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**Abnormal EEG Power Spectrum in Individuals with High Autistic Personality Traits: an eLORETA Study**

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18 **Abnormal EEG Power Spectrum in individuals with high autistic personality**  
19 **traits: an eLORETA study**

20

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50 **Abnormal EEG Power Spectrum in individuals with high autistic personality**  
51 **traits: an eLORETA study**

52

53 **Abstract**

54 **Aim:** Autistic traits lie on a continuously distributed spectrum ranging from non-clinical to clinical  
55 conditions. Indeed, autistic traits have been observed in general population at sub-threshold levels.  
56 Here, the main aim was to investigate differences in resting state (RS) electroencephalographic  
57 (EEG) power spectrum in individuals with high vs. low autistic traits.

58 **Methods:** Fifty undergraduates completed the Autism-Spectrum Quotient (AQ) and the Empathy  
59 Quotient (EQ). For each participant five minutes of RS-EEG were recorded and analysed by means  
60 of the exact Low-Resolution Electric Tomography software (eLORETA).

61 **Results:** A Two-Step Cluster Analysis revealed two groups: high autistic traits (AT+) and low  
62 autistic traits (AT-) group. Compared to AT-, AT+ individuals showed an increase of delta power  
63 in parietal/occipital and cortico-limbic areas. No alterations were observed in other frequency  
64 bands. Furthermore, both AQ and EQ total scores were positively correlated with delta EEG power  
65 after controlling for sex, age, and subclinical psychopathological traits.

66 **Conclusions:** Results show that AT+ individuals exhibit an increase in slow RS EEG power in  
67 regions involved in self-referential processes, suggesting a reduction in these internally directed  
68 mental activities and adding new evidence on the existence of a continuum in the autistic spectrum  
69 which spreads from clinical to non-clinical significance.

70

71

72 **Keywords:** Autism Spectrum Disorders; Autism Spectrum Quotient; Autistic traits; EEG Power  
73 Spectra; eLORETA; Empathy quotient; Personality; Psychopathology.

74

## 75 **1. Introduction**

76 Autism Spectrum Disorders (ASDs) are a set of heterogeneous highly heritable neurodevelopmental  
77 conditions characterized by repetitive behaviours and limited interests alongside a persistent  
78 impairment in social cognition (American Psychiatric Association 2013), including empathy  
79 domain (Baron-Cohen, 1995). Although from a clinical point of view it is necessary to meet certain  
80 criteria to receive a diagnosis of ASDs (American Psychiatric Association 2013), some evidences  
81 suggest that autistic traits could also be observed at sub-threshold levels in general population  
82 (Constantino and Todd 2003, 2005; Posserud et al. 2006). These traits are considered highly stable  
83 (Robinson et al. 2011), especially in males (Whitehouse et al. 2011). Furthermore, it has been  
84 proposed ( Abu-Akel et al. 2019; Wakabayashi et al. 2006), that in the general population, autistic  
85 traits are independent of the big five personality dimensions and may represent a sixth factor of  
86 personality (i.e., individual differences in autism-related domains such as communication, social  
87 skills, and attention to details). These evidences suggest that autism could be considered a condition  
88 extending along a continuum, which spreads from clinical significance to non-clinical individual  
89 difference (Constantino and Todd 2003; Robinson et al. 2016). In fact, as reported (Skuse et al.  
90 2009; Spiker et al. 2002), autistic traits show a graded nature in which ASDs represent the extreme  
91 condition and in which non-clinical individuals and clinically affected individuals are related to the  
92 same genetic susceptibilities (Lundstrom et al. 2012).

93 From a neurobiological point of view, several studies have shown functional (Iidaka et al.  
94 2019; Pierce et al. 2001) and structural abnormalities in subjects with ASDs (Sparks et al. 2002),  
95 including alterations in social cognition-related brain areas (e.g., fusiform face area and amygdala)  
96 (Pinkham et al. 2008). Moreover, functional Magnetic Resonance Imaging (fMRI) studies suggest  
97 that, compared to controls, patients with ASDs show an absent or a diminished activation in task-  
98 related regions (e.g., fusiform face area) in favour of an enhanced activity in task-unrelated areas  
99 (e.g., primary visual cortex) during perceptual and sensorimotor tasks (e.g. visually guided saccade  
100 task, face/shape perception tasks; Pierce et al. 2001; Takarae et al. 2007). Furthermore, task-free

101 condition studies in individuals with ASDs reported functional alterations in Resting-State (RS)  
102 related networks, particularly the Default Mode Network (DMN) (Cherkassky et al. 2006; Yerys et  
103 al. 2015). Lastly, quantitative electroencephalographic (qEEG) studies (i.e., numerical computations  
104 of EEG parameters) showed several neurophysiological alterations in individuals with ASDs during  
105 RS, such as higher power both in slow (mainly delta and theta) (Chan et al. 2007; Pop-Jordanova et  
106 al. 2010) and high frequency bands (i.e., beta and gamma; Murias et al. 2007; Orekhova et al.  
107 2007).

108 Although a large body of literature have explored the neural correlates of ASDs in the  
109 clinical samples (Iidaka et al. 2019; Pierce et al. 2001; Sparks et al. 2002), few studies have focused  
110 on non-clinical subjects with high autistic personality traits (von dem Hagen et al. 2011).  
111 Investigating the association between neurophysiological alterations and autistic traits in non-  
112 clinical samples is considered an important research topic in order to understand the neural  
113 correlates of ASDs within a dimensional perspective (Barttfeld et al. 2013).

114 Von dem Hagen et al. (2011) reported in subjects with high autistic traits structural and  
115 functional alterations in the posterior superior temporal sulcus (a brain region involved in  
116 processing social stimuli), which is known to be also altered in individuals with ASDs (Shih et al.  
117 2011). Alterations in cortical motor areas (e.g., supplementary motor area, motor and pre-motor  
118 cortex) have also been observed in a qEEG study performed during action observation task in  
119 individuals with high autistic traits (Puzzo et al. 2010).

120 Although RS EEG is a suitable technique to explore ASD-related abnormalities at different  
121 ages and all over the spectrum (Wang et al. 2013), up to now, qEEG correlates during RS in non-  
122 clinical individuals with high personality autistic traits have been rarely investigated (Barttfeld et al.  
123 2013; Carter Leno et al. 2018; Moore and Franz 2017). Barttfeld et al. (2013) documented a  
124 significant negative association between EEG functional connectivity (mainly in delta and theta  
125 bands) and increased levels of autistic traits. Carter Leno et al. (2018) reported a positive correlation  
126 between behavioural rigidity autistic trait and absolute alpha power in the parietal scalp region.

127 Lastly, Moore et al. (2017), in a within EEG study design, showed that autistic traits were positively  
128 associated with the difference between eyes-closed RS and eyes-open RS in the distribution of  
129 alpha and beta power in the parietal areas (i.e., high levels of autistic traits predicted a greater  
130 tendency for both alpha and beta power to be lower during eyes-open RS compared to eyes-closed  
131 RS).

132 The RS qEEG differences between individuals with high and low autistic personality traits  
133 have been still rarely investigated. Furthermore, it is possible that the relation between autistic traits  
134 and qEEG data found by previous studies is partially mediated by other potential variables (e.g., sex  
135 and general psychopathology). Therefore, the main aim of the present study was to extend previous  
136 studies, investigating differences in RS EEG power spectrum in individuals with high vs. low  
137 autistic personality traits, using the exact Low Resolution Electric Tomography software  
138 (eLORETA) a validated tool for localizing electrical brain activity (Pascual-Marqui et al. 2011).  
139 Furthermore, another aim was to investigate the association between qEEG data and autistic traits  
140 controlling for sex, age and psychopathology.

141

## 142 **2. Materials and methods**

### 143 *2.1 Participants*

144 Participants were 50 students (thirty-six women) enrolled through advertisements posted in the  
145 university. Enrolment procedures lasted from November 2017 to April 2018. The following  
146 inclusion criterion was considered: age between 18 and 30 years. Exclusion criteria were: left-  
147 handedness; head trauma; diagnosis or a history of major psychiatric disorders; history of  
148 neurological diseases and the previous central nervous system active drugs intake in the last two  
149 weeks before the assessment. A checklist with dichotomous items was used to assess  
150 inclusion/exclusion criteria and socio-demographic data. All participants received information  
151 concerning the aims and the procedures of the study, all of them subscribed the informed consent  
152 which was approved by the local ethics committee according to the Helsinki Declaration standards.

153

## 154 2.2 Questionnaires

155 All participants completed the Autism-Spectrum Quotient (AQ; Baron-Cohen et al. 2001b), the  
156 Empathy Quotient (EQ; Baron-Cohen and Wheelwright 2004) and the Brief Symptom Checklist  
157 (BSI; Derogatis and Melisaratos 1983).

158

159 2.2.1 *The Autism-Spectrum Quotient (AQ)*. The AQ (Baron-Cohen et al. 2001b) is a widely used  
160 self-report to assess autistic traits in adult population. It is composed of 50 items divided into 5  
161 subscales of 10 items (rated on a 4-point scale from “Definitely agree” to “Definitely disagree”), in  
162 which each subscale concerns a psychological domain implicated in ASDs (e.g., detail-oriented  
163 attention). The AQ also provides a global score, with higher values reflecting higher levels of  
164 autistic traits. From a psychometric point of view, the AQ has shown both good internal consistency  
165 and test-retest reliability (Baron-Cohen et al. 2001b). Furthermore, the AQ scores predict  
166 performance in tasks involving ASDs impaired cognitive function, both in clinical and in general  
167 population (Baron-Cohen et al. 2001a; Grinter et al. 2009). In the current study the Italian version  
168 of AQ (Ruta et al. 2012) has been used and AQ total score has been considered. In the present study  
169 the Cronbach’s  $\alpha$  was 0.84 for the AQ total score.

170

171 2.2.2 *The Empathy Quotient (EQ)*. The EQ (Baron-Cohen and Wheelwright 2004) is a forced-  
172 choice self-report questionnaire developed for people with normal intelligence used both in clinical  
173 and in general adult population. EQ is composed by 20 filler items and 40 items that assess  
174 affective, behavioural and cognitive empathic features (Baron-Cohen and Wheelwright 2004). The  
175 EQ also provides a global score designed to assess the overall levels of empathy with higher values  
176 reflecting higher levels of empathy. The EQ is often used in combination with the AQ due to their  
177 inverse correlation and this aspect could be considered an important sign of EQ validity (Baron-  
178 Cohen and Wheelwright 2004). Indeed, due to the centrality of this deficit in detecting others’



179 mental states, autism has also been conceptualized as an empathy disorder (Yirmiya et al. 1992;  
180 Baron-Cohen and Wheelwright 2004; Gillberg 1992). Satisfactory psychometric properties have  
181 been reported in the original validation study (Baron-Cohen and Wheelwright 2004) and in the  
182 Italian version (Preti et al. 2011) which was used in the present study. Good internal consistency for  
183 the EQ total score (Cronbach's  $\alpha=0.89$ ) was also observed in the current sample.

184

185 *2.2.3 The Brief Symptom Inventory (BSI).* The BSI is a widely used 53-item self-report which  
186 assesses psychopathology both in clinical and in general population, measuring several  
187 symptomatologic dimensions: somatization (SOM), obsessive-compulsive symptoms (OC),  
188 interpersonal sensitivity (IS), depression (DEP), anxiety (ANX), hostility (HOS), phobic anxiety  
189 (PHOB), paranoid ideation (PAR) and psychoticism (PSYC). The BSI provides some distress  
190 indices such as the Global Severity Index (GSI) which assesses the general level of  
191 psychopathology. In the present study, the Italian version of the BSI (De Leo et al. 1993) has been  
192 used and the Cronbach's  $\alpha$  for the GSI was 0.94.

193

### 194 *2.3 EEG recordings and analysis*

195 EEG recordings were performed in RS condition. In order to get RS condition, each subject  
196 was invited to sit in a comfortable armchair in a semi-darkened room in an EEG laboratory.  
197 Recordings were acquired for 5 minutes consecutively and subjects were asked to keep their eyes  
198 closed during the whole recording session. RS recordings were obtained for all subjects. All EEG  
199 recordings were visually evaluated and no significant alterations of background rhythm frequency  
200 (including evidence of drowsiness or sleep during the recordings) have been detected.

201 Micromed System Plus digital EEGraph (Micromed© S.p.A., Mogliano Veneto, TV, Italy)  
202 was used to obtain all EEG recordings. EEG signals were obtained by 31 standard scalp leads  
203 located conforming to the 10–20 system at the following recording sites: Fp1, AF3, F3, FC1, C3,  
204 CP1, P3, PO3, O1, F7, FC5, T7/T3, CP5, T5, Fz, Cz, Pz, Fp2, AF4, F4, FC2, C4, CP2, P4, PO4,

205 O2, F8, FC6, T8/T4, CP6, T6. Furthermore, in the current study, Electrooculography (EOG) and  
206 Electrocardiography (ECG) signals have been considered. Details about EEG recordings could be  
207 found elsewhere (Imperatorii et al. 2013; Imperatorii et al. 2016; Imperatorii et al. 2019c). Briefly, the  
208 reference electrodes were placed on right mastoid during the recording session and then were re-  
209 referenced offline to the algebraic average of the left and right mastoids. Impedances were kept  
210 below 5 k $\Omega$  before starting EEG recording and checked again at the end of EEG session for each  
211 participants. In particular, impedances of the right and left mastoid reference electrodes were  
212 checked to be identical. Sampling frequency was 256 Hz; A/D conversion was made at 16 bit;  
213 preamplifiers amplitude range was  $\pm 3,200 \mu\text{V}$  and low-frequency pre-filters were set at 0.15 Hz.

214 According to previous studies ( Adenzato et al. 2019a; Imperatorii et al. 2019a; Imperatorii et  
215 al. 2019b) artifact rejection procedure was performed visually and through the independent  
216 component analysis (ICA). Specifically, EEG intervals containing artifacts were first rejected by  
217 inspection. The remaining EEG was then subjected to ICA, and non-cerebral artifacts such as eye  
218 movements, cardiac pulses and muscular activations were corrected by removal of non-neural  
219 independent components.

220 At least 180 seconds of artifact-free EEG recording (not necessarily consecutive) were  
221 selected and analysed for each participant using the eLORETA software, a well validated  
222 instrument to localize brain electrical activity, characterized by satisfactory localization agreement  
223 with other neuro-imaging techniques (De Ridder et al. 2011; Horacek et al. 2007; Huang et al.  
224 2018; Kirino 2017; Muller et al. 2005; Pizzagalli et al. 2004; Zumsteg et al. 2005), also when low  
225 number of electrodes were used (i.e.,  $< 30$ ).

226 Power spectral analysis was performed on EEG artifact-free 2-sec epochs, using Fast Fourier  
227 Transform algorithm and boxcar windowing. The following frequency bands were considered: delta  
228 (0.5–4 Hz), theta (4.5–7.5 Hz), alpha (8–13 Hz), beta (13.5–30 Hz), and gamma (30.5–60 Hz)  
229 frequency bands were investigated. EEG frequency analysis was performed using monopolar EEG  
230 traces: each electrode referred to joint mastoids (i.e., A1 + A2). Topographic sources of EEG

231 activities were determined using the eLORETA, which estimates 3-dimensional current distribution  
232 throughout the brain volume by assuming that connected neurons are activated both simultaneously  
233 and synchronously (Kreiter and Singer 1992; Murphy et al. 1992). The computational task is to  
234 select the smoothest 3-dimensional current distribution (Grave de Peralta-Menendez and Gonzalez-  
235 Andino 1998; Grave de Peralta Menendez et al. 2000), providing a true 3-dimensional tomography,  
236 in which the localization of brain signals is conserved with a low amount of dispersion (Pascual-  
237 Marqui et al. 2011). The eLORETA solution space was limited to the cortical gray matter, including  
238 6239 voxels of 5 cubic mm spatial resolution.

239

#### 240 *2.4 Statistical analysis*

241 A Two Step Cluster Analysis procedure was performed to detect groups of subjects with low and  
242 high levels of autistic traits based on both AQ and EQ total scores. The Schwarz's Bayesian  
243 Criterion (BIC) was used to compare each cluster solution and select the best one. To explore qEEG  
244 differences between clusters, Mann-Whitney's U test was used for dimensional variables and Chi-  
245 squared ( $\chi^2$ ) test for dichotomous ones.

246 The power spectrum analysis was performed comparing clusters for each frequency band by  
247 means of the statistical nonparametric mapping (SnPM) methodology supplied by the eLORETA  
248 software (Nichols and Holmes 2002) . This eLORETA procedure provides empirical t values (both  
249 for a significance of  $p < 0.01$  and  $p < 0.05$ ) for all comparisons (Friston et al. 1991).

250 Lastly, partial correlations were performed among any significant qEEG data observed in  
251 the between comparisons and both AQ and EQ total scores, controlling for age, sex and  
252 psychopathology, which are known to be associated to ASDs (Baio et al. 2018; Hsiao et al. 2013;  
253 Lever and Geurts 2016). Cluster and correlational analysis were carried out using the IBM SPSS  
254 Statistics 18 software.

255

### 256 **3. Results**

257 AQ total score was negatively correlated with EQ total score ( $r = -0.53$ ;  $p < 0.001$ ; Figure 1). The  
258 Two Step Cluster Analysis procedure indicated a 2-group solution (BIC change =  $-10.09$ ; Ratio of  
259 distance measures = 2.19). 56.0% of the sample ( $N = 28$ ) was included in cluster #1, and 44% ( $N =$   
260 22) was included in cluster #2. Compared to participants included in cluster #2, those included in  
261 cluster #1 had significantly higher AQ total score and lower EQ total score (Table 1). Therefore,  
262 cluster #1 was mainly represented by subjects with higher level of autistic traits (AT+ group)  
263 compared to participants included in cluster #2 (AT- group). Groups did not differ in age and sex.  
264 However, compared to AT- subjects, AT+ individuals had higher scores in the GSI and in OC, IS,  
265 HOST, PAR, and PSYC dimensions.

266

### 267 *3.1 Power spectrum analysis*

268 Two-tailed significance thresholds were  $t = \pm 3.852$  for  $p < 0.05$  and  $t = \pm 4.386$  for  $p < 0.01$ . A  
269 significant modification in delta frequency band was detected by the eLORETA software. As shown  
270 in figure 2, compared to AT- participants, AT+ individuals showed an increase in delta frequency  
271 band in occipital, cortico-limbic and parietal areas. These alterations were localized by the  
272 eLORETA software in the following brain regions: cuneus [Brodmann Area (BA) 17 and BA 18],  
273 precuneus (BA 7 and BA 31), lingual gyrus (BA 17), middle occipital gyrus (BA 19) and posterior  
274 cingulate cortex (BA 23 and BA 30). Detailed information about BAs, coordinates, t values and  
275 statistical significance was reported in Table 2. No significant differences have been detected by  
276 eLORETA software in alpha, beta and gamma frequency bands.

277

### 278 *3.2 Correlations among both AQ and EQ total scores and EEG power spectra*

279 As reported in Table 3, all significant EEG delta power spectra data observed in the between  
280 comparisons (except for BA 18) were positively associated with AQ total scores ( $r_p > 0.31$ ,  $p < 0.05$ )  
281 after controlling for age, sex and GSI. All EEG delta power spectra data were also negatively  
282 related to EQ total scores ( $r_p > 0.41$ ,  $p < 0.05$ ) after controlling for age, sex and GSI.

283

#### 284 **4. Discussion**

285 The main aim of the current study was to investigate differences in RS EEG power spectrum  
286 between individuals with high vs. low autistic personality traits. A two Step Cluster Analysis  
287 procedure detected two groups of subjects with, respectively, high and low autistic traits.

288 Our results showed that, the two groups were different both on psychopathological and  
289 neurophysiological point of view. Compared to AT<sup>-</sup>, AT<sup>+</sup> individuals reported more  
290 psychopathological symptoms, especially in those dimensions investigating schizotypal traits (e.g.,  
291 paranoid ideation and psychoticism). These is accordance with previous studies suggesting that  
292 autistic traits overlap with schizotypal traits (Dinsdale et al. 2013; Ford and Crewther 2014; Ford et  
293 al. 2018; Mealey et al. 2014), particularly with negative schizotypal traits (Mealey et al. 2014).

294 On the neurophysiological point of view, our results showed that, compared to AT<sup>-</sup>, AT<sup>+</sup>  
295 individuals exhibit an increase in delta power in parietal, occipital and cortico-limbic areas (i.e.,  
296 cuneus, precuneus, middle occipital gyrus, posterior cingulate cortex and lingual gyrus). Controlling  
297 for sex, age and GSI, increased delta activity in these areas was significantly correlated with both  
298 AQ and EQ total scores.

299 Our results are consistent with previous literature showing alterations in slow frequency  
300 bands in subjects with both ASDs (for a review see Wang et al. 2013) and in subjects with high  
301 autistic traits (Barttfeld et al. 2013). No differences have been observed in other frequency bands,  
302 such as alpha and beta, which have been previously reported to be associated with autistic traits in  
303 non-clinical samples (Carter Leno et al. 2018; Moore and Franz 2017).

304 Our study showed that delta alterations were mainly localized in posterior midline structures  
305 including precuneus and PCC which are known to be functionally and structurally altered both in  
306 autism ( Kennedy et al. 2006; Lynch et al. 2013; Oblak et al. 2011) and in neurotypical adults with  
307 autistic traits (Jakab et al. 2013). These brain areas are involved in several critical cognitive  
308 functions, such as self-referential processing and mentalization (Andrews-Hanna et al. 2012;

309 Andrews-Hanna et al. 2010; Ciaramidaro et al. 2007; Tettamanti et al. 2017; Vicari and Adenzato,  
310 2016), which are known to be impaired in ASDs ( Bara et al. 2011; Lombardo et al. 2007).  
311 Coherently, posterior midline structures' EEG power alterations detected in the current study were  
312 localized in the right hemisphere, which is involved in self-related processes as self-recognition  
313 (Keenan et al. 2001). Furthermore, several studies reported right-hemisphere alterations (e.g., right  
314 hemisphere engagement when not necessary) in individuals with ASDs (Mason et al. 2008;  
315 Orekhova et al. 2009). It is also known that PCC and precuneus are crucial hubs of the DMN  
316 (Buckner et al. 2008; Greicius et al. 2003), a set of brain areas mostly active during RS. The DMN  
317 is a brain network involved in auto-referential processes, including mentalizing and  
318 autobiographical memory retrieval (Buckner et al. 2008; Greicius et al. 2003), and several studies  
319 reported its alteration in ASDs (Jung et al. 2014; Kennedy et al. 2006; Weng et al. 2010).  
320 Coherently, it has been reported that delta frequency band shows a negative correlation with DMN  
321 activity (Hlinka et al. 2010). Therefore, in line with previous data, it is possible to speculate that the  
322 increase of delta power in midline structures that we have detected in individuals with high autistic  
323 personality traits could reflect poor self-referential processes during RS condition similarly to what  
324 observed in individuals with ASDs.

325         We consider this finding as a convergent evidence, at a neurophysiological level, on the  
326 existence of a continuum in the autistic spectrum which spreads from clinical significance (the  
327 ASDs condition) to non-clinical one (the AT+ condition here investigated). Furthermore, our  
328 findings are in line with recent psychiatry research paradigms (Cauvet et al. 2019) recommending  
329 dimensional constructs to study behaviors from typical to atypical (RDoC - Research Domain  
330 Criteria, NIH).

331         In the current study there should be considered some limitations in generalizing results.  
332 Firstly, the sample was composed for the majority by women and it is known that sex differences  
333 could be observed in mentalizing abilities (Adenzato et al. 2017; Adenzato et al., 2019b) and in  
334 EEG pattern during RS condition (Miraglia et al. 2015). Secondly, although the eLORETA is

335 considered a validated tool for localizing the sources of electrical activity in cortico-limbic brain  
336 areas, we use scalp EEG recordings, which have an intrinsic limit in space resolution, mainly in the  
337 identification of deep subcortical sources (e.g., the cerebellum). Furthermore, we did not include a  
338 clinical group of patients with ASDs in order to perform a comparison with both AT+ and AT-  
339 participants that could have provide further important theoretical information according to the ASD  
340 dimensional perspective. Lastly, we considered only self-report measures which could be  
341 influenced by psychological aspects like social desirability (Arnold and Feldman 1981). Replication  
342 studies with other methods of data collection (e.g. cognitive/emotional tasks), according to a multi-  
343 method approach (Lauriola et al. 2011), as well as with the enrolment of clinical samples are  
344 welcome and would strengthen the interpretation of the current results.

345 Notwithstanding these limitations, to the best of our knowledge, this is the first study  
346 examining the differences of EEG power spectrum between individuals with high vs. low autistic  
347 personality traits during RS, using a well validated tool to localize brain electrical activity.

348 In conclusion, the current study has added new evidence that individuals with high autistic  
349 traits have similar neurophysiological alterations to those observed in individuals with ASDs  
350 supporting the hypothesis of an autism spectrum ranging from non-clinical to clinical conditions  
351 (Abu-Akel et al. 2019; Austin 2005; Ruzich et al. 2015). Examining the association between  
352 neurophysiological correlates and autistic traits in non-clinical samples is considered an important  
353 research topic in order to understand the neural underpinnings of ASDs within a dimensional  
354 perspective (Barttfeld et al. 2013). Future researches are still needed in order to identify specific  
355 neural patterns distinguishing autistic traits at different stages of the continuum, including ASDs  
356 which represents the extreme condition.

357

358 **Declarations of interest:** none

359

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644 **Tables**645 **Table 1.** Bivariate analysis of the clusters.

	AT- (n= 22)	AT + (n=28)	Test Statistics	p =
Age – M ± SD	22.50±2.20	22.71±2.59	U= 300	0.87
Males – N (%)	5 (22.73%)	9 (32.14%)	$\chi^2=0.542$	0.46
EQ Total Score – M ± SD	54.55±6.57	37.29±8.79	U= 21	<b>&lt;0.001</b>
AQ Total Score – M ± SD	11.09±2.97	18.11±5.96	U= 82.5	<b>&lt;0.001</b>
GSI – M ± SD	0.44±0.36	0.89±0.82	U= 202.5	<b>0.04</b>
SOM– M ± SD	0.38±0.48	0.74±0.78	U=223.5	0.09
OC– M ± SD	0.61±0.54	1.33±1.04	U=181	<b>0.01</b>
IS– M ± SD	0.43±0.60	0.90±0.85	U=188	<b>0.02</b>
DEP– M ± SD	0.53±0.59	0.90±0.90	U=223.5	0.10
ANX– M ± SD	0.67±0.51	1.20±1.05	U=260	0.35
HOST– M ± SD	0.29±0.38	0.91±0.97	U=174	<b>0.01</b>
PHOB– M ± SD	0.15±0.33	0.46±0.80	U=246.5	0.17
PAR– M ± SD	0.42±0.58	0.86±0.91	U=210	<b>0.05</b>
PSYC– M ± SD	0.33±0.42	0.78±0.84	U=200	<b>0.03</b>

Abbreviations: AQ (Autistic Quotient); AT+ (high autistic traits group); AT- (low autistic traits group); EQ (Empathy Quotient); GSI (General Severity Index); U (Mann- Whitney Test U),  $\chi^2$  (Chi-squared Test); SOM (somatization); OC (obsessive-compulsive); IS (interpersonal sensitivity); DEP (depression); ANX (anxiety); HOST (hostility); PHOB (phobic anxiety); PAR (paranoid ideation); PSYC (psychoticism).

646

647 **Table 2.** eLORETA statistical values for delta frequency band in AT+ group compared to AT-  
648 group.

BA	Structure	Delta (0.5-4 Hz)		p <
		MNI Coordinates (X, Y, Z)	Voxel Value t	
7	Precuneus	10, -65, 30	4.54	0.01
17	Lingual Gyrus	20, -95, -5	4.44	0.01
17	Cuneus	10, -95, 0	4.44	0.01
18	Cuneus	10, -75, 20	4.57	0.01
19	Middle Occipital Gyrus	30, -80, 15	4.52	0.01
23	Posterior Cingulate	5, -60, 15	4.59	0.01
30	Posterior Cingulate	5, -65, 15	4.61	0.01
31	Precuneus	10, -65, 20	4.64	0.01

Abbreviations: BA (Brodmann Area); eLORETA (exact Low-Resolution Electric Tomography software); MNI (Montreal Neurological Institute).

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651 **Table 3.** Partial correlations in total sample (n=50) among all significant Brodmann Areas observed  
 652 in the between comparisons and both AQ and EQ total Score, controlled for GSI, sex and age.

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		<b>Partial Correlations</b>							
		<b>Delta</b>							
		<b>(0.5-4 Hz)</b>							
<b>Control Variables</b>		<b>BA 7</b> <i>10, -65, 30</i>	<b>BA 17</b> <i>20, -95, -5</i>	<b>BA 17</b> <i>15, -95, 0</i>	<b>BA 19</b> <i>30, -80, 15</i>	<b>BA 18</b> <i>10, -75, 20</i>	<b>BA 23</b> <i>5, -60, 15</i>	<b>BA 30</b> <i>5, -65, 15</i>	<b>BA 31</b> <i>10, -65, 20</i>
<b>GSI, sex &amp; age</b>	<b>AQ TOT</b>	0.35*	0.32*	0.31*	0.33*	0.24	0.33*	0.32*	0.33*
	<b>EQ TOT</b>	-0.43**	-0.44**	-0.44**	-0.45**	-0.37**	-0.43**	-0.44**	-0.46**

Abbreviations: AQ TOT (Autism Quotient Questionnaire Total Score), BA (Brodmann Area), EQ TOT (Empathy Quotient Total Score), GSI (General Severity Index). Note: two tailed significance \*( $p < 0.05$ ), \*\*( $p < 0.01$ ).

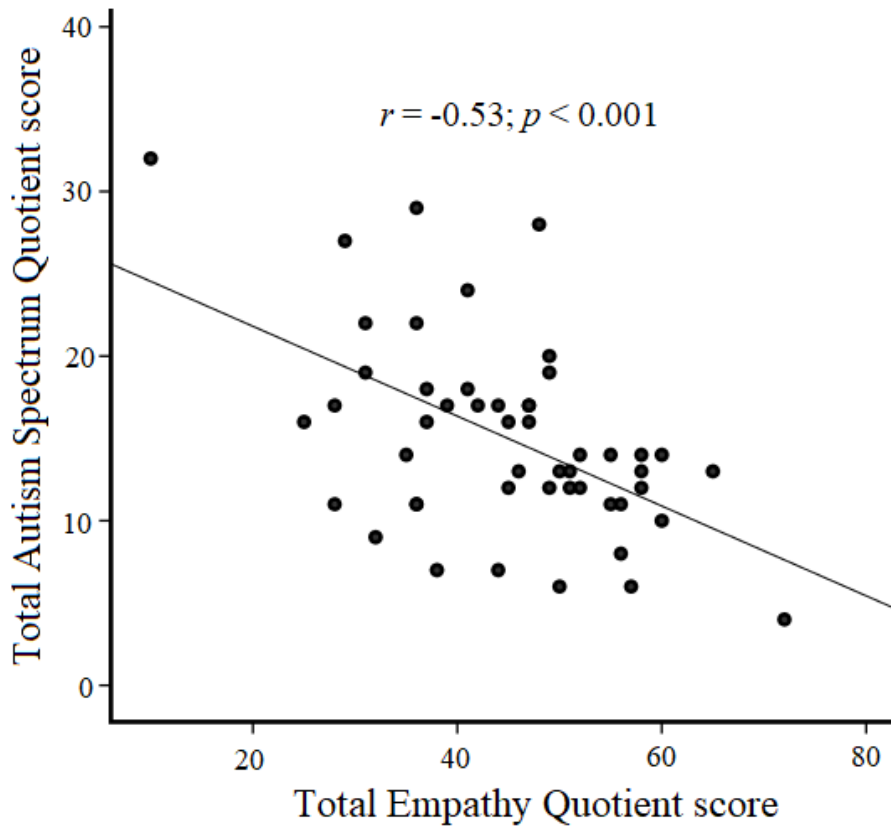
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656 **Figures**

657 **Figure 1.** Scatterplot of the correlation between AQ and EQ total score.

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661 **Figure 2.** EEG power spectra results from the comparison between groups (AT+ group versus AT-  
 662 group) in delta, theta, alpha, beta and gamma frequency bands. Compared to AT- participants, AT+  
 663 individuals showed a significant increase of delta power in the following Brodmann Areas: 7, 17,  
 664 18, 19, 23, 30, 31.

$t = +4.386$   $t = +3.852$   
 $**p < 0.01$   $p < 0.05$

$t = -3.852$   $t = -4.386$   
 $p < 0.05$   $p < 0.01$

