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**Activating attachment memories affects default mode network in a non-clinical sample with perceived dysfunctional parenting: An EEG functional connectivity study**

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1 **Activating attachment memories affects Default Mode Network in a**  
2 **non-clinical sample with perceived dysfunctional parenting: an EEG**  
3 **functional connectivity study**

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23

24 **Abstract**

25 Dysfunctional parenting constitutes a factor of psychopathological vulnerability affecting  
26 development both at neurobiological and psychological level. The default mode network (DMN), a  
27 large scale network for brain functional integration, is supposed to play a crucial role in those  
28 psychological functions altered by dysfunctional parenting. Here we investigate  
29 electroencephalography DMN functional connectivity in relation to perceived dysfunctional  
30 parenting (PDP) in a non-clinical sample. We hypothesized that participants with high PDP would  
31 exhibit decreased DMN connectivity after the activation of attachment memories. Our results  
32 support this hypothesis: participants with high PDP showed a decrease of theta connectivity  
33 between left temporoparietal junction and right anterior cingulate cortex after the activation of  
34 attachment memories, and, compared to participants with low PDP, showed a decrease of delta  
35 connectivity in the same brain areas. We interpret these decreased DMN connectivity in participants  
36 with high PDP as the “neurophysiological signature” of the impaired ability to mentalize their own  
37 relational experiences with significant others after the activation of early attachment memories.  
38 Thus, the activation of attachment memories in individuals exposed to dysfunctional parenting  
39 could lead to a transitory failure of functional brain connectivity and consequent disturbance of high  
40 integrative mental functions, such as emotional regulation and mentalization.

41

42

43 **Keywords:** Attachment, Default mode network, Dysfunctional parenting, Electroencephalography,  
44 Functional connectivity, Mentalization

## 45 **Introduction**

46           Dysfunctional parenting, such as very low care, emotional abuse and high overprotection,  
47 has been compared to others forms of child maltreatment [1-5]. Consistently, a significant and  
48 increasing body of evidence suggests that dysfunctional and/or neglectful parenting, like other  
49 forms of early relational trauma, is one of the major risk factor and negative prognostic cause for  
50 almost all psychiatric disorders [6-9]. Indeed, regardless of specific diagnosis, it has been reported  
51 that dysfunctional parenting constitutes a factor of psychopathological vulnerability affecting  
52 development both at neurobiological and psychological level [2, 6-8, 10-13]. Among the most  
53 common psychopathological consequences related to dysfunctional parenting there are emotive  
54 disorders and dysregulation [14, 15], alterations in inhibitory control and executive functions [10],  
55 cognitive and consciousness disturbances [2, 16, 17], self identity and self agency alterations [18,  
56 19], mentalization dysfunctions [5, 20], relational problems and low social competence [21, 22].  
57 Some scholars have hypothesized that a significant amount of these psychopathological  
58 disturbances have in common a lack of mental integration produced by dysfunctional parenting [2,  
59 16, 23, 24].

60 Under a neurobiological point of view it has been supposed that the default mode network (DMN),  
61 a crucial large scale network for brain functional integration [25-27], plays an important role in  
62 those psychological functions altered by dysfunctional parenting [28-31]. Consistently, the DMN  
63 and its subcomponents alterations are frequently reported in people with dysfunctional parenting  
64 and other forms of child maltreatment [8, 12].

65 One of the most intriguing issue in this area is that clinical observations and empirical research data  
66 lead to consider that some of these disturbances, such as emotive and behavioural dysregulation,  
67 dissociative symptoms, mentalization disruption, relational problems, are not stable symptoms, but  
68 may emerge when triggered by socio-emotional stimuli like the activation of early attachment  
69 memories [16, 24, 32]. According to attachment theory and its subsequent clinical applications, the  
70 automatic and implicit (i.e., unconscious) activation of attachment relational memories in

71 individuals with histories of neglect or maltreatment in childhood could trigger disintegrative  
72 psychopathological process that leads to typical psychopathology related to dysfunctional parenting  
73 [2, 23, 24].

74 For this reason the aim of the present study was to investigate electroencephalography DMN  
75 functional connectivity in relation to the quality of the perceived dysfunctional parenting (PDP), i.e.  
76 the self-reported experiences of neglect, abuse and/or overprotection within the relationships with  
77 one's parents [33-35], both in resting state (RS) and after the activation of attachment memories  
78 using the Adult Attachment Interview as a trigger (AAI) [36, 37] in a non-clinical sample. Based on  
79 empirical data and clinical grounds, we hypothesized that participants with PDP, compared to  
80 participants without PDP, would exhibit decreased DMN connectivity after the activation of  
81 attachment memories.

82

## 83 **Materials and Methods**

### 84 *Participants*

85 Participants were 50 students (fourteen men, mean age:  $22.62 \pm 2.41$  years) recruited  
86 through advertisements posted in the university. The enrollment lasted from October 2017 to May  
87 2018. Study participants contributed voluntarily and anonymously after providing informed  
88 consent. They did not receive payment or any other compensation (i.e., academic credit). Inclusion  
89 criteria were: age between 18 and 30 years, both genders. Exclusion criteria were: history of  
90 psychiatric disease and/or neurologic diseases; head trauma; left handedness; assumption of Central  
91 Nervous System active drugs in the two weeks prior to assessment. A checklist with dichotomous  
92 items was used to assess inclusion/exclusion criteria and socio-demographic data.

93 After receiving information about the aim of the study, all participants provided a written  
94 consent to participate in the study that was performed according to the Helsinki declaration  
95 standards. The research was approved by the European University's ethic review board.

96

97 *Procedure*

98           After providing the written informed consent, all participants were administered the  
99 Measure of Parental Style (MOPS) [38] and the Brief Symptom Inventory (BSI) [39]. Furthermore,  
100 in order to identify the presence of past and/or current psychiatric disorders, during the intake visit,  
101 participants were asked screening questions according to a checklist prepared for a previous study  
102 [40].

103           On a separate day from the self-report assessment, all participants underwent the Adult  
104 Attachment Interview (AAI) [37], a semi-structured interview able to activate the attachment  
105 system by the retrieval of childhood emotional and relational memories of past attachment  
106 experiences [37, 41]. Rigorous psychometric testing and meta-analyses of the AAI demonstrate  
107 stability and discriminant and predictive validity in both clinical and nonclinical populations [42,  
108 43]. In the present study, the AAI was used as “trigger stimulus” of the attachment behavioral  
109 system. Indeed, previous studies demonstrated that the AAI is able to alter psychophysiological  
110 parameters related to the emotion regulation of people with different attachment styles [36, 44, 45]  
111 and to modify the cortical functional connectivity related to the retrieval of early attachment  
112 memories in both healthy and clinical subjects [16].

113           Trained clinical psychologists (LP) administered the AAI in a quiet and comfortable room.  
114 EEG recordings were performed before (Pre-AAI condition) and immediately after (i.e., about 8-10  
115 minutes for each participant) the interview (Post-AAI condition). The interviews lasted on average  
116 one hour and 30 minutes.

117

118 *Questionnaire*

119           The MOPS [38] is the redefined version of the Parental Bonding Instrument [46] and it is  
120 composed by 30 items which separately investigate mother’ (15 items) and father’ (15 items)  
121 parental styles. Items are scored on a 4-point Likert scale (from “not true at all” to “extremely true”)  
122 and grouped in three dimensions for each parent, confirmed through principal components analysis

123 [38]: indifference, over-control, and abuse. Higher scores reflect higher self-reported experiences of  
124 neglect, abuse and/or overprotection during the first 16 years of life. The MOPS has been used  
125 extensively in clinical research [9, 34, 47] and it was developed to overcome some negative aspects  
126 (e.g., low clarity of several items) of the original version [9]. Satisfactory psychometric properties  
127 have been reported in the original validation study [38]. Furthermore, good cross-cultural adaptation  
128 has been observed [48, 49]. In the present research, the Italian version of the scale has been used  
129 [48] and the Cronbach's alpha in the present sample was 0.88 for the 30-item MOPS total scores.

130 The BSI [39] is the short version of the Symptom Checklist-90R [50] and it is composed by  
131 53 items evaluating a broad range of psychological symptoms during the past seven days. Items are  
132 scored on a 4-point Likert scale ranging from 0 (not at all) to 4 (extremely) and grouped in 9  
133 primary symptom dimensions: somatization, obsession-compulsion, interpersonal sensitivity,  
134 depression, anxiety, hostility, phobic anxiety, paranoid ideation and psychoticism. A measure of  
135 general level of psychopathology, the global severity index (GSI), is also calculated using the sums  
136 for the nine symptom dimensions (higher scores reflects more self-reported symptoms). The BSI is  
137 widely used in clinical research and it is characterized by good psychometric properties [51]. In the  
138 present study, the Italian version of the scale was used [52] and the Cronbach's alpha in the present  
139 sample was 0.94 for the GSI.

140

#### 141 *EEG recordings*

142 Resting State (RS) EEG was recorded using the Micromed System Plus digital EEGraph  
143 (Micromed© S.p.A., Mogliano Veneto, TV, Italy) in the European University EEG Lab, with each  
144 participants sitting in a comfortable armchair, with his/her eyes closed, in a quiet, semi-darkened  
145 silent room for 5 minutes. In order to avoid alcohol and or caffeine effects on EEG data,  
146 participants were asked to refrain from drinking alcohol and caffeine for 4 to 6 hours immediately  
147 before their EEG recordings.

148 EEG recordings included 31 standard scalp leads, positioned according to the 10-20 system  
149 (recording sites: Fp1, AF3, F3, FC1, C3, CP1, P3, PO3, O1, F7, FC5, T3, CP5, T5, Fz, Cz, Pz, Fp2,  
150 AF4, F4, FC2, C4, CP2, P4, PO4, O2, F8, FC6, T4, CP6, T6), and the Electrocardiography (ECG).  
151 The reference electrodes were placed on the linked mastoids. Impedances were kept below 5K $\Omega$   
152 before starting the recording and checked again at the end of the experimental recording. Sampling  
153 frequency was 256 Hz; A/D conversion was made at 16 bit; pre-amplifiers amplitude range was  
154  $\pm 3200$  and low-frequency pre-filters were set at 0.15 Hz. The following band-pass filters were used:  
155 HFF= 0.2 Hz; LFF= 128 Hz. In the present study the following frequency bands was considered:  
156 delta (0.5–4 Hz); theta (4.5–7.5 Hz); alpha (8–12.5 Hz); beta (13–30 Hz); gamma (30.5–60 Hz).

157 Details about artifact rejection have been described elsewhere [40]. Briefly, visual artifact  
158 rejection (e.g., cap adjustment) was firstly performed on the raw EEG trace. These segments were  
159 removed, and then independent component analysis (ICA) was applied to EEG recordings to  
160 identify and remove non-cerebral artifacts (i.e., eye and muscular movements, cardiac pulses)  
161 before data analysis. Although it has been reported that ICA correction may affect EEG  
162 connectivity [53], correcting artifacts using this procedure is widely used in EEG phase  
163 synchronization studies [54-56]. Furthermore, several reports [54-56] documented no significant  
164 modifications of EEG coherence data after ICA correction.

165 The minimum length of the artifact-free EEG recording included in the analysis was 180  
166 seconds (even if not consecutive) for each participant for each condition (i.e., pre-AAI and post-  
167 AAI).

168

### 169 *Connectivity analysis*

170 All EEG analysis were performed using the exact Low Resolution Electromagnetic  
171 Tomography software (eLORETA), a validated tool for localizing brain electric activity based on  
172 multichannel surface EEG recordings [57]. The eLORETA software is characterized by a  
173 satisfactory localization agreement with different multi-modal imaging techniques, and it is a

174 suitable tool for DMN assessment [58]. The connectivity analysis were performed using the lagged  
175 phase synchronization formula [59]. This algorithm has been widely used to assess EEG functional  
176 connectivity, and it is characterized by several advantages (e.g., it is resistant to non-physiological  
177 artifacts) [59]. Although functional magnetic resonance imaging (fMRI) is commonly used to  
178 investigate the functional connectivity of DMN, recent studies have shown that EEG is also suitable  
179 for investigating the functional properties of this network.

180         According to previous EEG connectivity studies [29, 30, 40, 60], in order to evaluate  
181 functional connectivity in the DMN, 12 Regions of Interest (ROIs) were selected (Figure 1) and the  
182 ‘single nearest voxel’ option (i.e., each ROI consisted of a single voxel, the closest to each seed)  
183 was chosen (detailed DMN Montreal Neurological Institute and Talairach coordinates can be found  
184 in [40]). Briefly, the “ROI-maker#2 method” available in the eLORETA software has been selected  
185 and, starting from 42 Brodmann Areas (BAs) in each hemisphere provided by the software [61], 12  
186 ROIs were defined according Thatcher et al. [58].

187 The eLORETA calculated the lagged phase synchronization values between all these ROIs (i.e., 144  
188 connections) and the source reconstruction algorithm [57].

189

#### 190 *Statistical analysis*

191         In order to reveal groups of subjects with high and low PDP (i.e., PDP+ and PDP- groups), a  
192 Two Step Cluster Analysis procedure was performed using MOPS sub-scales scores. Cluster  
193 solutions was assessed using Schwarz's Bayesian Criterion (BIC) as clustering criterion [62]. Chi-  
194 squared tests ( $\chi^2$ ), and Mann–Whitney’s U tests were used to investigate differences between  
195 clusters, respectively for dichotomous and dimensional variables.

196         EEG connectivity analysis was performed using the eLORETA software. Between and  
197 within comparisons were performed for each frequency band. Specifically the following statistical  
198 comparisons were performed: i) Pre-AAI PDP+ vs Pre-AAI PDP-, ii) Post-AAI PDP+ vs Pre-AAI  
199 PDP+, iii) Post-AAI PDP- vs Pre-AAI PDP-, iv) Post-AAI PDP+ vs Pre-AAI PDP-. All

200 comparisons were performed using the statistical non-parametric mapping (SnPM) methodology  
201 provided by the eLORETA software (i.e, a Fisher's permutation test) [63]. In order to avoid family-  
202 wise type-I errors, the non-parametric randomization procedure (supplied by the eLORETA  
203 software), was performed for the correction of multiple comparison [63]. For all comparisons, the  
204 eLORETA software provides experimental values of T, corresponding to a significance of  $p < 0.01$   
205 and  $p < 0.05$ .

206 Finally, Spearman's *rho* correlation coefficients were reported as measures of associations  
207 among MOPS subscales scores, GSI, and any significant EEG connectivity data observed in the  
208 between comparisons. Cluster Analysis, Chi-squared tests, Mann-Whitney's U tests, correlation  
209 analyses were performed using IBM SPSS Statistics for Windows, version 23.0. The use of  
210 nonparametric tests was chosen because none of the present variables were normally distributed  
211 (Shapiro-Wilk test,  $p < 0.05$ ).

212

## 213 **Results**

214 The Two Step Cluster Analysis procedure indicated a 2-group solution (BIC change= -  
215 36.01; Ratio of distance measures= 3.38). 34 % of the sample (N= 17) was included in the first  
216 cluster, and 66 % (N= 33) was included in cluster 2. Compared to individuals included in cluster 2  
217 (i.e., PDP- group), subjects included in cluster 1 (i.e., PDP+ group) had significantly higher scores  
218 in all PDP sub-subscales. Thus, cluster 1 is mostly characterized by individuals reporting higher  
219 PDP. Furthermore, compared to the individuals with low PDP, those with high PDP had a  
220 significantly higher scores in the GSI and in all BSI subscales, with the exception of interpersonal  
221 sensitivity, phobic anxiety and psychoticism subscales. Detailed bivariate analyses are listed in  
222 Table 1.

223 EEG recordings suitable for the analysis were obtained for all participants. Qualitative  
224 visual evaluation of the EEG recordings, performed by a trained neurophysiologist, showed no  
225 relevant modifications of the background rhythm frequency (e.g., epileptic discharges).

226 Furthermore, no relevant modifications of EEG signal (e.g., evidence of sleepiness) during the  
227 recordings were detected. The average time analyzed for the present sample was  $283 \pm 14$  sec and  
228  $276 \pm 15$  respectively for cluster 1 and cluster 2 subjects in pre-AAI condition and  $291 \pm 12$  sec and  
229  $277 \pm 19$  in post-AAI condition.

230

### 231 *Connectivity results*

232 In the between-groups comparison (PDP+ vs PDP-) for the Pre-AAI condition, the thresholds for  
233 significance were  $T = \pm 2.73$  corresponding to  $p < 0.05$ , and  $T = \pm 3.19$  corresponding to  $p < 0.01$ . In  
234 this condition, no significant modifications were observed between groups (Figure 2; Panel A).

235 In the within-group comparison (Post-AAI vs. Pre-AAI) for the PDP+ group, the  
236 thresholds for significance were  $T = \pm 3.99$  corresponding to  $p < 0.05$ , and  $T = \pm 4.63$ , corresponding  
237 to  $p < 0.01$ . In this comparison, significant modifications were observed in the theta frequency  
238 (Figure 3; Panel A). Compared to Pre-AAI condition, PDP+ individuals showed in Post-AAI  
239 condition a decrease of theta connectivity between left Temporoparietal Junction (TPJ; ROI 11) and  
240 right Anterior Cingulate Cortex (ACC; ROI 8) ( $T = -4.09$ ,  $p = 0.037$ ). No significant differences  
241 were observed in the other frequency bands, although a significant trend was observed between left  
242 TPJ and right ACC also in the delta band ( $T = -3.76$ ,  $p = 0.08$ ).

243 In the within-group comparison (Post-AAI vs. Pre-AAI) for the PDP- group, the thresholds  
244 for significance group were  $T = \pm 3.54$  corresponding to  $p < 0.05$ , and  $T = \pm 4.03$ , corresponding to  $p <$   
245  $0.01$ . In this comparison, significant modifications were observed in the alpha frequency band  
246 (Figure 3; Panel B). Compared to Pre-AAI condition, PDP- individuals showed in Post-AAI  
247 condition an increase of alpha connectivity between right TPJ (ROI 12) and both right and left  
248 Posterior Cingulate Cortex (PCC; ROI 6 and ROI 5) (respectively  $T = 3.56$ ;  $p = 0.047$  and  $T = 3.71$ ;  
249  $p = 0.030$ ). No significant differences were observed in the other frequency bands

250 In the between-groups comparison (PDP+ vs PDP-) for the Post-AAI condition, the  
251 thresholds for significance were  $T = \pm 2.92$  corresponding to  $p < 0.05$ , and  $T = \pm 3.47$  corresponding to

252  $p < 0.01$ . Significant modifications were observed in the delta band (Figure 3; Panel B). Compared  
253 to PDP- individuals, PDP+ participants showed a decrease of delta connectivity between left TPJ  
254 (ROI 11) and right ACC (ROI 8) ( $T = -3.29$ ;  $p = 0.018$ ). No significant differences were observed in  
255 the other frequency bands.

256

#### 257 *Association among EEG functional connectivity data, MOPS and GSI scores*

258 MOPS total score was negatively related with the strength of delta connectivity between left  
259 TPJ and right ACC ( $\rho = -0.28$ ;  $p = 0.048$ ). Furthermore, the strength of delta connectivity observed  
260 after the AAI between left TPJ and right ACC was negatively related with both maternal  
261 indifference ( $\rho = -0.36$ ;  $p = 0.010$ ) and maternal over-control ( $\rho = -0.33$ ;  $p = 0.020$ ) sub-scale.  
262 Although GSI was positively related with all MOPS sub-scales, no significant correlation was  
263 observed between EEG connectivity data and psychopathological score. Detailed correlations are  
264 reported in Table 2.

265

#### 266 **Discussion**

267 The a priori hypothesis of the present study was that participants with high PDP (i.e., PDP+  
268 group), compared to participants with low or without PDP (i.e., PDP- group), would exhibit  
269 decreased DMN connectivity after the activation of attachment memories. Our results support this  
270 hypothesis. Indeed, after the activation of attachment memories triggered by the AAI, PDP+  
271 participants (within-group comparison) showed a decrease of theta connectivity between left TPJ  
272 and right ACC. Furthermore, after the administration of the AAI, compared to PDP- participants,  
273 PDP+ individuals showed a decrease of delta connectivity in the same brain areas (i.e., left TPJ and  
274 right ACC). Consistently with our hypothesis, these connectivity modifications were observed  
275 exclusively after the activation of early attachment memories as no significant DMN connectivity  
276 differences were detected in the between-groups comparison before the administration of the AAI.

277 Our results are in line with previous studies reporting DMN alterations in people with  
278 dysfunctional parenting and other forms of early relational adverse experiences [8, 12]. The DMN is  
279 thought to be involved in several higher-order integrative mental functions such as self-  
280 consciousness, self-processing and episodic memory [58, 64] that are supposed to be impaired by  
281 dysfunctional parenting. This network has been conceptualized as a distributed and dynamic brain  
282 system composed by a set of interacting hubs and subsystems with specific functions [26, 27].  
283 Specifically, the dorsal medial subsystem, which includes several brain areas such as the TPJ and  
284 dorsal medial prefrontal cortex, has been associated with mentalization, social cognition as well as  
285 with semantic/conceptual processing. Conversely, the medial temporal subsystem, which involves  
286 anatomical regions such as hippocampal formation, the retrosplenial cingulate cortex and ventral  
287 medial prefrontal cortex, has been related with autobiographical thought, episodic memory and  
288 contextual retrieval. Finally, the midline hubs of the DMN, namely the mPFC, the rostral anterior  
289 cingulate and the posterior cingulate cortex, are involved across a wide range of self-related  
290 processes integrating the dorsal medial and medial temporal subsystem [26, 27].

291 Therefore, taking into account DMN related functions and processes as well as the type of  
292 mental processes elicited by the AAI, we may speculate that the decreased DMN connectivity  
293 observed between left TPJ and right ACC in the PDP+ participants is the “neurophysiological  
294 signature” of the impaired ability to mentalize their own relational experiences with significant  
295 others after the activation of early attachment memories.

296 Indeed, the role of both left and right TPJ and of the ACC in mentalization [i.e., the ability to  
297 attribute mental states to oneself and to others and to understand that others have mental states  
298 independent from one’s own; see for example 65, 66, 67] is widely recognized in the literature [68-  
299 70].

300 Our interpretation is also strengthened by the increase of DMN connectivity between right TPJ and  
301 both right and left PCC observed in PDP- participants after the administration of the AAI. The PCC  
302 is considered the crucial node of the DMN [71] and it is involved in several emotion and cognitive

303 processing [72], with critical relevance in maintaining a sense of self-consciousness and self-  
304 referential thoughts during RS [73]. It is also interesting to note that PDP- participants showed  
305 increased connectivity in the alpha frequency band, which is considered to be positively related to  
306 DMN activity as well as with spontaneous self-referential processes, such as mentalization [25].  
307 Therefore, this result seems to support our interpretation according to which during the AAI  
308 participants are induced to mentalize their own relational experiences, presumably activating a set  
309 of self-related processes such as episodic and autobiographical memories.

310 Taken together, our results support both clinical observations and experimental results  
311 according to which the alterations associated with PDP are not stable symptoms but may emerge  
312 when triggered by early attachment relational memories [16, 23, 32]. These results seem to be also  
313 partially consistent with MRI studies indicating that early-life adversity may be associated with  
314 structural alterations in brain white matter, specifically in the cingulate cortex [11].  
315 According to our data, it is possible to hypothesize that, in individuals with early adverse relational  
316 experiences, the structural connectivity deficit becomes functionally evident and clinically  
317 symptomatic when the system is overloaded by affective and cognitive attachment related stimuli.  
318 Moreover, although it was not among the goals of the present research, future work could  
319 investigate whether and to what extent the attachment style can influence the DMN functional  
320 connectivity. In fact, the same adverse experiences can lead to very different development paths and  
321 the possibility that different attachment styles may be associated with different patterns of alteration  
322 of functional connectivity cannot be excluded in principle.

323 Another result rising from this study is the usefulness of MOPS in detecting the  
324 psychopathological vulnerability related to the early relational adverse experiences. In our opinion,  
325 MOPS based screening could be useful in the clinical practice where the clients report their own  
326 perception of parental way to protect and control, and combined with EEG connectivity seems to be  
327 a useful and reliable tool to improve our understanding on the psychopathological processes  
328 underlying PDP.

329 In spite of our interesting results, the study has some limitations that should be considered.  
330 Firstly, the sample size is limited, which may affect the generalizability of the results. Furthermore,  
331 our sample included mostly female participants, and previous studies showed sex differences in  
332 EEG brain activity during RS condition [74].  
333 Secondly, we have investigated PDP and psychopathology using self-report measures, which are  
334 known to be potentially affected by social desirability. Thirdly, we used scalp EEG recordings,  
335 which have an intrinsic limit in space resolution. Finally, we have investigated DMN functional  
336 connectivity after AAI, which make our interpretations specific to the activation of attachment  
337 memories. It is possible that others not-related attachment triggers (e.g., viewing negative emotional  
338 facial expressions) may be associated with different DMN alterations in high PDP individuals.  
339 Although these ideas are purely hypothetical, they might be useful in guiding future research.

340 Despite these limitations, to the best of our knowledge, this is the first study which  
341 investigated the association between DMN EEG functional connectivity and PDP both in RS and  
342 after the activation of attachment memories. In conclusion, our results seem to support the  
343 hypothesis according to which the activation of attachment memories in individuals exposed to  
344 dysfunctional parenting and other forms of early relational adverse experiences could lead to a  
345 transitory failure of functional brain connectivity and consequent disturbance of high integrative  
346 mental functions. These transitory alterations might explain, even partially, the emergence of some  
347 typical psychopathological symptoms such as emotional dysregulation, dissociative symptoms,  
348 inhibitory control and executive functions disturbances, self identity and self agency and  
349 mentalization impairments [10, 16, 23, 24]. Therefore, our result also highlights the possibility of  
350 developing new therapeutic approaches focused on the self neuro-modulation, such as alpha/theta  
351 neurofeedback, which may increases mentalization and DMN EEG connectivity [60].

352

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358 **Reference**

359

- 360 [1] N. Draijer, W. Langeland, Childhood trauma and perceived parental dysfunction in the etiology of  
361 dissociative symptoms in psychiatric inpatients, *Am J Psychiatry* 156 (3) (1999) 379-385.
- 362 [2] L. Dutra, J.F. Bureau, B. Holmes, A. Lyubchik, K. Lyons-Ruth, Quality of early care and childhood  
363 trauma: a prospective study of developmental pathways to dissociation, *J Nerv Ment Dis* 197 (6)  
364 (2009) 383-390.
- 365 [3] M.H. Teicher, J.A. Samson, Childhood maltreatment and psychopathology: A case for  
366 ecophenotypic variants as clinically and neurobiologically distinct subtypes, *Am J Psychiatry* 170  
367 (10) (2013) 1114-1133.
- 368 [4] B. Farina, G. Liotti, Does a dissociative psychopathological dimension exist? A review on  
369 dissociative processes and symptoms in developmental trauma spectrum disorders, *Clin*  
370 *Neuropsychiatry* 10 (1) (2013) 11-18.
- 371 [5] F.J. Infurna, C.T. Rivers, J. Reich, A.J. Zautra, Childhood trauma and personal mastery: their  
372 influence on emotional reactivity to everyday events in a community sample of middle-aged adults,  
373 *PLoS One* 10 (4) (2015) e0121840.
- 374 [6] A.R. Lima, M.F. Mello, J. Mari Jde, The role of early parental bonding in the development of  
375 psychiatric symptoms in adulthood, *Curr Opin Psychiatry* 23 (4) (2010) 383-387.
- 376 [7] M.H. Teicher, J.A. Samson, C.M. Anderson, K. Ohashi, The effects of childhood maltreatment on  
377 brain structure, function and connectivity, *Nat Rev Neurosci* 17 (10) (2016) 652-666.
- 378 [8] E.J. McCrory, M.I. Gerin, E. Viding, Annual Research Review: Childhood maltreatment, latent  
379 vulnerability and the shift to preventative psychiatry - the contribution of functional brain imaging, *J*  
380 *Child Psychol Psychiatry* 58 (4) (2017) 338-357.
- 381 [9] R. Icick, S. Lauer, L. Romo, G. Dupuy, J.P. Lepine, F. Vorspan, Dysfunctional parental styles  
382 perceived during childhood in outpatients with substance use disorders, *Psychiatry Res* 210 (2)  
383 (2013) 522-528.
- 384 [10] P. Pechtel, D.A. Pizzagalli, Effects of early life stress on cognitive and affective function: an  
385 integrated review of human literature, *Psychopharmacology (Berl)* 214 (1) (2011) 55-70.
- 386 [11] P.E. Lutz, A. Tanti, A. Gasecka, S. Barnett-Burns, J.J. Kim, Y. Zhou, G.G. Chen, M. Wakid, M.  
387 Shaw, D. Almeida, M.A. Chay, J. Yang, V. Lariviere, M.N. M'Boutchou, L.C. van Kempen, V.  
388 Yerko, J. Prud'homme, M.A. Davoli, K. Vaillancourt, J.F. Theroux, A. Bramouille, T.Y. Zhang, M.J.  
389 Meaney, C. Ernst, D. Cote, N. Mechawar, G. Turecki, Association of a History of Child Abuse With  
390 Impaired Myelination in the Anterior Cingulate Cortex: Convergent Epigenetic, Transcriptional, and  
391 Morphological Evidence, *Am J Psychiatry* 174 (12) (2017) 1185-1194.
- 392 [12] N.S. Philip, L.H. Sweet, A.R. Tyrka, L.H. Price, R.F. Bloom, L.L. Carpenter, Decreased default  
393 network connectivity is associated with early life stress in medication-free healthy adults, *Eur*  
394 *Neuropsychopharmacol* 23 (1) (2013) 24-32.
- 395 [13] J.R. Measelle, J. David, J.C. Ablow, Increased levels of inflammation among infants with  
396 disorganized histories of attachment, *Behav Brain Res* 325 (Pt B) (2017) 260-267.

- 397 [14] E.C. Dunn, K. Nishimi, S.H. Gomez, A. Powers, B. Bradley, Developmental timing of trauma  
398 exposure and emotion dysregulation in adulthood: Are there sensitive periods when trauma is most  
399 harmful?, *J Affect Disord* 227(2018) 869-877.
- 400 [15] S. Kench, H.J. Irwin, Alexithymia and childhood family environment, *J Clin Psychol* 56 (6) (2000)  
401 737-745.
- 402 [16] B. Farina, A.M. Speranza, S. Dittoni, V. Gnoni, C. Trentini, C.M. Vergano, G. Liotti, R. Brunetti, E.  
403 Testani, G. Della Marca, Memories of attachment hamper EEG cortical connectivity in dissociative  
404 patients, *Eur Arch Psychiatry Clin Neurosci* 264 (5) (2014) 449-458.
- 405 [17] E. McCrory, S.A. De Brito, E. Viding, Research review: the neurobiology and genetics of  
406 maltreatment and adversity, *J Child Psychol Psychiatry* 51 (10) (2010) 1079-1095.
- 407 [18] E.A. Carlson, T.M. Yates, L.A. Sroufe, Dissociation and the Development of the Self. In: P. Dell,  
408 J.A. O'Neil (Eds.), *Dissociation and Dissociative Disorders: DSM-V and beyond*, Routledge, New  
409 York, 2009, pp. 39-52.
- 410 [19] S. Pallini, A. Alfani, L. Marech, F. Laghi, Unresolved attachment and agency in women victims of  
411 intimate partner violence: A case-control study, *Psychol Psychother* 90 (2) (2017) 177-192.
- 412 [20] M. Brune, S. Walden, M.A. Edel, G. Dimaggio, Mentalization of complex emotions in borderline  
413 personality disorder: The impact of parenting and exposure to trauma on the performance in a novel  
414 cartoon-based task, *Compr Psychiatry* 64(2016) 29-37.
- 415 [21] A. van Dijke, J.A.B. Hopman, J.D. Ford, Affect dysregulation, psychoform dissociation, and adult  
416 relational fears mediate the relationship between childhood trauma and complex posttraumatic stress  
417 disorder independent of the symptoms of borderline personality disorder, *Eur J Psychotraumatol* 9  
418 (1) (2018) 1400878.
- 419 [22] G. Attili, P. Vermigli, A. Roazzi, Children's social competence, peer status, and the quality of  
420 mother-child and father-child relationships: A multidimensional scaling approach, *Eur Psychol* 15  
421 (1) (2010) 23-33.
- 422 [23] G. Liotti, B. Farina, Painful Incoherence: The Self in Borderline Personality Disorder. In: M. Kyrios,  
423 R. Moulding, M. Nedeljkovic, S.S. Bhar, G. Doron, M. Mikulincer (Eds.), *The self in understanding  
424 and treating psychological disorders*, Cambridge University Press, Cambridge, 2016, pp. 169-178.
- 425 [24] R. Meares, *The Dissociation Model of Borderline Personality Disorder*, Norton Professional Books,  
426 New York, 2012.
- 427 [25] G.G. Knyazev, EEG correlates of self-referential processing, *Front Hum Neurosci* 7(2013) 264.
- 428 [26] J.R. Andrews-Hanna, The brain's default network and its adaptive role in internal mentation,  
429 *Neuroscientist* 18 (3) (2012) 251-270.
- 430 [27] J.R. Andrews-Hanna, J. Smallwood, R.N. Spreng, The default network and self-generated thought:  
431 component processes, dynamic control, and clinical relevance, *Ann N Y Acad Sci* 1316(2014) 29-  
432 52.
- 433 [28] P. Qin, G. Northoff, How is our self related to midline regions and the default-mode network?,  
434 *Neuroimage* 57 (3) (2011) 1221-1233.
- 435 [29] C. Imperatori, G. Della Marca, R. Brunetti, G.A. Carbone, C. Massullo, E.M. Valenti, N. Amoroso,  
436 G. Maestoso, A. Contardi, B. Farina, Default Mode Network alterations in alexithymia: an EEG  
437 power spectra and connectivity study, *Sci Rep* 6(2016) 36653.

- 438 [30] B. Farina, G. Della Marca, G. Maestoso, N. Amoroso, E.M. Valenti, G.A. Carbone, C. Massullo, A.  
439 Contardi, C. Imperatori, The Association among Default Mode Network Functional Connectivity,  
440 Mentalization, and Psychopathology in a Nonclinical Sample: An eLORETA Study,  
441 *Psychopathology* 51 (1) (2018) 16-23.
- 442 [31] X. Xie, S. Mulej Bratec, G. Schmid, C. Meng, A. Doll, A. Wohlschlager, K. Finke, H. Forstl, C.  
443 Zimmer, R. Pekrun, L. Schilbach, V. Riedl, C. Sorg, How do you make me feel better? Social  
444 cognitive emotion regulation and the default mode network, *Neuroimage* 134(2016) 270-280.
- 445 [32] B. Vai, L. Sforzini, R. Visintini, M. Riberto, C. Bulgarelli, D. Ghiglini, E. Melloni, I. Bollettini, S.  
446 Poletti, C. Maffei, F. Benedetti, Corticolimbic Connectivity Mediates the Relationship between  
447 Adverse Childhood Experiences and Symptom Severity in Borderline Personality Disorder,  
448 *Neuropsychobiology* 76 (2) (2017) 105-115.
- 449 [33] M.V. Torrado, S.S. Ouakinin, L. Bacelar-Nicolau, Alexithymia, Emotional Awareness and Perceived  
450 Dysfunctional Parental Behaviors in Heroin Dependents, *Int J Ment Health Addiction* 11 (6) (2013)  
451 703-718.
- 452 [34] K. Fletcher, G. Parker, A. Bayes, A. Paterson, G. McClure, Emotion regulation strategies in bipolar  
453 II disorder and borderline personality disorder: differences and relationships with perceived parental  
454 style, *J Affect Disord* 157(2014) 52-59.
- 455 [35] K. Otani, A. Suzuki, Y. Matsumoto, T. Shirata, Link of negative core beliefs about the self with  
456 perceived dysfunctional parenting, *Psychiatry Res* 270(2018) 715-719.
- 457 [36] M. Dozier, R.R. Kobak, Psychophysiology in attachment interviews: converging evidence for  
458 deactivating strategies, *Child Dev* 63 (6) (1992) 1473-1480.
- 459 [37] C. George, N. Kaplan, M. Main, *Adult Attachment Interview Protocol*, (3rd ed). University of  
460 California at Berkeley, 1996.
- 461 [38] G. Parker, J. Roussos, D. Hadzi-Pavlovic, P. Mitchell, K. Wilhelm, M.P. Austin, The development  
462 of a refined measure of dysfunctional parenting and assessment of its relevance in patients with  
463 affective disorders, *Psychol Med* 27 (5) (1997) 1193-1203.
- 464 [39] L.R. Derogatis, N. Melisaratos, The Brief Symptom Inventory: an introductory report, *Psychol Med*  
465 13 (3) (1983) 595-605.
- 466 [40] C. Imperatori, B. Farina, M. Adenzato, E.M. Valenti, C. Murgia, G.D. Marca, R. Brunetti, E.  
467 Fontana, R.B. Ardito, Default mode network alterations in individuals with high-trait-anxiety: An  
468 EEG functional connectivity study, *J Affect Disord* 246(2019) 611-618.
- 469 [41] G. Liotti, Trauma, dissociation and disorganized attachment: three strands of a single braid,  
470 *Psychother Theor Res Pract Train* 41(2004) 472-486.
- 471 [42] M.J. Bakermans-Kranenburg, M.H. Van IJzendoorn, A psychometric study of the Adult Attachment  
472 Interview: Reliability and discriminant validity, *Dev Psychol* 29 (5) (1993) 870-879.
- 473 [43] P. Ravitz, R. Maunder, J. Hunter, B. Sthankiya, W. Lancee, Adult attachment measures: a 25-year  
474 review, *J Psychosom Res* 69 (4) (2010) 419-432.
- 475 [44] M.D. Beijersbergen, M.J. Bakermans-Kranenburg, M.H. van Ijzendoorn, F. Juffer, Stress regulation  
476 in adolescents: physiological reactivity during the adult attachment interview and conflict  
477 interaction, *Child Dev* 79 (6) (2008) 1707-1720.

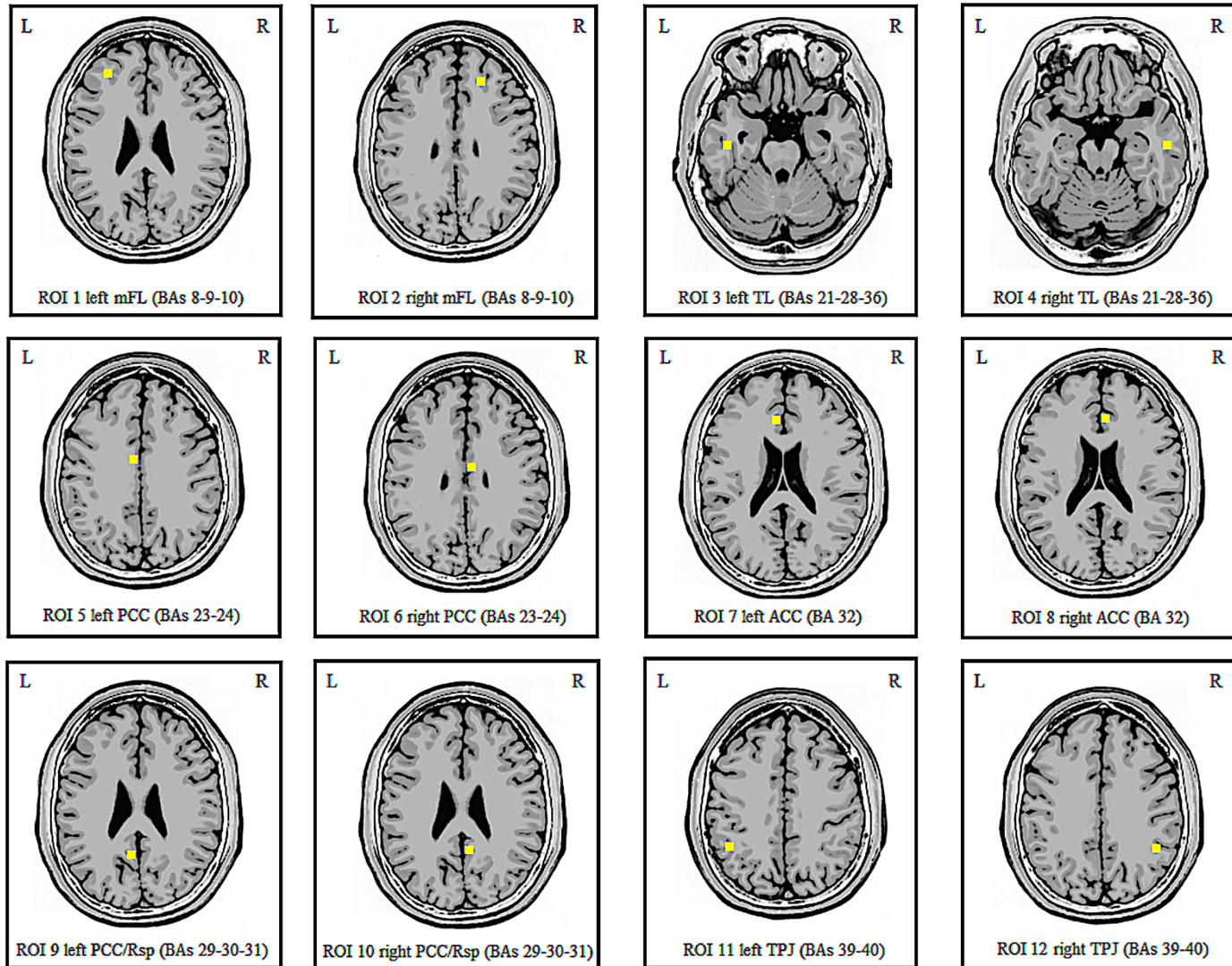
- 478 [45] G.I. Roisman, J.L. Tsai, K.H. Chiang, The emotional integration of childhood experience:  
479 physiological, facial expressive, and self-reported emotional response during the adult attachment  
480 interview, *Dev Psychol* 40 (5) (2004) 776-789.
- 481 [46] G. Parker, H. Tupling, L.B. Brown, A parental bonding instrument, *Br J Med Psychol* 52 (1) (1979)  
482 1-10.
- 483 [47] S. Beshai, P. Parmar, Trait mindfulness may buffer against the deleterious effects of childhood abuse  
484 in recurrent depression: A retrospective exploratory study, *Clin Psychol* 23 (1) (2018) 1-11.
- 485 [48] A. Picardi, L. Tarsitani, A. Toni, A. Maraone, V. Roselli, E. Fabi, F. De Michele, I. Gaviano, M.  
486 Biondi, Validity and reliability of the Italian version of the Measure of Parental Style (MOPS), *J*  
487 *Psychopathol* 19 (1) (2013) 54-59.
- 488 [49] F.L. Baeza, M.A. Caldieraro, D.O. Pinheiro, M.P. Fleck, Translation and cross-cultural adaptation  
489 into Brazilian Portuguese of the Measure of Parental Style (MOPS)--a self-reported scale--according  
490 to the International Society for Pharmacoeconomics and Outcomes Research (ISPOR)  
491 recommendations, *Rev Bras Psiquiatr* 32 (2) (2010) 159-163.
- 492 [50] L. Derogatis, *The SCL-90-R Manual*, Clinical Psychometric Research Unit. Johns Hopkins  
493 University School of Medicine, Baltimore, MD., 1977.
- 494 [51] L.R. Derogatis, K.L. Savitz, *The SCL-90-R, Brief Symptom Inventory, and Matching Clinical*  
495 *Rating Scales*. In: M.E. Maruish (Ed.), *The use of psychological testing for treatment planning and*  
496 *outcomes assessment* Lawrence Erlbaum Associates Publishers, Mahwah, NJ, US, 1999, pp. 679-  
497 724.
- 498 [52] D. De Leo, G.B. Frisoni, R. Rozzini, M. Trabucchi, Italian community norms for the Brief Symptom  
499 Inventory in the elderly, *Br J Clin Psychol* 32 ( Pt 2)(1993) 209-213.
- 500 [53] N.P. Castellanos, V.A. Makarov, Recovering EEG brain signals: artifact suppression with wavelet  
501 enhanced independent component analysis, *J Neurosci Methods* 158 (2) (2006) 300-312.
- 502 [54] X. Li, R. Ma, L. Pang, W. Lv, Y. Xie, Y. Chen, P. Zhang, J. Chen, Q. Wu, G. Cui, Y. Zhou, X.  
503 Zhang, Delta coherence in resting-state EEG predicts the reduction in cigarette craving after  
504 hypnotic aversion suggestions, *Sci Rep* 7 (1) (2017) 2430.
- 505 [55] M. Moazami-Gouadarzi, J. Sarnthein, L. Michels, R. Moukhtieva, D. Jeanmonod, Enhanced frontal  
506 low and high frequency power and synchronization in the resting EEG of parkinsonian patients,  
507 *Neuroimage* 41 (3) (2008) 985-997.
- 508 [56] J.W. Kam, A.R. Bolbecker, B.F. O'Donnell, W.P. Hetrick, C.A. Brenner, Resting state EEG power  
509 and coherence abnormalities in bipolar disorder and schizophrenia, *J Psychiatr Res* 47 (12) (2013)  
510 1893-1901.
- 511 [57] R.D. Pascual-Marqui, C.M. Michel, D. Lehmann, Low resolution electromagnetic tomography: a  
512 new method for localizing electrical activity in the brain, *Int J Psychophysiol* 18 (1) (1994) 49-65.
- 513 [58] R.W. Thatcher, D.M. North, C.J. Biver, LORETA EEG phase reset of the default mode network,  
514 *Front Hum Neurosci* 8(2014) 529.
- 515 [59] R.D. Pascual-Marqui, D. Lehmann, M. Koukkou, K. Kochi, P. Anderer, B. Saletu, H. Tanaka, K.  
516 Hirata, E.R. John, L. Prichep, R. Biscay-Lirio, T. Kinoshita, Assessing interactions in the brain with  
517 exact low-resolution electromagnetic tomography, *Philos Trans A Math Phys Eng Sci* 369 (1952)  
518 (2011) 3768-3784.

- 519 [60] C. Imperatori, G. Della Marca, N. Amoroso, G. Maestoso, E.M. Valenti, C. Massullo, G.A. Carbone,  
520 A. Contardi, B. Farina, Alpha/Theta Neurofeedback Increases Mentalization and Default Mode  
521 Network Connectivity in a Non-Clinical Sample, *Brain Topogr* 30 (6) (2017) 822-831.
- 522 [61] R.D. Pascual-Marqui, Review of Methods for Solving the EEG Inverse Problem, *Int J*  
523 *Bioelectromagn* 1 (1) (1999) 75-86.
- 524 [62] M. Pompili, M. Innamorati, Z. Rihmer, X. Gonda, G. Serafini, H. Akiskal, M. Amore, C. Niolu, L.  
525 Sher, R. Tatarelli, G. Perugi, P. Girardi, Cyclothymic-depressive-anxious temperament pattern is  
526 related to suicide risk in 346 patients with major mood disorders, *J Affect Disord* 136 (3) (2012)  
527 405-411.
- 528 [63] T.E. Nichols, A.P. Holmes, Nonparametric permutation tests for functional neuroimaging: a primer  
529 with examples, *Hum Brain Mapp* 15 (1) (2002) 1-25.
- 530 [64] R.L. Buckner, J.R. Andrews-Hanna, D.L. Schacter, The brain's default network: anatomy, function,  
531 and relevance to disease, *Ann N Y Acad Sci* 1124(2008) 1-38.
- 532 [65] M. Adenzato, M. Brambilla, R. Manenti, L. De Lucia, L. Trojano, S. Garofalo, I. Enrici, M. Cotelli,  
533 Gender differences in cognitive Theory of Mind revealed by transcranial direct current stimulation  
534 on medial prefrontal cortex, *Sci Rep* 7(2017) 41219.
- 535 [66] M. Tettamanti, M.M. Vaghi, B.G. Bara, S.F. Cappa, I. Enrici, M. Adenzato, Effective connectivity  
536 gateways to the Theory of Mind network in processing communicative intention, *Neuroimage*  
537 155(2017) 169-176.
- 538 [67] M. Adenzato, F. Garbarini, The *As If* in cognitive science, neuroscience and anthropology: a journey  
539 among robots, blacksmiths and neurons, *Theory Psychol* 16 (6) (2006) 747-759.
- 540 [68] A. Ciaramidaro, M. Adenzato, I. Enrici, S. Erk, L. Pia, B.G. Bara, H. Walter, The intentional  
541 network: how the brain reads varieties of intentions, *Neuropsychologia* 45 (13) (2007) 3105-3113.
- 542 [69] R.N. Spreng, R.A. Mar, A.S. Kim, The common neural basis of autobiographical memory,  
543 prospection, navigation, theory of mind, and the default mode: a quantitative meta-analysis, *J Cogn*  
544 *Neurosci* 21 (3) (2009) 489-510.
- 545 [70] A. Abu-Akel, S. Shamay-Tsoory, Neuroanatomical and neurochemical bases of theory of mind,  
546 *Neuropsychologia* 49 (11) (2011) 2971-2984.
- 547 [71] R. Leech, S. Kamourieh, C.F. Beckmann, D.J. Sharp, Fractionating the default mode network:  
548 distinct contributions of the ventral and dorsal posterior cingulate cortex to cognitive control, *J*  
549 *Neurosci* 31 (9) (2011) 3217-3224.
- 550 [72] B.A. Vogt, D.M. Finch, C.R. Olson, Functional heterogeneity in cingulate cortex: the anterior  
551 executive and posterior evaluative regions, *Cereb Cortex* 2 (6) (1992) 435-443.
- 552 [73] P. Fransson, G. Marrelec, The precuneus/posterior cingulate cortex plays a pivotal role in the default  
553 mode network: Evidence from a partial correlation network analysis, *Neuroimage* 42 (3) (2008)  
554 1178-1184.
- 555 [74] F. Miraglia, F. Vecchio, P. Bramanti, P.M. Rossini, Small-worldness characteristics and its gender  
556 relation in specific hemispheric networks, *Neuroscience* 310(2015) 1-11.

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559 **Legend to figure 1.** Axial view of the Default Mode Network regions of interest.



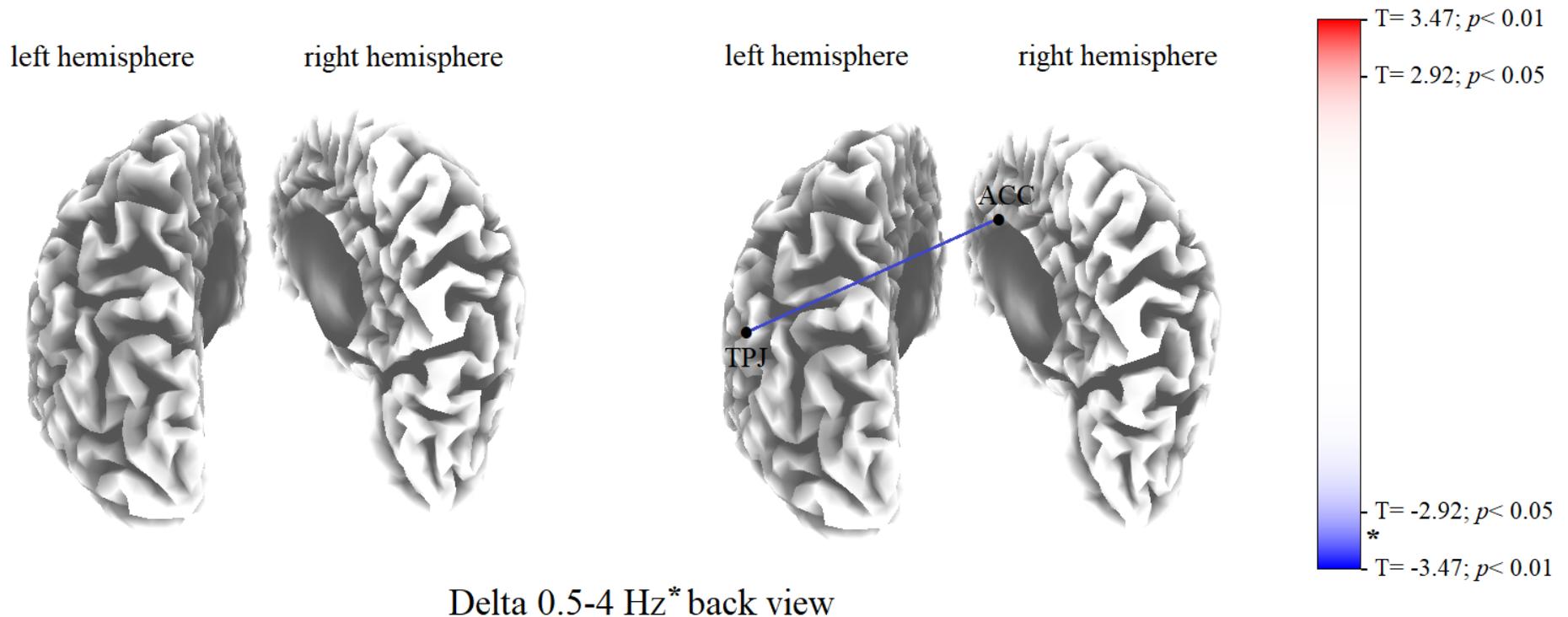
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561 Abbreviation:L= left; R= right; ROI= region of interest; mFL= medial Frontal Lobe; BA (Brodmann area); TL= Temporal Lobe; PCC= Posterior Cingulate cortex; ACC=  
562 Anterior Cingulate Cortex; PCC/Rsp= Posterior Cingulate/Retrosplenial cortex; TPJ= temporo-parietal junction

563 **Legend to figure 2.** Results of the eLORETA between comparisons of EEG functional connectivity in the delta bands. Panel A: PDP+ vs PDP- in  
 564 Pre-AAI condition; Panel B: PDP+ vs PDP- in Post-AAI condition. Blue lines indicate connections presenting a significant decrease of EEG  
 565 functional connectivity. Red lines would indicate increase of EEG functional connectivity (not present). Threshold values (T) for statistical  
 566 significance (corresponding to  $p < 0.05$  and  $p < 0.01$ ) are reported in the right side of the figure. In this comparison, significant modifications (\*) were  
 567 observed in the Post-AAI condition. Compared to PDP- individuals, PDP+ subjects showed a decrease of delta connectivity between left TPJ and  
 568 right ACC.  
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Panel A: Pre-AAI: PDP + vs PDP-

Panel B: Post-AAI: PDP + vs PDP-



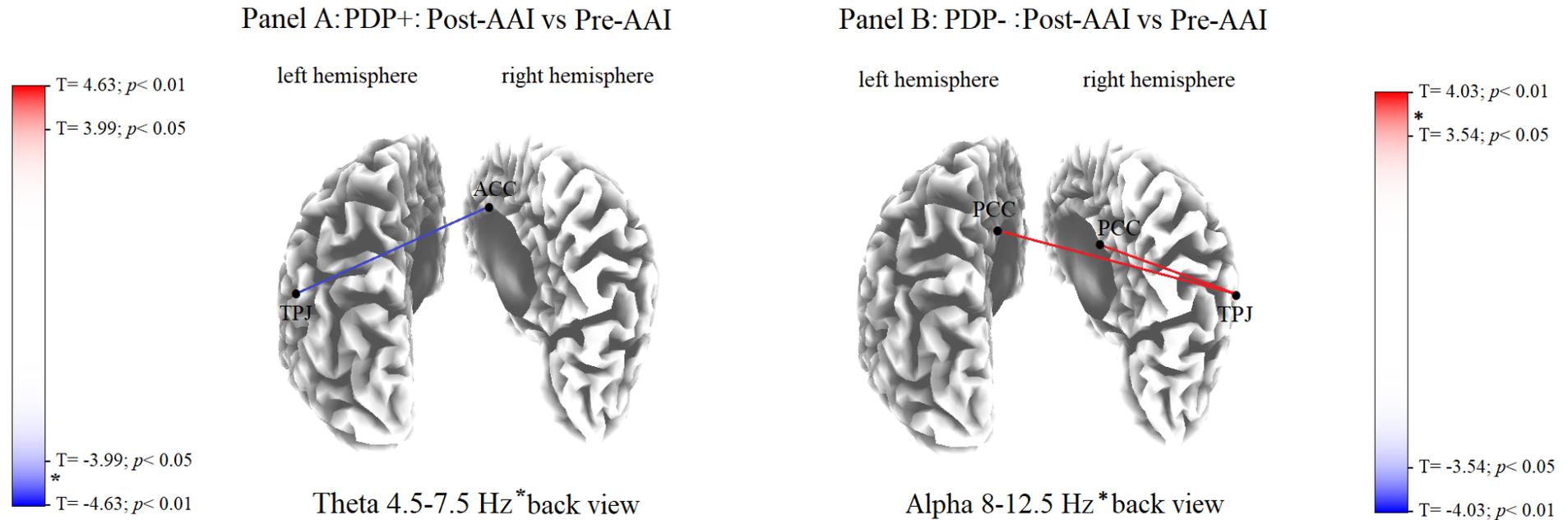
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Abbreviations: AAI= Adult Attachment Interview; PDP+= high perceived dysfunctional parenting group; PDP-= low perceived dysfunctional parenting group; TPJ= temporoparietal junction; ACC= anterior cingulate cortex

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**Legend to figure 3.**

Results of the eLORETA within comparisons (pre-AAI vs post-AAI) of EEG functional connectivity in PDP+ and PDP- group, respectively for theta and alpha band. Panel A: Post-AAI vs Pre-AAI for PDP+ group; Panel B: Post-AAI vs Pre-AAI for PDP- group. Blue lines indicate connections presenting a significant decrease of EEG functional connectivity. Red lines indicate connections presenting an increase of EEG functional connectivity. Threshold values (T) for statistical significance (corresponding to  $p < 0.05$  and  $p < 0.01$ ) are reported in the right and left side of the figure, respectively for PDP+ and PDP- group. In this comparisons, significant modifications were observed for both PDP+ (\*) and PDP- group (\*). Compared to Pre-AAI condition, PDP+ individuals showed in Post-AAI condition a decrease of theta connectivity between left TPJ and right ACC. Conversely, compared to Pre-AAI condition, PDP- individuals showed in Post-AAI condition an increase of alpha connectivity between right TPJ and both right and left PCC.



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**Abbreviations:**

AAI= Adult Attachment Interview; PDP+= high perceived dysfunctional parenting group; PDP-= low perceived dysfunctional parenting group; TPJ= temporoparietal junction; ACC= Anterior Cingulate Cortex; PCC= Posterior Cingulate Cortex

590 Table 1. **Bivariate analyses**

<b>Variables</b>	<b>Cluster 1 (PDP+) (N = 17)</b>	<b>Cluster 2 (PDP-) (N = 33)</b>	<b>Test Statistics</b>	<b>p =</b>
Age – M ± DS	22.59 ± 2.90	22.64 ± 2.16	U= 265	0.748
Educational level (years) – M ± SD	15.71 ± 1.65	15.44 ± 1.84	U= 249	0.624
Women - N (%)	13 (76.5)	23 (69.7)	$\chi^2_{1}= 0.26$	0.613
MOPS total score – M ± DS	40.65 ± 15.81	9.47 ± 6.32	U= 0.05	<b>&lt; 0.001</b>
MOPS sub-scales				
Maternal indifference – M ± DS	5.59 ± 5.72	0.94 ± 1.50	U= 89	<b>&lt; 0.001</b>
Maternal over-control – M ± DS	7.94 ± 2.79	3.39 ± 2.18	U= 57.5	<b>&lt; 0.001</b>
Maternal abuse – M ± DS	6.24 ± 4.63	0.94 ± 1.48	U= 57	<b>&lt; 0.001</b>
Paternal indifference – M ± DS	8.47 ± 5.63	1.13 ± 1.34	U= 43.5	<b>&lt; 0.001</b>
Paternal over-control – M ± DS	5.18 ± 2.38	2.53 ± 2.34	U= 113	<b>&lt; 0.001</b>
Paternal abuse – M ± DS	7.24 ± 3.82	0.44 ± 0.88	U= 22.5	<b>&lt; 0.001</b>
BSI-GSI – M ± DS	0.95 ± 0.66	0.56 ± 0.68	U= 143.5	<b>0.005</b>
Somatization – M ± DS	0.86 ± 0.72	0.44 ± 0.63	U= 172	<b>0.024</b>
Obsession-Compulsion – M ± DS	1.40 ± 0.86	0.81 ± 0.90	U= 153	<b>0.009</b>
Interpersonal Sensitivity – M ± DS	0.94 ± 0.88	0.57 ± 0.70	U= 203	0.106
Depression – M ± DS	0.98 ± 0.75	0.62 ± 0.79	U= 170	<b>0.023</b>
Anxiety – M ± DS	1.21 ± 0.82	0.75 ± 0.87	U= 168	<b>0.021</b>
Hostility – M ± DS	0.98 ± 0.99	0.46 ± 0.67	U= 186.5	<b>0.049</b>
Phobic Anxiety – M ± DS	0.47 ± 0.72	0.25 ± 0.61	U= 238	0.324
Paranoid Ideation – M ± DS	1.01 ± 0.75	0.49 ± 0.79	U= 138	<b>0.003</b>
Psychoticism – M ± DS	0.76 ± 0.71	0.48 ± 0.71	U= 194	0.071

Abbreviations:

PDP+= high perceived dysfunctional parenting group; PDP-= low perceived dysfunctional parenting group; SD = standard deviation; MOPS= Measure of Parental Style; BSI-GSI = Brief Symptom Inventory-Global Severity Index;

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**Table 2.** Values of Spearman’s *rho* correlation coefficient among EEG connectivity data, MOPS and BCSL-GSI scores in all sample (N = 50). Significant correlations are indicated by stars (\*).

	MOPS Total	Maternal indifference	Maternal over-control	Maternal abuse	Paternal indifference	Paternal over-control	Paternal abuse	GSI	Delta ROIs 11-8
MOPS total	-								
Maternal indifference	0.78**	-							
Maternal over-control	0.83**	0.57**	-						
Maternal abuse	0.81**	0.73**	0.72**	-					
Paternal indifference	0.79**	0.60**	0.52**	0.59**	-				
Paternal over-control	0.70**	0.38**	0.58**	0.41**	0.41**	-			
Paternal abuse	0.80**	0.51**	0.52**	0.64**	0.66**	0.60**	-		
GSI	0.55**	0.51**	0.46**	0.46**	0.42**	0.34*	0.36*	-	
Delta ROIs 11-8	-0.28*	-0.36*	-0.33*	-0.26	-0.13	-0.08	-0.22	-0.26	-

Abbreviations: ROIs= Regions of interest; MOPS= Measure of Parental Style; = Global Severity Index;  
\*  $p < 0.05$ ; \*\*  $p < 0.01$