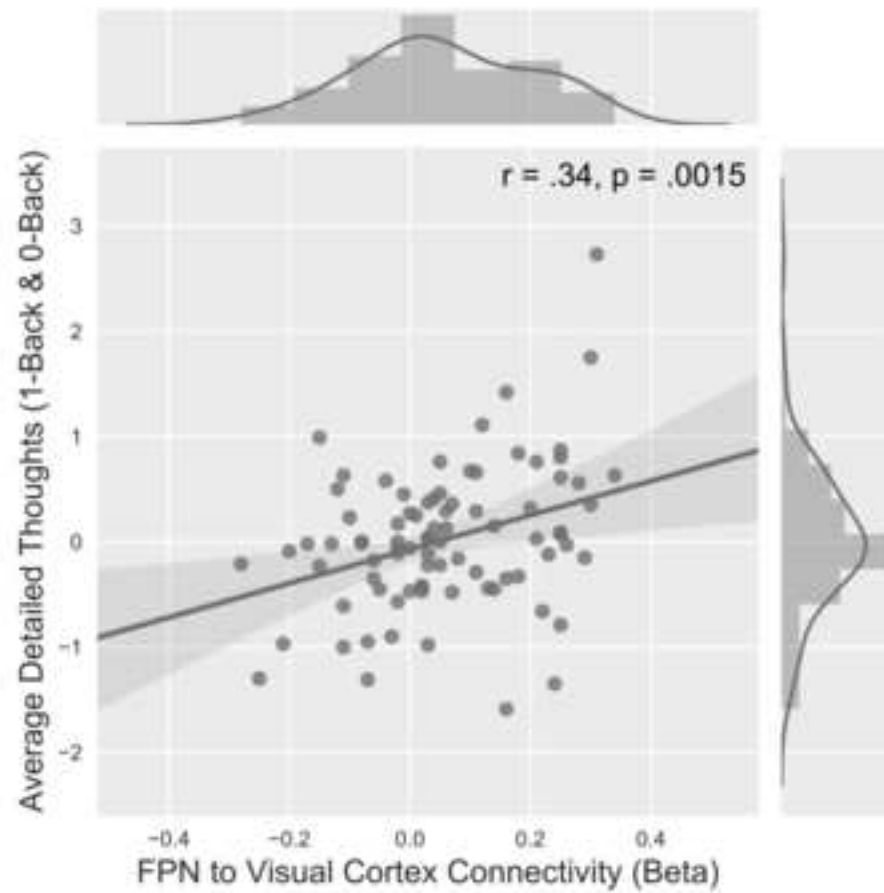
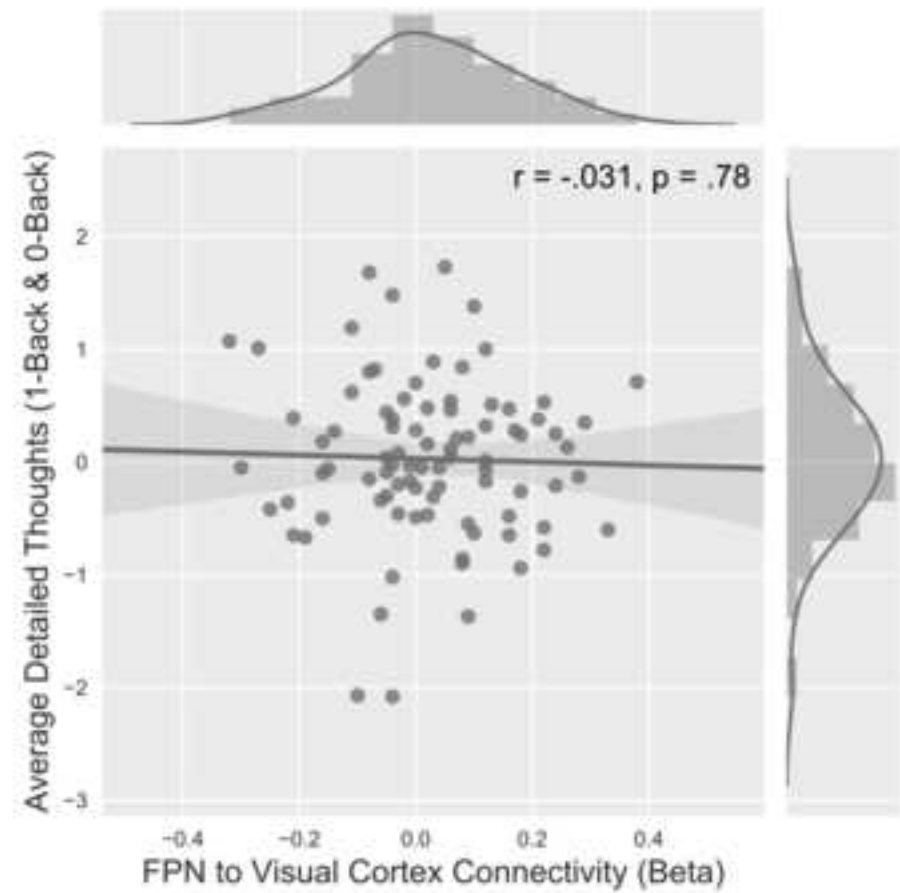


0 T-score -3.59



0 T-score -3.80



**(a) Low ADHD Scores****(b) High ADHD Scores**



## Supplementary Material: The Devil is in the Detail: Exploring the Intrinsic Neural Mechanisms that Link Attention-Deficit/Hyperactivity Disorder Symptomatology to Ongoing Cognition

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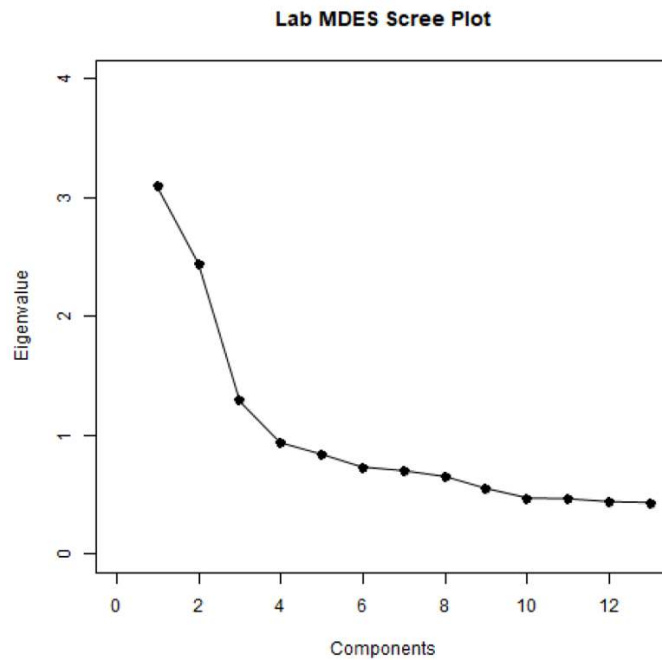
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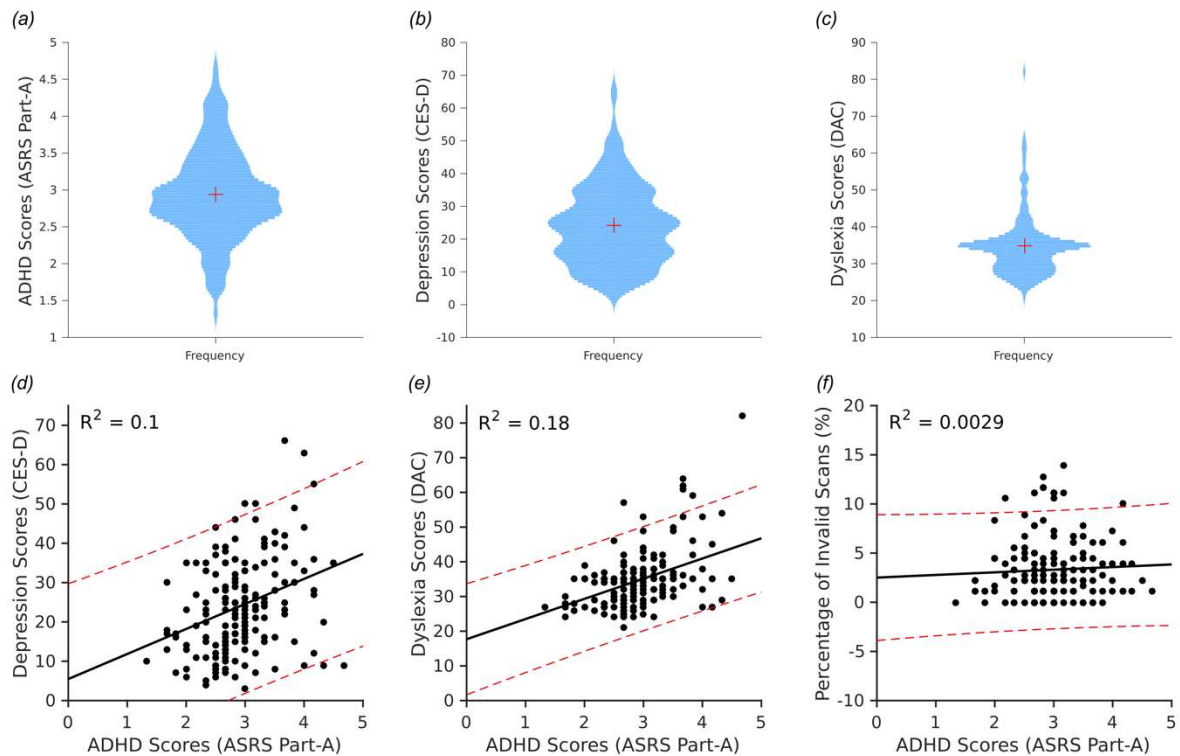
## Supplementary Results and Figures

### Thought Sampling Method



**Supplementary Figure 1. Principal component analysis of the thought sampling ratings.** The participants' ratings for each of the 13 Multidimensional Experience Sampling (MDES) questions were decomposed into four patterns of thought using principal component analysis (PCA). The number of components was chosen based on the scree plot for each PCA, indicating the eigenvalue of each subsequent decomposition and its ability to explain variability in the data.

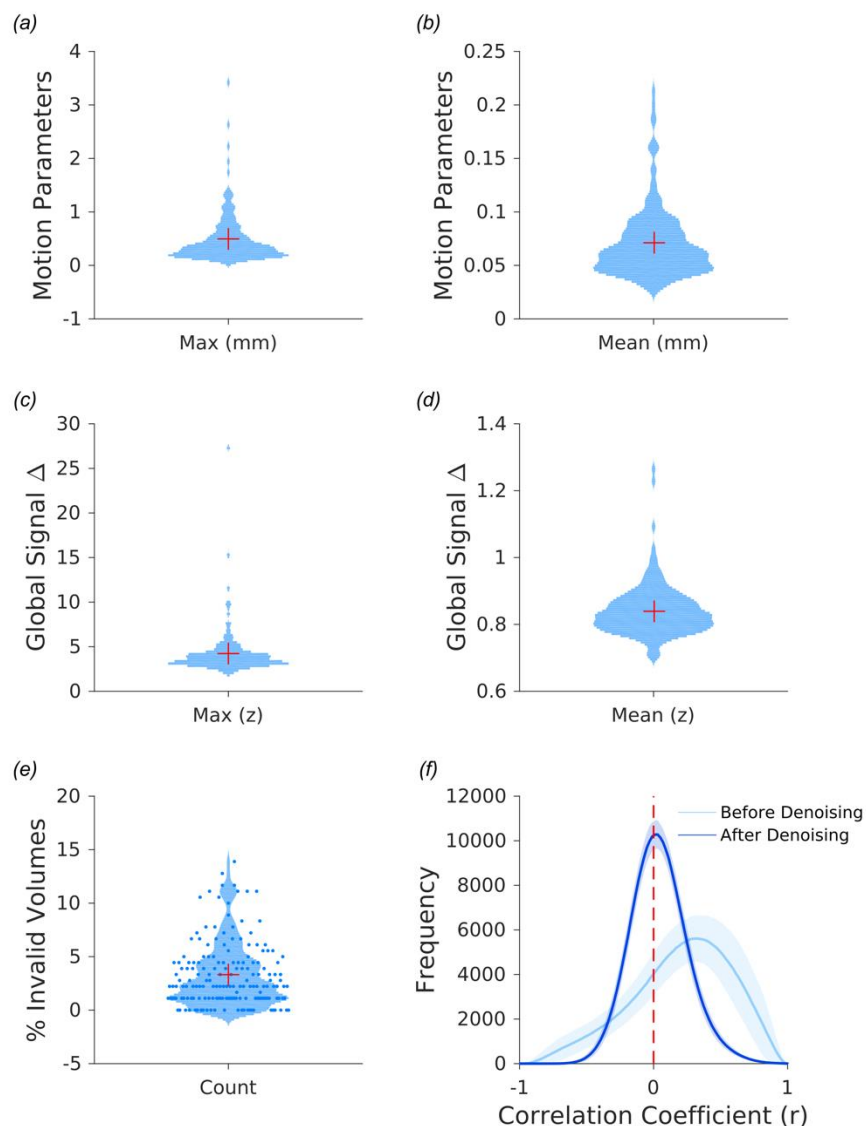
## Quality Assessment of ADHD Symptomatology Scores



**Supplementary Figure 2. Quality assessment of the ADHD symptomatology scores.** Violin plots representing the distribution of (a) ADHD scores from Part A subscale of the ASRS, (b) depression scores based on the Center for Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977), and (c) dyslexia scores based on the Dyslexia Adult Checklist (DAC) (Smythe and Everatt, 2001). There was a significant correlation between (d) ADHD scores and depression as well as (e) dyslexia scores. However, no significant relationship was observed between (f) ADHD scores and the percentage of invalid scans based on the composite motion-correction scores calculated via the employed scrubbing procedure. While the black lines illustrate the best linear fit, the red lines represent 95% confidence intervals. In order to ensure that these nuisance variables did not confound our data, they were all included as covariates of no interest in the subsequent statistical analyses.

## Quality Assessment of MRI Data

The distributions of maximum and average motion parameter values, as well as the average correlation coefficients before and after the employed denoising procedure are provided in Supplementary Figure 3. Following a strict motion-correction procedure, 9 participants who had more than 15% of their data affected by motion were excluded from the analysis.



**Supplementary Figure 3. MRI data quality assessment and motion correction.** An extensive motion-correction procedure was employed including the removal of motion parameters and their second-order derivatives, CompCor components attributable to white matter and cerebrospinal fluid and linear detrending. In addition, the volumes associated with excessive motion were identified and scrubbed. Participants with a

percentage of invalid volumes greater than 15% of their total data were excluded from the analysis. Distributions of *(a-b)* mean and maximum framewise displacement parameters (mm), *(c-d)* mean and maximum global BOLD signal change (*z*), and the *(e)* percentage of invalid scans for the final cohort of participants that were included in this analysis are provided using violin plots. The red stars indicate the 50<sup>th</sup> percentile. *(f)* In addition, the histogram of the average voxel-based correlation coefficients (*r*) across participants showed a normal distribution following the denoising steps employed in this study. The shaded areas represent standard deviation.

## **Supplementary References**

**Radloff, LS** (1977). The CES-D Scale: A Self-Report Depression Scale for Research in the General Population. *Applied Psychological Measurement* **1**, 385-401.

**Smythe, I & Everatt, J** (2001). A new dyslexia checklist for adults. In *The Dyslexia Handbook*. British Dyslexia Association.

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Dear Ms. Smith,

30 October 2018

**Re: PSM-D-18-00689, Detailed Response to Reviewer's Comments**

We thank the reviewer for the helpful comments, which we feel have considerably improved our manuscript. Below we provide point-by-point, detailed responses (regular type font) to the reviewer's comments (**bold**) and have modified the manuscript accordingly with [track changes](#). Where relevant, we have included modified sections of the edited manuscript below ("*italics*"). In addition, we have attached a clean version of the manuscript to aid with the revision process.

Kind regards,

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**Reviewer #2:**

**In this paper, Vatansever and colleagues examine neurotypical ADHD characteristics in a large sample of adults, and assess their relationship with the intrinsic functional connectivity of the default and frontoparietal control networks using fMRI. The large sample allowed for a well powered assessment of individual differences. Preprocessing and analytic procedures were appropriate, including diligent attention to motion and network selection. The authors found a performance-based measure of off task thought to be associated with self-reported ADHD symptomatology.**

**Critically, the authors found patterns of connectivity associated with ADHD scores. These included default-to-somatomotor connections, and frontoparietal-to-ventral visual regions. This pattern of connectivity was not necessarily predicted from the literature, but is appropriately interpreted. Additional emphasis on the exploratory nature of the approach would improve transparency. Overall, however, this is a well written manuscript with a novel set of interesting findings.**

First of all, we thank the reviewer for these insightful comments. We provide detailed responses to the questions raised below and have altered the manuscript accordingly.

**I have two recommendations for the discussion:**

**1) How do the results fit into a recent framework proposed by Christoff et al., 2016 Nature Reviews Neuroscience? If inconsistent, please explain.**

We thank the reviewer for raising this important point. We do believe that there are certain inconsistencies between the framework put forward by Christoff et. al., 2016 and the results of our study. Specifically, in contrast to the arguments made which suggest the excessive generation of off-task thoughts as the underlying cause of the cognitive deficits observed in ADHD, we do not find any evidence indicating that task-unrelated thoughts were related to ADHD symptoms. Instead, the results highlight that, at least in part, ADHD symptoms were related more to problems associated with maintaining detailed task representations, linked to intrinsic FPN connectivity. We have now altered the manuscript to highlight this point and added the following paragraph to our discussion section.

*“Contemporary accounts of spontaneous thought have argued that individuals with ADHD are unable to suppress internally-oriented cognition that is supported by the DMN (Andrews-Hanna et al., 2014, Christoff et al., 2016). Our analysis using MDES found no evidence that ADHD was linked to greater off-task thought. Moreover, while high levels of ADHD were linked to low levels of connectivity between the DMN and motor cortex, unlike the neural activity in the FPN, this connection showed no relationship with changes in detailed thought that were associated with ADHD scores. These results suggest that instead of problems in suppressing internally-oriented cognition related to over activity within the DMN, experiential differences in ADHD may be, at least in part, mediated by problems in maintaining detailed task representations. As is made explicit in executive failure views of mind-wandering (McVay and Kane, 2009), the inability to sustain attention on task relevant information, could indirectly produce periods of elevated off-task thought since individuals would spend less time focused on the task in hand (Smallwood et al., 2013a).”*



**2) What is the clinical utility of this finding? How can these results inform remediation of ADHD?**

We thank the reviewer for this suggestion. We have now included the following paragraph to our discussion section with the aim of answering this question.

*“We also consider the implications of our results for the occurrence and management of ADHD symptoms in the real world. Our study provides complementary neural and subjective markers that, if replicated within a clinical population, would provide an important metric for assessing the efficacy of both psychological and pharmacological interventions for individuals with this disorder. For example, psychological interventions, such as mindfulness training (Mitchell et al., 2015), and drug interventions (Turner et al., 2005) have both shown promise in reducing ADHD symptomatology. Based on our results, studies combining experience sampling with measures of neural function may provide important insight into the specific neurocognitive changes that underlie the effectiveness of such interventions. In addition, given mounting evidence on the genetic basis of ADHD (Mick and Faraone, 2008, Pironti et al., 2014), population studies that examine experiential and neural differences that emerge in this cohort may provide unique insight into the link between genes, behaviour and cognition.”*