Cortical deficits of emotional face processing in adults with ADHD: Its relation to social cognition and executive function

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Although it has been shown that adults with attention-deficit hyperactivity disorder (ADHD) have impaired social cognition, no previous study has reported the brain correlates of face valence processing. This study looked for behavioral, neuropsychological, and electrophysiological markers of emotion processing for faces (N170) in adult ADHD compared to controls matched by age, gender, educational level, and handedness. We designed an event-related potential (ERP) study based on a dual valence task (DVT), in which faces and words were presented to test the effects of stimulus type (faces, words, or face-word stimuli) and valence (positive versus negative). Individual signatures of cognitive functioning in participants with ADHD and controls were assessed with a comprehensive neuropsychological evaluation, including executive functioning (EF) and theory of mind (ToM). Compared to controls, the adult ADHD group showed deficits in N170 emotion modulation for facial stimuli. These N170 impairments were observed in the absence of any deficit in facial structural processing, suggesting a specific ADHD impairment in early facial emotion modulation. The cortical current density mapping of N170 yielded a main neural source of N170 at posterior section of fusiform gyrus (maximum at left hemisphere for words and right hemisphere for faces and simultaneous stimuli). Neural generators of N170 ( fusiform gyrus) were reduced in ADHD. In those patients, N170 emotion processing was associated with performance on an emotional inference ToM task, and N170 from simultaneous stimuli was associated with EF, especially working memory. This is the first report to reveal an adult ADHD-specific impairment in the cortical modulation of emotion for faces and an association between N170 cortical measures and ToM and EF.

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Attention-deficit hyperactivity disorder (ADHD) is a neuropsychiatric condition with onset in childhood that extends over adolescent and adult life with a considerable symptomatic burden and functional impairment (Malloy-Diniz, Fuentes, Leite, Correa, & Bechara, 2007). Its medical profile includes problems of self-regulation and self-motivation, distractibility, procrastination, and prioritization (Barkley, 2001, 2010; Safren, 2006). A recent meta-analysis suggested that the prevalence of adult ADHD is currently underestimated (Simon, Czobor, Balint, Meszaros, & Bitter, 2009). It has been shown that adults with ADHD have impaired social cognition (Uekermann et al., 2010). However, no studies have focused on the brain correlates of the adult ADHD deficits in emotion processing. The aim of this study is to identify cortical markers of emotion processing in adult ADHD and explore their relation to individual neuropsychological profiles.

ADHD in childhood is more related to hyperactivity and impulsiveness, whereas in adulthood it presents a different profile, with fewer externalizing symptoms and a higher rate of psychiatric comorbidity (Klassen, Katzman, & Chokka, 2010). Nevertheless, deficits in executive functions (e.g., the capacity for formulating goals, planning, and execute plans) (Lezak, 1982) have been consistently demonstrated in adults with ADHD (Adler, 2010). Some studies have shown deficits in adults with ADHD in domains related to executive functioning, such as working memory (Torralva et al., 2010), phonologic fluency (Schecklmann et al., 2009), and inhibitory control (Rapport, Van Voorhis, Tzelepis, & Friedman, 2001; Wodushek & Neumann, 2001).

Despite the fact that deficits in social cognition are an evident clinical phenomenon in ADHD, very little research has been developed in this area (Uekermann et al., 2010). In ADHD children, a few reports have suggested various deficits in domains such as facial affect recognition (Pelc, Kornreich, Foisy, & Dan, 2006; Sinzig, Morsch, & Lehmkühl, 2008), theory of mind (ToM) (Buitelaar, van der Wees, Swaab-Barneveld, & van der Gaag, 1999; Sodian, Hulskens, & Thoermer, 2003; but for different results see Charman et al., 2001), social skills (King et al., 2009; Matthys, Cuperus, & Van, 1999), and empathy (Braaten & Rosen, 2000; Dyck, Ferguson, & Shochet, 2001). In adults with ADHD, there are even fewer studies that have reported deficits in domains related to facial emotion processing (Marsh & Williams, 2006) and prosody perception (Shapiro, Gordon, Hack, & Killackey, 1993). Facial emotion processing seems to be the social cognition process that is most affected in ADHD adults (Marsh & Williams, 2006). In general terms, these social cognition impairments are consistent with frontostriatal dysfunction in ADHD (Uekermann et al., 2010), suggesting the central nature of social dysfunction in this disorder (Hoza, Waschbusch, Pelham, Molina, & Milich, 2000; Maedgen & Carlson, 2000). Despite the classical association of frontostriatal deficits and executive functions, a link between social cognition deficits, frontostriatal network, and ADHD has been highlighted (Sonuga-Barke, 2003; Uekermann et al., 2010).

Emotional inference of facial clues is one of the most important steps in the development of complex social cognition behaviors (Grossmann, 2010). Faces are multidimensional stimuli directly related to important social incentives (Ohman & Mineka, 2001). Moreover, the central role of eyes and gaze in social cognition has been acknowledged (Itier & Batty, 2009). Facial emotional expression gives an automatic and fast shortcut to alarm signs, mentalizing, and intersubjective communication. People with low social competence are impaired in recognizing emotions from facial expressions (Edwards, Manstead, & MacDonald, 1984; Feldman, Philippot, & Custrini, 1991; Philippot & Feldman, 1990). Thus, the ability to identify emotions from faces in ADHD participants can be easily assessed by the presentation of faces.

An approach which combines measures from neuropsychological and neurophysiological markers represents a reference standard in order to understand abnormal cognitive processing in neuropsychiatry and individual differences. This study seeks to identify possible behavioral, neuropsychological, and electro-physiological markers of abnormal emotion processing for faces in adult ADHD compared with controls matched by age, gender, educational level, and handedness.

Event-related potentials (ERPs) provide excellent temporal resolution of cognitive brain processing. The N170 is a cortical marker specifically linked to facial processing, with neural generators in the fusiform gyrus and superior temporal sulcus (Deffke et al., 2007; Sadeh, Zhdanov, Podlipsky, Hendler, & Yovel, 2008). The N170 represents an early cortical
response specialized for facial processing compared with objects or words (Proverbio, Riva, Martín & Zani, 2010; Rossion, Joyce, Cottrell, & Tarr, 2003). The N170 can be modulated by emotion processing (Ibáñez et al., 2010d; Righart & de Gelder, 2008). Thus, this component represents an ideal brain marker to assess possible cortical markers of emotion processing for faces in ADHD.

To our knowledge, only a single study has previously assessed facial processing in ADHD indexed by the N170, which was done with adolescents. Williams et al. (2008) reported an abnormal emotion-related N170, suggesting that the structural facial processing stage is affected in adolescents with ADHD. However, these results must be taken with caution because participants with ADHD also had comorbid depression and anxiety. For adults with ADHD, even though evidence of deficits in the processing of emotion has been reported (Herrmann et al., 2009), no N170 valence effects elicited by facial processing have been previously assessed.

We designed an ERP study based on a dual valence task (DVT) (Ibáñez et al., 2011a; Petroni et al., in press), in which faces and words were presented to test the effects of stimulus type (ST) (faces, words, or face-word stimuli), valence (positive vs. negative), and compatibility (compatible vs. incompatible word and face valence combinations). Adult participants with ADHD and controls classified stimuli according to its emotional valence (positive or negative).

In order to identify individual signatures of cognitive functioning in participants with ADHD and controls, a comprehensive neuropsychological assessment was carried out: general neuropsychology, executive functioning, and ToM. Because one plausible hypothesis is the assumption that emotion facial processing would be related to more basic (executive functions) as well as more complex (ToM) deficits in adult ADHD, we combined, in a multivariate analysis, the ERP measures with the neuropsychological assessment.

It has previously been proposed that processing of facial emotion is intertwined with complex social skills (Grossmann, 2010; Itier & Batty, 2009) and executive functioning (Pessoa, 2009). Both executive and social cognition deficits in ADHD have been reported elsewhere (Kain & Perner, 2003; Uekermann et al., 2010). This relationship between facial processing and other social or executive functions can be explained by the existence of a relatively higher-order cognitive process, in which the structural representation of the face is associated with semantic and cognitive information (Balconi & Lucchiari, 2005; Bruce & Young, 1986). Consequently, we predict that the adult participants with ADHD will show abnormal cortical processing of facial valence indexed by the N170. Moreover, these cortical deficits will be associated with basic tasks of ToM related to emotional inference. We also address two additional hypotheses: (1) Cortical deficits of emotional processing in participants with ADHD will be independent of encoding of structural facial processing (since emotional processing impairments in ADHD seem to be not exclusive for face processing); and (2) valence deficits will be related to executive functioning in participants with ADHD.

**MATERIALS AND METHODS**

**Participants**

Ten adult participants with ADHD, one female, (\(M = 33.1\) years of age, \(SD = 3.6\)), three left-handed, completed the dual valence task. Ten healthy controls, matched for gender, age (\(M = 33.0, SD = 3.8\) years old), handedness, and years of education were recruited (see below and Table 1). Participants with ADHD and controls received a thorough neuropsychological battery that comprised measures of general neuropsychology, executive functioning, and social cognition. A questionnaire was given to healthy participants to rule out hearing, visual, psychiatric, or neurological deficits. All participants gave signed, informed consent in agreement with the Helsinki Declaration. All experimental procedures were approved by the ethics committee of the Institute of Cognitive Neurology, Buenos Aires, Argentina.

**Participant criteria and recruitment process**

All participants with ADHD fulfilled DSM-IV criteria for ADHD. Diagnosis was made by three experts (A.L., F.T., and F.M.). Participants were recruited from among the patients of the Institute of Cognitive Neurology (INECO, Buenos Aires, Argentina), from the adult ADHD clinic. From the initial set, eight patients presented with ADHD combined type (ADHD/C), and two patients presented with predominantly inattentive type (ADHD/I). All patients were taking methylphenidate medication, which was suspended on the day of ERP recordings. ADHD diagnosis based on the DSM-IV criteria was assessed with the following protocol for adults:
1. ADHD Rating Scale for Adults (Barkley & Murphy, 1998), in patient and informant versions. It identifies current and retrospective childhood symptoms corresponding to DSM-IV characterization of ADHD (see supplementary material for details).

2. Depression Inventory II (BDI-II) (Beck, Steer, Ball, & Ranieri, 1996) and the Young Mania Rating Scale (YMRS) (Young, Biggs, Ziegler, & Meyer, 1978), to assess depression and mania, respectively. Scales were administered to patients and controls.

3. Neuropsychological assessment (see next section for details).

Neuropsychological assessment

Both participants with ADHD and controls received a comprehensive neuropsychological battery that lasted approximately 120 min. It included general neuropsychology, executive functioning, and social cognition.

General neuropsychology

we used general neuropsychology to evaluate participants’ basic cognitive functioning (see supplementary material for details). Memory was evaluated by the Rey Verbal Learning Test (RVLT), which comprises verbal learning, immediate and delayed recall, and a distractor list. Attention and concentration were assessed by the Trail Making Test A (TMT-A) (Partington, 1949). Phonological and semantic fluency were assessed by the Controlled Oral Word Association Test (COWAT) (Benton et al., 1994). An arithmetic test, Wechsler Adult Intelligence Scale III (WAIS III) (Wechsler, 1997), was also included.

Executive functioning

Several tests were compiled to evaluate executive functioning. The INECO Frontal Screening (Torralva, Roca, Gleichgerrcht, López, & Manes, 2009) was used to assess frontal lobe function indexed by several subtasks: Motor Programming, Conflicting Instructions, Verbal Inhibitory Control, Abstraction, Backward Digit Span, Spatial Working Memory, and Go/No Go. Backward Digit Span and TMT-B (Partington, 1949) were used to assess attentional flexibility, attentional speed, and sequencing and planning skills. Numbers Key and Searching Symbols (Wechsler, 1997) were used to evaluate visual perception and organization, visual scanning, and the efficient production of multiple motor responses. Ordering Letters and Numbers (Letters & Numbers hereafter) was used to assess mental manipulation and working memory (Wechsler, 1997). Finally, a working memory index was derived from performance on the Digit Span, Arithmetic, and Letter–Number Sequencing subtests (Hill et al., 2010).

Social cognition

Only one social cognition test was included: the Reading Mind in the Eyes Test (RMET) (Baron-Cohen et al., 2001), which assesses individual differences in the ability to infer the affective mental states of other humans. All neuropsychological performance data are shown in Table 1.

Procedure

Dual valence task

In a two-alternative, forced-choice task, participants classified words or faces displayed on a computer screen according to their valence, into one of two categories (positive or negative), as quickly as possible. When responses were not correct, an X appeared briefly in the center of the screen. The task comprised two blocks of 320 trials.

Trial structure. A trial (Figure 1) started with a fixation cross for 1000 ms. Then a stimulus was presented for 100 ms, followed by a fixation cross until participants responded. If the response was incorrect, a red cross was presented as feedback for 100 ms, and the trial ended. Otherwise, the trial ended without feedback. After responses or feedback, an ISI of 1000 ms was added (not shown in Figure 1).

Simultaneous stimuli block. In this block, participants were exposed in each trial to a face in the center of the screen and a word 4° below, simultaneously for 100 ms. Participants had to respond as to whether the face was angry or happy, ignoring the word presented below. Congruent trials presented a face and a word of the same valence, and incongruent trials presented stimuli with opposite valences (e.g., an angry face with a pleasant word).

Single stimulus block. In a second, counterbalanced block, participants were exposed on each trial to a face or a word in the center of the screen, and responded as to whether the stimulus was angry or happy in the case of faces, or pleasant or unpleasant, in the case of
words. Single-stimulus block trials were presented one by one with strict alternation between words and faces.

Two response keys were used. Each block was separated into two subblocks of 160 trials, in which the response keys were inverted explicitly. Each subblock was preceded by written instructions with the correct correspondence between stimulus category and response key and six trials of practice. This procedure was taken from a previous two-choice task (Hurtado, Gonzalez, Haye, Manes, & Ibáñez, 2009; Ibáñez et al., 2010d, 2011a).

Stimulus construction and validation

Facial pictures were taken from a data set used in previous studies (Hurtado, Gonzalez, Haye, Manes, & Ibáñez, 2009; Ibáñez et al., 2010d, 2011a, 2011b) (see supplementary data for stimuli validation). A set of 10 happy and 10 angry pictures controlled for intensity, brightness, color, and contrast was included. Each of the 10 actors was present in one happy and one angry stimulus. Thirty-three pleasant and 32 unpleasant words controlled for arousal, content, length, and frequency were also selected from a previous study (Ibáñez, López, & Cornejo, 2006; see supplementary data for validation details).

ERP recordings

EEG signals were sampled at 500 Hz from a Biosemi 128-channel Active Two system. Data were band-pass filtered (0.1 to 100 Hz) while recording and (0.3 to 30 Hz) off-line to remove unwanted frequency components. During recording, the reference was set by default to link mastoids and re-referenced off-line to average electrodes. Two bipolar derivations monitored vertical and horizontal ocular movements (EOG). EEG data were segmented from 200 ms before to 800 ms after the stimulus onset. All segments with eye-movement contamination were removed from further analysis by an automatic (Gratton, Coles, and Donchin method for removing eye-blink artifacts) visual procedure. Artifact-free segments were averaged to obtain ERPs.

Source localization

Distributed source models (8000 dipoles) of the N170 component for each condition were estimated by the standardized, low-resolution, brain electromagnetic tomography algorithm (sLORETA) (Pascual-Marqués, 2002). These locations were derived by performing a location-wise inverse weighting of data, with a minimum norm least-squares analysis of their estimated variances, leading to a smooth solution. An average head model built from a sample of 152 MRIs provided by the International Consortium of Brain Mapping (ICBM) was used (Mazziotta et al., 2001). To make a more realistic model, we considered white matter anisotropy by using a diffusion tensor atlas of 81 healthy participants (Mori et al., 2008) coregistered with the ICBM model. The forward problem was solved by a finite element method (Zhang et al., 2004).

Possible solutions were constrained for location to the cortical surface but were not constrained for orientation (Valdez-Hernández et al., 2009). This head model is useful for source localization when individual MRI data are not available. Given that temporal
differences occur between participants and STs, the local minimum within the N170 window was considered for each of them. Average signal for the N170 representative electrodes A9, A10, A11, A12 (left) and B6, B7, B8, B9 (right) was obtained, within a 150–210-ms time window for faces and simultaneous stimuli, and within a 160–230-ms time window for word stimuli for each subject. N170 peak amplitude was found as the local minimum of this average. Potentials of all channels at local minimum were extracted for each participant and condition. Standardized current density power was obtained for each condition and subject. Finally, the average of this source images for faces, simultaneous, and words stimuli were obtained for the each group.

Data analysis

Off-line processing and analysis of EEG data were performed by Matlab software, EEGLAB toolbox, and T-BESP software (http://neuro.udp.cl/software). To analyze scalp topography of the ERP components, we used regions of interest (ROIs), as recommended for dense arrays (e.g., Aravena et al., 2010; Ibañez et al., 2010c; San Martín, Manes, Hurtado, Isla, & Ibañez, 2010), since it improves statistical power. ROIs were chosen after visual inspection of each component. Each N170 ROI (left and right) included four adjacent electrodes around T8 and T7 (Rossion & Jacques, 2008): the N170 ROIs were A9, A10, A11, and A12 for the left and B6, B7, B8, and B9 for the right hemisphere (see Figure 2, for the channel location selection). For ERP analysis, the 160–210-ms time window for N170 was visually selected for mean amplitude analysis.

Accuracy and N170 mean amplitudes were averaged for faces, words, and simultaneous stimuli separately and analyzed by repeated-measures ANOVA with ST (faces, words, simultaneous) and Valence (positive vs. negative) as within-subject factors. In the simultaneous stimuli condition, the factor Congruency was considered. Congruency had two categories: congruent (a positive face plus a positive word or a negative face plus a negative word) and incongruent (a negative face plus a positive word or a positive face plus a negative word). Finally, only for ERP data, the factor Hemisphere (left and right locations) was considered. A between-subject factor Group (ADHD vs. controls) was included.

In order to perform multivariate comparisons between ERPs and neuropsychology, we calculated global scores for ERPs as follows:

1. Stimulus discrimination (face-minus word) and stimulus interference (face minus simultaneous-stimuli) scores were calculated for N170 mean amplitude.
2. Valence discrimination scores were calculated by subtracting positive from negative stimuli, for N170 mean amplitude (for faces, words, and simultaneous stimuli).
3. Congruency. For simultaneous stimuli, the difference between congruent (e.g., positive face and positive word) and incongruent (i.e., negative face and positive word) was calculated for N170 mean amplitude.

To test whether ERP measures of ST and valence were associated with individual cognitive profiles, global scores were correlated with all neuropsychological tests (general, social, and executive neuropsychology), using Spearman’s rank correlations corrected for multiple comparisons (false discovery rate (FDR) correction, which controls the fraction of rejections that are false positives).

RESULTS

Demographic and clinical assessment

Table 1 shows the overall results from the demographic, clinical, and neuropsychological assessments.

Demographic data

No differences regarding age, $F(1, 18) = 0.001, p = .96$; gender, $\chi^2(1) = 0.000, = 1$; educational level, $F(1, 18) = 2.240, p = .15$; or handedness, $\chi^2(1) = 0.260, p = .60$, were observed between groups.

Clinical evaluation

ADHD participants showed significantly higher scores on behavioral measures of ADHD symptoms than did control subjects (Barkley ADHD Rating Scale for Adults). There was an expected significant between-group difference between the ADHD-RS-Inattention scale, $F(1, 18) = 13.598, p < .005$, and the ADHD-RS-Hyperactivity/Impulsivity subscale, $F(1, 18) = 5.66, p = .02$, indicating that ADHD participants had significantly higher scores for inattention and impulsivity than did control subjects. A difference between groups for BDI-II scores, $F(1, 18) = 6.438, p = .02$, was observed, indicating
high levels of depression in the ADHD group. No differences between groups were observed for the Young scale, $F(1, 18) = 0.545, p = .47$.

**Neuropsychological assessment**

**General neuropsychology**

No group differences regarding memory were observed for the RVLT total score, $F(1, 18) = 1.108, p = .30$, and the delayed, $F(1, 18) = 2.568, p = .13$. However, the RVLT recognition revealed a deficit in the ADHD group, $F(1, 18) = 9.184, p < .01$. No differences were observed in attention and concentration assessed with the TMT-A, $F(1, 18) = 1.049, p = .32$. No group differences were found on the arithmetic evaluation: WAIS-III, $F(1, 18) = 0.253, p = .62$. The phonological fluency task, $F(1, 18) = 7.862, p < .05$, yielded lower scores in the ADHD group.

**Executive functioning**

The global score on the IFS showed a trend toward lower performance for the ADHD group compared with controls, $F(1, 18) = 3.79, p = .07$. On closer examination, only the IFS subtasks of Abstraction Capacity, $F(1, 18) = 4.47, p = .04$, and Spatial Working Memory, $F(1, 18) = 6.37, p = .02$, yielded lower scores in the ADHD group. As regards the other measures of executive functioning, attention deficits in the ADHD group were revealed, as measured by digit repetition, $F(1, 18) = 34.184, p < .001$. No differences were observed on the Working Memory Index, $F(1, 18) = 2.66, p = .11$. In contrast, no deficits in attentional flexibility, attentional speed, or sequencing were observed in the ADHD group as measured by the TMT-B, $F(1, 18) = 0.016, p = .90$; Backward Digit Span, $F(1, 18) = 2.070, p = .17$; and Letters and Numbers task, $F(1, 18) = 0.76, p = .39$.

**Social cognition**

When we compared percentage accuracy on the RMET, a small deficit was indicated in the ADHD group, shown by a trend, $F(1, 18) = 3.48, p = .08$, suggesting that patients had a subtle deficit in the emotional inference process.

**Dual valence paradigm**

**Behavioral results**

Both groups performed the task with an accuracy of 88% or more (see Table 2 for mean fractions and SD).
TABLE 1

Results are shown as mean (SD), and statistical comparison test results are shown in the right-hand column. Statistical comparison test result p values are shown when significance was achieved. In all other cases, ns is used to indicate a “nonsignificant” difference.

<table>
<thead>
<tr>
<th>Demographics</th>
<th>ADHD (n = 10)</th>
<th>Control (n = 10)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>33.1 (3.42)</td>
<td>33.3 (3.64)</td>
<td>ns</td>
</tr>
<tr>
<td>Gender (M:F)</td>
<td>1:9</td>
<td>1:9</td>
<td>ns</td>
</tr>
<tr>
<td>Education (years)</td>
<td>15.9 (0.87)</td>
<td>17.8 (0.89)</td>
<td>ns</td>
</tr>
<tr>
<td>Handedness (L:R)</td>
<td>3:7</td>
<td>2:8</td>
<td>ns</td>
</tr>
<tr>
<td>Barkley Inattention</td>
<td>12.30 (2.60)</td>
<td>1.80 (1.14)</td>
<td>.005</td>
</tr>
<tr>
<td>Clinical profile</td>
<td></td>
<td></td>
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<tr>
<td>Hyperactivity</td>
<td>9.20 (2.21)</td>
<td>2.70 (1.60)</td>
<td>.02</td>
</tr>
<tr>
<td>BDI-II</td>
<td>17.90 (4.29)</td>
<td>5.50 (2.32)</td>
<td>.02</td>
</tr>
<tr>
<td>YMRS</td>
<td>1.10 (0.64)</td>
<td>0.50 (0.50)</td>
<td>ns</td>
</tr>
<tr>
<td>General neuropsychology</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TMT-A</td>
<td>32.20 (4.41)</td>
<td>38.60 (4.48)</td>
<td>ns</td>
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<tr>
<td>Phonological fluency</td>
<td>17.30 (1.41)</td>
<td>22.90 (1.82)</td>
<td>.05</td>
</tr>
<tr>
<td>RVLT</td>
<td>47.10 (3.80)</td>
<td>52.20 (2.33)</td>
<td>ns</td>
</tr>
<tr>
<td>DL</td>
<td>7.90 (1.07)</td>
<td>6.60 (0.60)</td>
<td>ns</td>
</tr>
<tr>
<td>Delayed</td>
<td>10.80 (0.92)</td>
<td>12.90 (0.99)</td>
<td>ns</td>
</tr>
<tr>
<td>Recognition</td>
<td>12.80 (0.54)</td>
<td>14.80 (0.46)</td>
<td>.01</td>
</tr>
<tr>
<td>Arithmetic (WAIS-III)</td>
<td>14.20 (1.26)</td>
<td>15.10 (0.96)</td>
<td>ns</td>
</tr>
<tr>
<td>WMI</td>
<td>100.60 (4.38)</td>
<td>110.02 (3.91)</td>
<td>ns</td>
</tr>
<tr>
<td>Digit repetition</td>
<td>11.80 (1.32)</td>
<td>19.50 (0.93)</td>
<td>.001</td>
</tr>
<tr>
<td>Digits backward</td>
<td>4.44 (0.34)</td>
<td>5.10 (0.34)</td>
<td>ns</td>
</tr>
<tr>
<td>Executive functions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TMT-B</td>
<td>71.00 (6.77)</td>
<td>72.20 (4.23)</td>
<td>ns</td>
</tr>
<tr>
<td>Letters and Numbers</td>
<td>11.20 (0.80)</td>
<td>12.20 (0.80)</td>
<td>ns</td>
</tr>
<tr>
<td>IFS total score</td>
<td>25.30 (0.85)</td>
<td>27.90 (0.56)</td>
<td>.045</td>
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<tr>
<td>RMET</td>
<td>71.32 (3.02)</td>
<td>79.21 (2.43)</td>
<td>.08</td>
</tr>
</tbody>
</table>

Notes: BDI-II: Beck Depression Inventory-II; YMRS: Young Mania Rating Scale; RVLT: Rey Auditory Verbal Learning Task; DL: Distractor List; TMT: Trail Making Test; IFS: INECO Frontal Screening; WMI: Working Memory Index.

TABLE 2

Performance on the DVT for patients and controls (fractions). The signs + and – depict emotional valences. The double signs in the last four columns indicate valence for faces and words, respectively.

<table>
<thead>
<tr>
<th>Face +</th>
<th>Face –</th>
<th>Word +</th>
<th>Word –</th>
<th>Sim ++</th>
<th>Sim +</th>
<th>Sim –</th>
<th>Sim +</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADHD mean</td>
<td>0.92</td>
<td>0.93</td>
<td>0.90</td>
<td>0.90</td>
<td>0.91</td>
<td>0.90</td>
<td>0.90</td>
</tr>
<tr>
<td>ADHD SD</td>
<td>0.09</td>
<td>0.05</td>
<td>0.06</td>
<td>0.10</td>
<td>0.07</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>Controls mean</td>
<td>0.92</td>
<td>0.90</td>
<td>0.91</td>
<td>0.9</td>
<td>0.92</td>
<td>0.89</td>
<td>0.88</td>
</tr>
<tr>
<td>Controls SD</td>
<td>0.07</td>
<td>0.07</td>
<td>0.06</td>
<td>0.07</td>
<td>0.05</td>
<td>0.08</td>
<td>0.07</td>
</tr>
</tbody>
</table>

The performance on the overall task was very similar for both groups: 91%, SD = 0.02, for the ADHD group and 90%, SD = 0.02, for the control group, F(1, 18) = 0.04, p = .89. For faces, word, and simultaneous stimuli, no differences were found for ST, F(1, 36) = 1.8, p = .17, or group differences, F(1, 18) = 0.08, p = .77. For faces, no main effects of valence, F(1, 18) = 0.07, p = .79, or group, F(1, 18) = 0.95, p = .34, were found. For words, no main effects for valence, F(1, 18) = 0.26, p = .61, or group, F(1, 18) = 0.003, p = .95, were found. Finally, for simultaneous stimuli, an effect of valence was observed—accuracy: positive > negative, F(2, 18) = 4.10, p = .03—but no group effect, F(2, 17) = 0.55, p = .58. No congruency effects, F(2, 18) = 0.06, p = .94, or group differences were observed, F(2, 18) = 0.55, p = .58, for simultaneous stimuli comparing the congruency of face and word valence. There were no any interactions between any of the previously mentioned factors. Table 2 shows the descriptive statistics.

Summarizing the behavioral results, accuracy was high across all conditions for both groups, and, despite the small mean differences between conditions, no significant effects were observed for ST in either
group. For valence effects, only a small difference was obtained in simultaneous stimuli for both ADHD and control groups (performance was better for positive than for negative valence). No other effects yielded significant differences.

ERPs

ST effects

In a comparison of the N170 amplitudes elicited by faces, words, and simultaneous stimuli, a main effect of ST was obtained, $F(2, 36) = 4.40, p < .01$, mainly caused by an amplitude enlargement of the N170 for faces. The ST effect was more accentuated over the right hemisphere, as evidenced by ST x Hemisphere interaction, $F(2, 36) = 8.71, p < .001$. We performed a post-hoc analysis of this interaction (Tukey HSD test, $MS = 1.44, df = 36$) and found that faces elicited enhanced N170 amplitudes compared with simultaneous stimuli ($p < .001$) and words ($p < .0005$) in the right hemisphere. Although face stimuli presented right > left amplitude differences, this effect was not significant ($p = .20$). In the left hemisphere, words showed a trend toward enhanced amplitude compared to the right hemisphere ($p = .057$). No N170 amplitude differences were observed for words compared with faces ($p = .99$) on the left side. However, significant word N170 amplitude enlargement was obtained compared with simultaneous stimuli ($p < .005$) in the left hemisphere. In summary, faces elicited an enhanced amplitude over the right hemisphere (compared with words or simultaneous stimuli), and words elicited an enhanced amplitude over the left hemisphere (compared with simultaneous stimuli). No group differences or other factor interactions were observed for ST modulation. Both groups presented N170 modulation of faces to the right and words to the left. Figure 3A shows the ERPs for ST modulation and Figure 3B the scalp topography for both groups.

Valence effects

Faces. No main effects of valence, $F(1, 18) = 1.07, p = .31$; group, $F(1, 18) = 0.05, p = .81$; or hemisphere, $F(1, 18) = 2.04, p = .17$, were observed. A significant interaction between valence x group, $F(1, 18) = 6.49, p = .02$, and a strong interaction between valence x group x hemisphere, $F(1, 18) = 18.32, p < .0005$, were found, evidencing different cortical patterns of emotional processing between groups in the right hemisphere. Post-hoc comparisons performed on this last interaction (Tukey HSD test, $MS = 59.05, df = 18.07$) showed that N170 of controls distinguished facial valence in the right hemisphere, but ADHD participants lacked N170 valence modulation. Increased N170 amplitude for positive faces compared with negative ones in the right hemisphere yielded significant effects ($p < .0005$) in control participants. In contrast to controls, no effects of valence were observed in the left ($p = .31$) or right hemispheres ($p = .87$) for ADHD patients. Moreover, when we compared the specific valences between groups in the right hemisphere, no differences were observed with negative stimuli ($p = .98$). Nevertheless, a strong effect indicated that the ADHD group had a significantly reduced amplitude for positive stimuli, compared with controls ($p < .01$). No other relevant pairwise comparisons were significant. Figure 3 and Table 3 show the N170 effects on valence for controls and patients.

Words. For word stimuli, N170 was not modulated by the valence, $F(1, 18) = 0.03, p = .84$. The interaction between word valence x hemisphere was not significant, $F(1, 18) = 0.32, p = .57$. No group effects or interactions between group and other factors were observed. Means are shown in Table 3. In brief, word valence was not discriminated by N170 in either controls or patients in either hemisphere.

Simultaneous stimuli. Similar results were observed for simultaneous stimuli as were reported for facial valence modulation. An interaction between valence x group x hemisphere, $F(1, 18) = 5.63, p < .05$, suggested that controls still presented valence effects of simultaneous stimuli in the right hemisphere. Post-hoc comparisons performed on this last interaction (Tukey HSD test, $MS = 30.15, df = 18.26$) showed that controls presented facial valence modulation in the right hemisphere ($p < .05$), but participants with ADHD lacked an N170 valence modulation in the right hemisphere ($p = .89$). No other pairwise comparisons yielded significant effects.

We performed additional analyses to investigate valence effects in relation to congruency between faces and words in the simultaneous stimuli. No effects of valence congruency were observed in the N170 window, in either hemisphere or group, nor was there any interaction. Table 3 shows the means and SDs for these conditions.

Source activity

Figure 4A shows the distributed activation evoked by the ST conditions (face, words, and simultaneous) in both, controls and ADHD participants. The
source of N170 neural activity was observed at different posterior portions of the fusiform gyrus (FG): left hemisphere for words, peak at –30, –81, and –20 for controls, and –25, 87, and –21 for patients; right hemispheres for faces, peak at 40, 67, and –12 for controls, and 25, –86, and –18 for patients; and simultaneous stimuli, peak at 26, –76, and –16 for controls and 20, –86, and –20 for patients. Table 4 shows the results of the estimation of cortical sources for N170. Standardized current density power was higher in the case of controls against patients, as consistent with greater amplitude of sources for controls. Controls presented decreasing FG activation from face to simultaneous and word stimuli. Consistent with the ERP results, the patient group presented a reduced activation of fusiform gyrus and N170 peak compared to controls. Figure 4B and 4C shows the average intensity of the source peak and the FG for N170 window in all conditions and groups.

Multivariate analysis

**ERP correlations with general neuropsychology**

*ADHD patients.* Phonological fluency was correlated with N170 stimulus discrimination (face–word; \( r = .52, p = .03 \)) and face valence (positive–negative, \( r = .64, p = .02 \)).

*Controls.* The TMT-A was positively correlated with N170 stimulus discrimination (face–word; \( r = .31, p = .04 \)).

*Executive functioning*

*ADHD patients.* Working memory (Backward Digit Span) correlated with face valence (positive–negative, \( r = .58, p < .04 \)). The N170 of valence discrimination for simultaneous stimuli (positive–negative) correlated with Digit Repetition, \( r = .71, p = 0.001 \), Letters and Numbers (WAIS-III) \( r = .68, p = .02 \), and the Working Memory Index \( r = .54, p < .02 \).

*Controls.* The TMT-B correlated positively with the N170 valence discrimination for simultaneous stimuli \( r = .589, p < .05 \) \( r = .64, p = .02 \). The N170 valence discrimination for simultaneous stimuli correlated with the Working Memory Index \( r = .68, p < .01 \).

*Theory of mind*

*ADHD patients.* RMET scores correlated significantly with N170 face valence discrimination (happy–angry; \( r = .51, p = .03 \)).

*Controls.* RMET scores correlated significantly with N170 face valence discrimination (happy–angry; \( r = .32, p = .04 \)).

**DISCUSSION**

The primary goal of this report was to investigate cortical markers of facial and semantic emotion processing in adults with ADHD and controls matched for gender,

### TABLE 3
N170 amplitude values in response to stimulus type, valence, and congruency factors

<table>
<thead>
<tr>
<th></th>
<th>ADHD mean (SD)</th>
<th>Controls mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left</td>
<td>Right</td>
</tr>
<tr>
<td></td>
<td>Left</td>
<td>Right</td>
</tr>
<tr>
<td><strong>a. Stimulus type effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Faces</td>
<td>–2.49 (1.64)</td>
<td>–2.59 (1.88)</td>
</tr>
<tr>
<td>Words</td>
<td>–1.77 (1.28)</td>
<td>–0.68 (1.15)</td>
</tr>
<tr>
<td>Simultaneous</td>
<td>–0.51 (1.58)</td>
<td>–0.88 (1.95)</td>
</tr>
<tr>
<td><strong>b. Face valence effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>–2.70 (1.63)</td>
<td>–2.81 (1.88)</td>
</tr>
<tr>
<td>Negative</td>
<td>–2.37 (1.69)</td>
<td>–2.47 (1.92)</td>
</tr>
<tr>
<td><strong>c. Word valence effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>–1.56 (1.39)</td>
<td>–0.76 (1.15)</td>
</tr>
<tr>
<td>Negative</td>
<td>–1.98 (1.18)</td>
<td>–0.60 (1.21)</td>
</tr>
<tr>
<td><strong>d. Simultaneous valence effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>–0.74 (1.57)</td>
<td>–1.14 (1.97)</td>
</tr>
<tr>
<td>Negative</td>
<td>–0.28 (1.72)</td>
<td>–0.73 (1.95)</td>
</tr>
<tr>
<td><strong>e. Congruency effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congruent</td>
<td>–0.37 (1.23)</td>
<td>–0.82 (1.90)</td>
</tr>
<tr>
<td>Incongruent</td>
<td>–0.64 (1.87)</td>
<td>–0.95 (2.02)</td>
</tr>
</tbody>
</table>

**source of N170 neural activity was observed at different posterior portions of the fusiform gyrus (FG):** left hemisphere for words, peak at –30, –81, and –20 for controls, and –25, 87, and –21 for patients; right hemispheres for faces, peak at 40, 67, and –12 for controls, and 25, –86, and –18 for patients; and simultaneous stimuli, peak at 26, –76, and –16 for controls and 20, –86, and –20 for patients. Table 4 shows the results of the estimation of cortical sources for N170. Standardized current density power was higher in the case of controls against patients, as consistent with greater amplitude of sources for controls. Controls presented decreasing FG activation from face to simultaneous and word stimuli. Consistent with the ERP results, the patient group presented a reduced activation of fusiform gyrus and N170 peak compared to controls. Figure 4B and 4C shows the average intensity of the source peak and the FG for N170 window in all conditions and groups.

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*Controls.* The TMT-A was positively correlated with N170 stimulus discrimination (face–word; \( r = .31, p = .04 \)).
handedness, educational level, and age. The secondary goal was to assess the individual variability of cognitive processing that related to the cortical markers of the DVT in both groups. Although both groups showed high accuracy on the DVT, there were important between-group differences in cortical processing. Compared with controls, the adult ADHD group showed deficits in N170 emotion modulation for facial stimuli. Those N170 impairments were observed despite there being no deficit in processing of facial structure, suggesting an ADHD-specific impairment in early facial emotion modulation. The two groups showed slightly dissimilar cognitive profiles associated with N170 processing. Notably, in ADHD participants, N170 emotion processing was associated with performance on an emotional inference ToM task, and N170 for simultaneous stimuli was associated with executive functioning, especially working memory. In summary, this is the first report to reveal an adult ADHD-specific impairment in the cortical modulation of face valence (independent of facial processing per se) and an association of cortical measures with emotional ToM and executive functioning.

**Behavioral performance (DVT)**

Accuracy was high in both groups, evidencing an adequate comprehension and execution of the task. This high accuracy means that we can be confident that our findings for the ADHD group cannot be explained as reflecting inattention or distractibility.

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**Figure 3.** Main ERP results. (A) N170 component for both hemispheres and groups. First row: stimulus type effects. Second row: Face Valence effects. (B) Topographical maps of N170 window for stimulus type effects (faces, words, and simultaneous stimuli) in both ADHD and control groups.
Moreover, the demonstration of cortical differences in the absence of behavioral divergences highlights the power of using ERPs as a tool for measuring the subclinical brain processes that are related to cognition (e.g., Gray, Ambady, Lowenthal, & Deldin, 2004; Guerra et al., 2009; Ibáñez et al., 2006; Ibáñez, San Martín, Hurtado, & López, 2008a; Ibáñez, San Martín, Hurtado, & López, 2008b; Ibáñez et al., 2010a, 2010b, 2010c; Kotchoubey et al., 2006), and pathophysiology in ADHD (e.g., Herrmann et al., 2009; López et al., 2006).

N170 results for ST, valence, and simultaneous stimuli processing

Our results replicate a previous DVT study carried out with healthy volunteers with no disorder (Ibáñez et al., 2011a; Petroni et al., in press) and other studies about the modulation of N170 amplitude via ST modulation (face > word; Rossion et al., 2003) and facial valence modulation (positive > negative: Ibáñez et al., 2011; Schacht & Sommer, 2009). It replicates the finding that there are no effects of word valence (Schacht & Sommer, 2009, but see Ibáñez et al., 2010d) and no effects of congruency between the valence of faces and words (Krombholz, Schaefer, & Boucsein, 2007). In summary, the N170 component seems to be part of early facial structural processing that is sensitive to the specific valence of faces and less responsive to other more complex processes related to compatibility or arousal.

For the ADHD group, cortical deficits in emotion modulation for faces were observed. Moreover, on closer examination, a deficit in face valence modulation followed reduced N170 amplitude for positive stimuli in the right hemisphere. This main ERP result suggests a specific impairment of right hemisphere early processing of emotional faces, triggered by positive emotions. This finding is consistent with recent results in other domains. Firstly, ADHD seems to involve a deficit in positive valence picture processing at middle latency (EPN) (Herrmann et al., 2009) and abnormal affective processing of positive stimuli (Conzelmann et al., 2009). Recently, it has been proposed that, in ADHD, a possible reduction in amygdala activity (see Plessen et al., 2006) in response to positive stimuli may lead to reduced activation of the reward system and in turn to impaired processing of positive emotional stimuli (Herrmann et al., 2009;
### TABLE 4
Estimation of N170 neural generators

<table>
<thead>
<tr>
<th>Source peak</th>
<th>MNI coordinates</th>
<th>Anatomical description: (HOCSA) Harvard-Oxford Cortical Structural Atlas</th>
<th>SCDP</th>
<th>SCDP</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Faces</strong></td>
<td></td>
<td></td>
<td>Mean</td>
<td>Mean</td>
</tr>
<tr>
<td>Control</td>
<td>X: 40, Y: −67, Z: −12</td>
<td>33% Occipital Fusiform Gyrus, 21% Lateral Occipital Cortex, inferior division, 1% Temporal Occipital Fusiform Cortex, 1% Inferior Temporal Gyrus, temporo-occipital part</td>
<td>0.253</td>
<td>0.224</td>
</tr>
<tr>
<td>ADHD</td>
<td>X: 25, Y: −86, Z: −18</td>
<td>42% Occipital Fusiform Gyrus, 8% Lateral Occipital Cortex, inferior division, 6% Occipital Pole, 1% Lingual Gyrus</td>
<td>0.123</td>
<td>0.099</td>
</tr>
</tbody>
</table>

| Simultaneous | X: 26, Y: −76, Z: −16 | 68% Occipital Fusiform Gyrus, 4% Lingual Gyrus, 1% Lateral Occipital Cortex, inferior division | 0.151 | 0.133 |
| ADHD         | X: 20, Y: −86, Z: −20 | 10% Occipital Fusiform Gyrus, 5% Lateral Occipital Cortex, inferior division, 3% Occipital Pole, 1% Lingual Gyrus | 0.090 | 0.072 |

| Words        | X: −30, Y: −81, Z: −20 | 38% Occipital Fusiform Gyrus, 11% Lateral Occipital Cortex, inferior division, 1% Lingual Gyrus | 0.094 | 0.076 |
| ADHD         | X: −25, Y: −87, Z: −21 | 27% Occipital Fusiform Gyrus, 13% Lateral Occipital Cortex, inferior division, 2% Lingual Gyrus, 1% Occipital Pole | 0.082 | 0.050 |

*Notes: TOFG: temporo-occipital fusiform gyrus; SCDP: standardized current density power.*

Herrmann, Biehl, Jacob, & Deckert, 2010). This positive bias may be a specific deficit in ADHD, opposed to other emotional impairments present in comorbid disorders (such as the negative bias reported in depression and mania; e.g., Lennox, Jacob, Calder, Lupson, & Bullmore, 2004). Secondly, ADHD appears to involve predominantly right hemispheric dysfunction (for a review, see Barr, 2001; see also Booth et al., 2005). Thirdly, impaired emotional facial processing is the most consistently reported form of social cognitive impairment in ADHD (Uekermann et al., 2010). In children and adolescents with ADHD, abnormal N170 facial processing has been reported (Williams et al., 2008) as well as abnormal activity of frontal and posterior cingulated cortex activated by emotional expressions, indexed by fMRI (Marsh & Blair, 2008).

Consistent with previous reports, the main source of the N170 was estimated as being located in the right temporo-occipital fusiform gyrus (TOFG) for faces (Rossion et al., 2002, 2003; Sadeh et al., 2008) and in the left TOFG for words (Maillard et al., 2010; Rossion et al., 2003). Theoretical models of emotion face perception (Vuilleumier & Pourtois, 2007) propose a parallel and interactive system indexing object recognition (e.g., triggered by the FG) and emotional discrimination (e.g., triggered by the amygdala). The amygdala mediates emotional processing and valence and is involved in the processing of facial affect.
The more basic and structural face integration process seems to be preserved in patients, yet more subtle processes, such as the emotional processing of the face, seem to be affected at early stages of processing (e.g., triggered by reduced connectivity between amygdala and FG). This speculative remark is consistent with recent reports of ADHD abnormal amygdala activation to emotional stimuli (e.g., Brotman et al., 2010; Herrmann et al., 2010).

Our result confirms previous reports of abnormal facial processing in ADHD (children and adolescents), and suggests that in adult ADHD the early stage of cortical face valence processing is affected. In addition, this impairment is related to emotional inference of mental states and executive functioning. Summarizing our data and related reports, we may propose that early right hemisphere dysfunction of processing positive facial expressions in adult ADHD is a neurocognitive marker of basic social cognition deficits, calling for further examination.

**ADHD cognitive profile and its association with ERP processing**

Our patients evidenced mild to moderate levels of depressive symptoms as revealed by scores on the BDI-II, as is usual in this clinical population and congruent with previous reports from our team (Torrvalva et al., 2010; Torrente et al., 2010) and other studies (e.g., LeBlanc & Morin, 2004). Notwithstanding, in the current study, no associations between depression and ERP processing were found, suggesting a relative independence of those domains. Further research is called for in this area.

Participants with ADHD presented deficits in recall performance on the RAVLT, as well as some executive impairment, which is not a new issue (e.g., Torralva et al., 2010). In addition, we found a subtle deficit in ToM indexed by the RMRT, and this task correlated with cortical deficits in face valence. At the same time, in the ADHD patients, the ERPs for simultaneous stimuli valence discrimination were associated with higher levels of executive functioning and working memory. Both executive and ToM deficits in ADHD have been reported elsewhere, and these deficits are often both associated with the disorder (Kain & Perner, 2003; Uekermann et al., 2010).

The finding of a combined executive and social impairment is consistent with current neural models of cognition (Pessoa, 2009) and particularly with dysfunction of frontostriatal structures in ADHD (for reviews, see Bush, Valera, & Seidman, 2005; Marsh & Williams, 2006; Uekermann et al., 2010). Thus, a subtle frontostriatal deficit in ADHD could be the neural signature of the combined profile of executive and social cognitive deficits that was reported in this study.

Recent reports on individual differences and N170 processing have stressed the importance of combining a multilevel analysis of cortical measures of facial processing with neuropsychological assessment (Herzmann, Kunina, Sommer, & Wilhelm, 2010; Ibáñez, Haye, González, Hurtado, & Henríquez, 2009; Marsh & Williams, 2006; Petroni et al., in press). Combining neuropsychology with brain function measures of emotion processing may lead to improved clinical assessment of emotional disturbances in ADHD (Williams, 2008). Our results highlight this by demonstrating an ADHD-specific pattern of association between executive and social deficits on ERP and neuropsychology measures; this supports a frontostriatal model of ADHD related to emotional and cognitive functioning. In addition, between-group individual differences suggested a different cognitive profile for people with ADHD, probably reflecting the use of different cognitive strategies in ADHD and controls (see Durston et al., 2003).

**Limitations and future studies**

Our results suggest an abnormal brain processing of emotional facial stimuli in adult ADHD, consistent with a broad body of research about frontostriatal dysfunction. This preliminary report should be recreated in the near future considering several possible improvements. First, a larger sample including groups of different ADHD subtypes and gender differences is required. The sample size of this study was small, since we included only ADHD patients with no comorbidity. Our results are restricted to males with ADHD. All patients were under medication, and stimulants may have permanent effects on brain function. Assessing other types of emotion (e.g., six basic emotions) and comparing those effects in drug-naive participants (in order to avoid the possible long-term effects of medication) would be additional steps. Finally, it would be relevant to compare the present result with other disorders that occur comorbidly with ADHD, such as bipolar disorder and schizophrenia (Barr, 2001; Ibáñez et al., 2011c; Lus & Mukaddes, 2009; Peralta et al., 2010).

**CONCLUSION**

In this report, we identified brain markers of impaired facial emotion modulation in participants with ADHD. Those deficits were related to subtle differences in ToM and executive functioning, supporting the
frontostriatal dysfunction hypothesis of ADHD. By a multilevel approach, we highlighted the advantage of combining neuropsychological assessment with brain measures from translational neuroscience.

Although a broad range of studies have assessed social cognitive impairments in ADHD, as well as their relation to executive function, the clinical and everyday impact of those deficits is still unclear (Marsh & Williams, 2006; Nijmeijer et al., 2008). Further research on social cognitive deficits would help us understand various problems that tend to occur in the clinical profile of children and adults with ADHD, such as fewer friends and difficulty in keeping friends (Nijmeijer et al., 2008), an increased risk of mood and anxiety disorders or antisocial personality disorder (see Nijmeijer et al., 2008), and higher rates of marriage difficulties (Biederman et al., 1993; Murphy & Barkley, 1996). Social cognitive deficits could account at least partially for troubles in school life and employment, together with basic cognitive deficits, especially in relation to discipline and acceptance of norms, both of which have been described as problematic areas of functioning for people with ADHD (Biederman et al., 1993; Murphy & Barkley, 1996). Unfortunately, current social skills training programs for children and adults with ADHD (Hesslinger et al., 2005) or bipolar disorder. American Journal of Psychiatry, 167(1), 61–69.


REFERENCES


