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ORIGINAL PAPER

Tracking Social Motivation Systems Deficits: The Affective Neuroscience View of Autism

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Abstract Abnormal functioning of primary brain systems that express and modulate basic emotional drives are increasingly considered to underlie mental disorders including autism spectrum disorders. We hypothesized that ASD are characterized by disruptions in the primary systems involved in the motivation for social bonding. Twenty adults with ASD were compared to 20 neurotypical participants on the basis of self-reports and clinical assessments, including the Social Anhedonia Scale (SAS) and the Affective Neuroscience Personality Scales (ANPS). ASD diagnosis was related to SAS, as well as to positive (PLAYFULNESS) and negative (FEAR) ANPS-traits. In the overall sample, levels of autistic traits (AQ) were related to SAS and PLAYFULNESS. We argue that PLAYFULNESS could be at the root of social bonding impairments in ASD.

Keywords Autism · Emotion · Social motivation · Social bonding · Social anhedonia · Playfulness

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Introduction

General thinking about the basis of psychiatric disorders has changed with recent advances in biological psychiatry (Northoff and Panksepp 2008; Pawelzik 2013; Walter 2013). More specifically, the role of motivational and emotional regulatory dysfunctions is now emphasized to explain the emergence and/or maintenance of various mental illnesses (Panksepp and Biven 2012). Within this framework related to affective neurosciences, Panksepp and colleagues have characterized different neuro-affective systems that constitute the neurophysiological foundation of motivations and emotions, i.e. the primary-process affective networks and associated hormones that mold the development of higher-order mental skills and frame the individual's subjective feelings, behaviors and relationships (Panksepp 2006; Davis and Panksepp 2011; Panksepp 2007; Panksepp and Panksepp 2013; Toronchuk and Ellis 2013). More precisely, six evolutionary-related brain and behavioral systems have been identified based on animal research. These systems result from gene-environment interactions and correspond to three positive and three negative emotional systems and their associate core affect programs [names in capital letters refer to the name of the systems in Panksepp's model followed by its behavioral counterpart in humans (Panksepp 1998, 2005)]: (1) SEEKING/interest (being curious, exploring, striving for solutions to problems, positively anticipating new experiences), (2) PLAYFULNESS/joy (having fun, playing games with physical contacts, humor, and laughter), (3) CARING/nurturance (being drawn to young children and pets, feeling softhearted toward animals and people in need, feeling empathy), (4) ANGER/rage (feeling hotheaded, being easily irritated and frustrated, experiencing frustration leading to anger, expressing anger verbally or physically), (5) FEAR/anxiety (feeling tense, worrying, struggling with decisions, ruminating), (6) SADNESS/panic and separation distress (feeling lonely, crying frequently, thinking about loved ones and past relationships, and feeling distress). In Panksepp's framework, the functioning of these systems is considered to promote a form of affective consciousness that is 'anoetic', i.e. an unreflective consciousness that precedes our cognitive understanding of the world (Panksepp and Biven 2012, p. 14). These systems imbue internal and external events with values (Panksepp 2001, p. 141) and this valence tagging is progressively influenced by affective-learning and regulatory mechanisms that develop with the maturation of executive functions systems. From a developmental perspective, the interplay between these positive and negative systems is thought to have a substantial influence on the emotional character of the child and the developmental trajectories of his/her academic and socio-affective skills (Cromwell and Panksepp 2011; Panksepp 2001; Toronchuk and Ellis 2013).

Davis et al. (2003) operationalized this theoretical framework with a self-report questionnaire: the Affective Neuroscience Personality Scales (ANPS). In a general way, the ANPS is a psychometric assessment of vulnerability traits based on a comprehensive taxonomy of emotional traits (Panksepp 2006). Indeed each scale of the ANPS was modeled in line with the idea that an accurate questionnaire to assess emotional personality should aim to "carve personality along the lines of emerging brain systems that help generate the relevant psychological attributes" (Cloninger 1987; Cloninger et al. 1993; Davis et al. 2003, p. 58; Ersche et al. 2010; Gray 1987; Pingault et al. 2012b). The elaboration of the ANPS was based not only on ethological considerations, but also on pharmacological and neural studies which established that these core emotional systems have distinct—though partly overlapping—subcortical networks (Panksepp 1998, 2005, 2006; Panksepp and Biven 2012). Therefore, the ANPS traits do not stem from a psychometric grouping of lexical descriptors of personality, contrasting with personality scales such as the Five Factor Model (FFM) (Abella et al. 2011; Pingault et al. 2012a). As such, using the ANPS in neurodevelopmental disorders might provide a more fine-grained evaluation of the emotional and motivational difficulties that are present in many of these conditions (see also Montag and Reuter 2014; Reuter et al. 2015 for a recent discussion on the advantage of the ANPS relative to FFM scales for the investigation of the molecular genetic bases of personality).

In Panksepp et al.'s neuro-ethological framework, the CARING/nurturance, SADNESS/panic and separation distress and PLAYFULNESS/joy systems are considered to play a key role in social bonding and, hence, in pathologies of bonding (Panksepp 1998; Siviy and Panksepp 2011; Toronchuk and Ellis 2013; Waterhouse 2012). Moreover, deficits in social interplay are considered a core symptom in many neurodevelopmental disorders (Panksepp 1981, 2007; Panksepp et al. 1984; Trezza et al. 2010), particularly in autism spectrum disorders (ASD) (Jordan 2003). Individuals with ASD are characterized by impairments in socialization, interactions and spontaneous seeking to share enjoyment or emotional reciprocity (American Psychiatric Association 2013). Impairments in developing relationships and sharing social as well as imaginative playfulness activities constitute central components in the clinical assessment of ASD (e.g. see DSM5 criterion A, APA 2014). We thus hypothesized that the CARING, SADNESS and PLAYFULNESS systems might be critically involved in the expression of these symptoms. Besides these three systems, we hypothesized that the SEEKING system—which is viewed as a core system triggering not only exploratory behaviors but also interest in pleasurable activities and in incentive reward -, may be involved in the socio-affective difficulties observed in ASD

(Damiano et al. 2012; Dawson et al. 2005; Dichter et al. 2012; Johnson et al. 2005; Kohls et al. 2011; Lai et al. 2013; Waterhouse 2012).

More precisely, failure to seek and enjoy social interactions is increasingly considered to be linked to impairments in social motivation in ASD (Chevallier et al. 2012b, 2014; Kohls et al. 2013; Levy et al. 2009; Schultz 2005; Trevarthen and Delafield-Butt 2013), and the level of pleasure and the feelings that individuals with ASD experience from interpersonal relationships (social hedonia) is currently considered to be a major emotional component of these disorders (Cinque et al. 2012; Trezza et al. 2010). For instance, individuals with ASD display reduced reward sensitivity in response to social stimuli (Dawson et al. 2005, 2002, 2012; Grelotti et al. 2002; Waterhouse et al. 1996) and, concomitantly, heightened interest in response to certain non-social stimuli (Pierce et al. 2011). Moreover, in a study that attempted to determine temperamental predictors of autistic symptomatology, Schwartz and colleagues found that parents of children with ASD described them as experiencing more negative affectivity and fear, and less pleasure (Schwartz et al. 2009). In line with this result, lack of experienced pleasure in social situations (social anhedonia) has recently been identified using self-reports in children with ASD (Chevallier et al. 2012a), as well as in adults with ASD and their relatives (Berthoz et al. 2013).

In this study, our objective was twofold: (1) to replicate recent reports of high levels of social anhedonia in ASD, beyond a potential confounding effect of depression (Berthoz et al. 2013; Chevallier et al. 2012a), (2) to investigate the links between autism and deficits in the primary emotional systems on the basis of Panksepp's affective neuroscience model and corresponding self-report (Baars and Gage 2010; Panksepp 1998, 2006; Davis and Panksepp 2011; Panksepp and Panksepp 2013; Waterhouse 2012). We expected ASD patients to display high levels of FEAR and SADNESS and low levels of SEEKING, PLAYFULNESS and CARING. Moreover, as the distribution of autistic traits and symptoms appear highly quantitative (Constantino 2011; Ruzich et al. 2015), we wanted to explore with a dimensional approach in the entire sample the associations between the level of autistic socio-affective traits and social anhedonia and the level of functioning in each primary emotional system.

Methods

Participants

All participants gave their written informed consent. The study was approved by the Ethics Committee, Paris Ile de France VI. The sample of ASD participants stem from a study comparing anorexia nervosa patients and ASD socio-affective functioning (DETENDOEMO; RGB:2007-A01068-45) using multi-level evaluations (psychometric, behavioral, neuroimaging). ASD participants were from a community active-base of patients addressed to our research group by expert clinicians (AP, FP) after having received a formal diagnosis of an ASD according to DSM-IV-TR criteria. Exclusion criteria concerned the past or present history of neurological or psychiatric disorder (except ASD for patients), including substance-related disorders, as well as non-compliance with the protocol. In addition to expert clinician judgment, current ASD presentation was assessed using the Autism Diagnostic Observational Schedule (ADOS-G) to characterize current level of functioning (Lord et al. 2000). In the present analyses, only the participants who met ADOS criteria for ASD were included. Twenty participants (14 men) with ASD (mean ADOS-G score $= 11$, SD $= 2.13$, [8–16]), and 20 healthy controls (14 men) with similar age, IQ and level of education, were recruited for this study (see Table 1). Intellectual Quotient (IQ) assessment was conducted using the short version of the Wechsler Adult Intelligence Scale (WAIS-III) form D (Grégoire and Wierzbicki 2009). Due to unanticipated circumstances, IQ data was not collected for two participants.

We focused our analyses on the self-report instruments which we considered to be the most robustly validated to assess the constructs we were interested in: the Affective Neuroscience Personality Scales (ANPS) to assess Panskepp's model dimensions, the Social Anhedonia Scale (SAS) to measure social anhedonia and the Autism and Empathy Quotients to measure autistic socio-affective traits. Moreover, given the well-known association between anhedonia and depression (Treadway and Zald 2011), participants also completed a brief self-report of current depression. These instruments are described below.

Affective Neuroscience Personality Scales

We used the French adaptation of the Affective Neuroscience Personality Scales [ANPS version 2.4 (Pahlavan et al. 2008; Pingault et al. 2012b)]. Each ANPS scale comprises 14 items. Answers are provided on a 4-point scale. ANPS scoring includes two super-factors scores (ANPS Negative: FEAR + ANGER + SADNESS; ANPS Positive: CARING + SEEKING + PLAYFULNESS), as well as separate primary emotion scores. Besides the studies on the ANPS psychometric properties (see for reviews Barrett et al. 2013; Pingault et al. 2012a, b), ANPS scores have been related to genetic (e.g. FEAR and SADNESS with the serotonin transporter polymorphism and the oxytocin receptor gene markers; ANGER with dopaminergic polymorphism) and neurobiological substrates (e.g. a negative association between ANGER or FEAR scores and

Table 1 Mean scores (and Standard Deviations) on clinical measures for the ASD and Control Groups

Variable	Patients (N = 20)	Min–Max	Controls (N = 20)	Min–Max	*t* value	*Df*	*p* value	Cohen's *d*	Effect size *r*
Age	26.3 (7.00)	19–42	22.9 (4.59)	19–37	1.82	38	0.078	0.57	0.28
IQ	103.94 (14.52)	77–135	109.50 (13.28)	80–130	−1.23	36	0.226	−0.40	−0.20
AQ-total	34.05 (6.55)	23–45	13.85 (4.85)	7–26	11.09	38	<0.001	3.51	0.87
AQ Switching	7.75 (1.89)	5–10	3.25 (1.89)	0–7	7.54	38	<0.001	2.38	0.77
AQ Social skills	7.70 (1.81)	4–10	2.30 (1.52)	0–5	10.21	38	<0.001	3.23	0.85
AQ Communication	6.75 (2.29)	3–10	1.75 (1.37)	0–4	6.75	38	<0.001	2.65	0.80
AQ Details	6.35 (2.50)	0–10	4.20 (1.85)	1–7	3.09	38	0.004	0.98	0.44
AQ Imagination	5.5 (1.79)	3–10	2.3 (1.22)	0–5	6.60	38	<.001	2.09	0.72
EQ-total	9.05 (6.21)	1–23	20.70 (5.88)	8–36	−6.09	38	<0.001	−1.93	−0.69
BDI-13	5.75 (5.56)	0–21	2.55 (2.26)	0–7	2.38	28	0.022	0.75	0.35
SAS	21.00 (8.12)	8–34	9.00 (4.58)	3–17	5.76	38	<.001	1.82	0.67
ANPS-Positive	59.50 (14.28)	28–84	79.60 (9.83)	65–102	−5.18	38	<0.001	−1.64	0.63
Seeking	23.05 (5.91)	12–32	27.25 (3.54)	21–37	−2.73	38	0.009	−0.86	−0.40
Playfulness	16.35 (7.84)	1–27	27.35 (4.64)	20–36	−5.40	38	<0.001	−1.71	−0.65
Caring	20.10 (6.77)	8–32	25.00 (4.71)	17–32	−2.66	38	0.011	−0.84	−0.39
ANPS-Negative	75.95 (18.69)	46–107	63.65 (10.48)	42–82	2.57	38	0.014	0.81	0.38
Anger	23.60 (8.52)	3–38	18.25 (5.22)	9–29	2.39	38	0.022	0.76	0.35
Fear	29.20 (7.35)	16–41	23.60 (4.45)	14–30	2.91	38	0.006	0.92	0.42
Sadness	23.15 (8.20)	7–38	21.80 (4.79)	16–31	0.64	38	0.528	0.20	0.10

According to Cohen (1988), a "small" effect-size is 0.20, a "medium" effect-size is 0.50, and a "large" effect-size is 0.80

the volume of the amygdala) (Berthoz et al. 2010; Felten et al. 2011; Montag et al. 2011; Montag et al. 2013; Montag and Reuter 2014; Reuter et al. 2009).

Social Anhedonia

The Revised Social Anhedonia Scale (SAS) (Chapman et al. 1976; Kosmadakis et al. 1995) is a 40-item (true or false) questionnaire measuring social withdrawal, lack of interest in social relationships and/or lack of pleasure derived from interpersonal relationships. It was designed to tap trait-related social anhedonia. The SAS cut-off above which a subject can be categorized as 'anhedonic' is set at ≥12 (Assouly-Besse et al. 1995). Here, 16 ASD (80 %) and 6 control (30 %) participants scored above the social anhedonia cut-off.

Autistic Socio-Affective Traits

The Autism Spectrum Quotient (AQ) (Baron-Cohen et al. 2001) is a 50-item questionnaire measuring traits associated with the autism spectrum. Participants rate their own behavior in five subscales—Social skills, Attention switching, Attention to details, Communication and Imagination—on a 4-point scale. Total scores can be classified into one of four categories: Typical (<23), Broader Autism Phenotype (23–28), Medium Autism Phenotype (29–34) or Narrow Autism Phenotype (>35) (Wheelwright et al. 2010). People with a Narrow Autism Phenotype have a large number of traits associated with the autism spectrum and most, but not all, will have a diagnosis of ASD. Individuals with Medium or Broad Autism Phenotype are unlikely to require clinical intervention, but these phenotypes are commonly observed in relatives of individuals with ASD (Berthoz et al. 2013; Wheelwright et al. 2010). Regarding the ASD group, 4 participants scored in the Broad Autism Phenotype range, 9 in the Medium range, and 7 in the Narrow range. Among the controls, 19 were typical and one scored in the Broad Autism Phenotype range.

The short form of the Empathy Quotient (EQ-short, 22 items) was used to measure empathy in vicarious situations. This questionnaire is intended to measure how participants pick up on and are affected by other people's feelings (Baron-Cohen and Wheelwright 2004; Wakabayashi et al. 2006). Participants provide their answers on a 4-point scale.

Depression

The 13-item Beck Depression Inventory (BDI-13) assesses current levels of depression and can be considered a depressive state scale (Beck et al. 1988; Collet and Cottraux 1986). Participants provide their answers on a 4-point scale.

Data Analyses

Statistical analyses were performed using R software version 2.11.1 ("Psy" and "Psych" packages). The significance level was defined at $p < 0.05$. Owing to the exploratory nature of this study, no adjustments for multiple testing were made (Bender and Lange 2001; Rothman 1990).

Descriptive statistics were first processed; the characteristics of the participants were studied according to the presence of an autistic syndrome (ASD patients versus healthy Controls) using univariate independent t tests and associated effect sizes (According to Cohen 1988, a "small" effect size is 0.20, a "medium" effect size is 0.50, and a "large" effect size is 0.80).

Then the relationships of ASD diagnosis with Social Anhedonia or Affective Neuroscience Personality scales' scores were assessed by odds ratios (OR) and 95 % confidence intervals (CI) computed via logistic models with the group (ASD vs Controls) entered as the outcome variable and the scales scores entered as the predictors. As the rule of thumb is that logistic models should be used with a minimum of five or even 10 outcome events per predictor variable (Peduzzi et al. 1996; Vittinghoff and McCulloch 2007), and given the present sample size (20 subjects per group), we did not design a single model accounting for all scores. Instead, serial logistic models with the group (ASD vs Controls) entered as the outcome variable but with different sets of predictor variables and covariates were planned:

1. Given the well-known association between anhedonia and depression (Treadway and Zald 2011), both the SAS and BDI-13 scores were entered as predictors.
2. Both ANPS Positive and Negative super-factor scores were entered as predictors to examine the relationships between each super-factor score and ASD diagnosis by controlling for the reported associations between these two ANPS scores. Where significant super-factor effects were found, we tested in the same way which underlying primary emotion scores were involved (one model with the three separate ANPS positive scales' scores: CARING, SEEKING and PLAYFULNESS; and one model with the three separate ANPS negative scales' scores: FEAR, ANGER and SADNESS).

Due to the correlation between the independent variables, potential multicollinearity was verified using the variance inflation factor (vif-index), which were all correct (the highest was 2.9).

In line with the more recent conceptualization of autistic syndromes as part of a continuum of natural social variation and the associated recommendation to examine the core social abnormality of autism as a quantitative trait (Constantino 2011; Ruzich et al. 2015), the links between the level of core autistic traits (as measured by the AQ and EQ) and that of social anhedonia (SAS) or primary emotional systems (ANPS scores) was explored in each group using Pearson product-moment correlation coefficients. Given the observed consistency in results between ASD and healthy controls participants, linear multiple regressions with the AQ score as the outcome variable were performed on the overall sample. Regarding the predictors, the same sets of variables than in the logistic models were used. Moreover, as linear regressions require a smaller number of subjects per independent variable than logistic regressions (Austin and Steyerberg 2015), we also planned to enter all the significant predictors in the same model.

Results

Descriptive and Multivariable Analyses According to an Autism Syndrome: ASD Versus Controls

Participant characteristics are shown in Table 1.

Independent-sample t tests showed that controls and ASD participants were significantly different for all questionnaire scores, except for SADNESS ($p = 0.53$). Effect sizes were large for variables that targeted autistic traits (i.e. AQ, EQ) and emotional profile (i.e., SAS, ANPS) (Cohen's $d > 0.80$, or r effect size >0.50), except for ANPS-ANGER and BDI-13, which were medium. Regarding the ANPS scales scores, as illustrated in Fig. 1, the largest between-group difference concerned the evaluation of the PLAYFULNESS system.

The findings of all rounds of multivariable logistic analyses are presented in Table 2. Regarding social anhedonia, adjusting for dysphoric affects (BDI-13)—which are considered to be a potential confounder, did not reduce the SAS estimate, and the odds ratio remained significant (raw OR 1.30, 95 % CI [1.16–1.69], adjusted OR 1.30, 95 % CI [1.12–1.66]).

Further, in the logistic regression that included the two ANPS Positive and Negative super-factor scores as independent predictors, both scores appeared significantly associated with the ASD diagnosis (see Table 2). Then, in the model accounting for the effect of each positive emotional system, where SEEKING, PLAYFULNESS and CARING scores were all entered, PLAYFULNESS remained the only significant predictor of the diagnosis (OR 0.72, 95 % CI [0.53–0.87]). In the model accounting for the effect of each negative emotional system, where ANGER, FEAR and SADNESS were all included, it appeared that FEAR was the only significant predictor (OR 1.28, 95 % CI [1.08–1.58]).

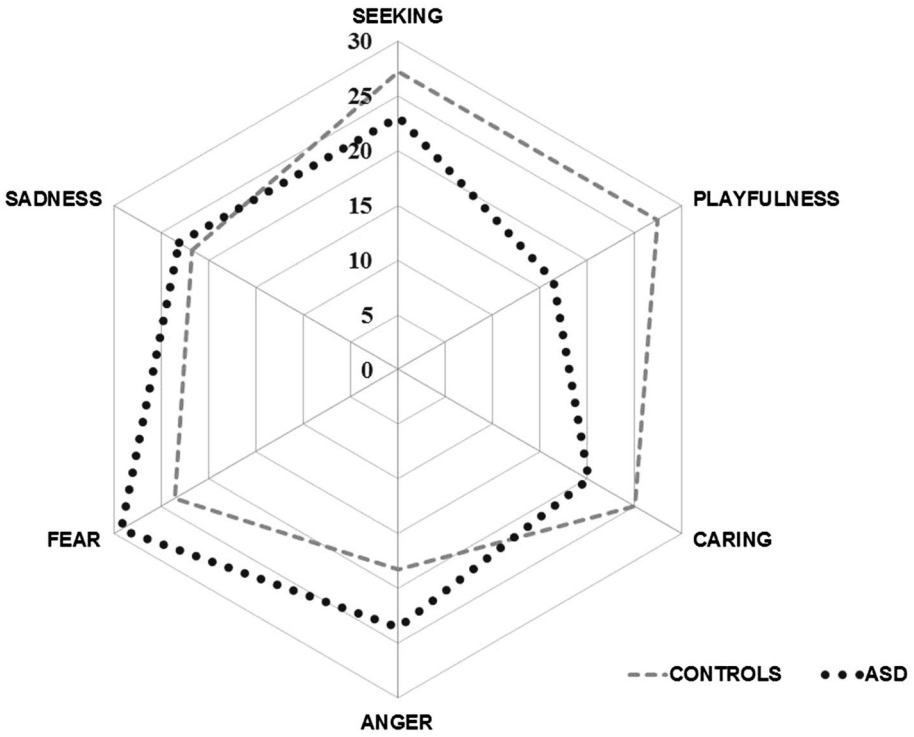

Fig. 1 Graphical representation of the mean ANPS scores for ASD (*black*) and control (*gray*) participants

Table 2 Summary of the logistic regressions predicting autism

	Odds-ratios (*OR raw*)	CI (2.5–97.5)	Estimate	SD	*z* value	*p* value
Intercept			−4.097	1.27	−3.24	0.001
Social anhedonia	1.30 (*1.30*)	1.12–1.66	0.259	0.10	2.70	0.007
Depression	1.15 (*1.27*)	0.80–1.72	0.140	0.19	0.73	0.467
Intercept			7.09	4.64	1.53	0.127
ANPS-positive	0.80 (*0.85*)	0.65–0.90	0.23	0.08	−2.84	0.005
ANPS-negative	1.14 (*1.06*)	1.04–1.31	0.13	0.057	2.30	0.021
Intercept			12.40	4.85	2.56	0.011
Seeking	0.82 (*0.82*)	0.61–1.03	−0.20	0.13	−1.56	0.120
Playfulness	0.72 (*0.72*)	0.53–0.87	−0.33	0.12	−2.66	0.008
Caring	1.01 (*0.86*)	0.82–1.25	0.01	0.10	0.07	0.948
Intercept			−5.70	2.31	−2.47	0.014
Anger	1.13 (*1.12*)	1.00–1.33	0.12	0.07	1.78	0.076
Fear	1.28 (*1.73*)	1.08–1.58	0.24	0.09	2.57	0.010
Sadness	0.87 (*1.03*)	0.72–1.00	−0.14	0.08	−1.73	0.084

Odds-ratios refer to adjusted values, and OR in brackets and italics to unadjusted odds-ratios

Dimensional Analyses

We first explored the correlations between the level of autistic traits and empathic skills (AQ, EQ) and Social Anhedonia or Affective Neuroscience Personality Scales scores, separately in ASD and Controls (see Table 3). Significant positive correlations were found between AQ total score and SAS scores in both groups (ASD: $r = 0.59$; Controls $r = 0.47$). In each group, this was the case for all the AQ subscores (available on request from the authors). Significant negative correlations were found between EQ and SAS scores in the ASD group only ($r = -0.49$).

Concerning the associations between the ANPS scores and the AQ and EQ scores, no significant relationship was found with any of the ANPS negative primary emotion subscale scores, whatever the group. With respect to the

ANPS positive scores, the SEEKING score was found significantly correlated (positively) only with the EQ scores of the Controls. Conversely, in both groups, significant negative correlations were found between the AQ and the PLAYFULNESS scores (ASD: $r = -0.76$; Controls: $r = -0.69$) and the AQ and the CARING scores (ASD: $r = -0.59$; Controls: $r = -0.45$). Moreover, in both groups, significant positive correlations were found between the EQ and the PLAYFULNESS scores (ASD: $r = 0.70$; Controls: $r = 0.53$) and the EQ and the CARING scores (ASD: $r = 0.50$; Controls: $r = 0.55$).

Table 4 shows the results of the multiple linear analyses conducted in the overall sample, with the main findings summarized as follows: (1) SAS scores remained significantly associated with AQ score (Estimate = 0.93; 95 % CI [0.66; 1.20]) after controlling for BDI-13; (2) when the PLAYFULNESS, CARING and SEEKING scores were all entered in the same model accounting for each ANPS primary positive emotion, PLAYFULNESS was the only scale that remained significantly associated with AQ scores (Estimate = −1.07; 95 % CI [−1.40; −0.75]); (3) when the FEAR, ANGER and SADNESS scores were all entered in the same model accounting for each ANPS primary negative emotion, only FEAR was found significantly associated with higher AQ scores (Estimate = 0.87; 95 % CI [0.17; 1.57]). The final model that tested whether these predictors remained significant when accounting for the effect of each separately (i.e. where SAS, PLAYFULNESS and FEAR scores were entered in the same model), PLAYFULNESS and SAS remained significant predictors of the level of AQ score in the overall sample (see Table 4).

Table 4 Linear regressions using AQ scores across all participants (overall sample)

	Estimate	*SD*	*t* value	*p* value
Intercept	7.46	2.17	3.43	0.0015
Social anhedonia	**0.93**	**0.13**	**6.96**	**<0.0001**
Depression	**0.62**	**0.26**	**2.35**	**0.0243**
Intercept	51.23	7.62	6.72	<0.0001
ANPS-positive	**−0.55**	**0.07**	**−7.79**	**<0.0001**
ANPS-negative	**0.16**	**0.07**	**2.32**	**0.0256**
Intercept	53.56	5.69	9.41	<0.0001
Seeking	−0.09	0.21	−0.43	0.668
Playfulness	**−1.07**	**0.16**	**−6.66**	**<0.0001**
Caring	−0.17	0.21	−0.82	0.416
Intercept	3.57	7.68	0.46	0.6447
Anger	0.36	0.24	1.49	0.1456
Fear	**0.87**	**0.34**	**2.52**	**0.0162**
Sadness	−0.45	0.34	−1.33	0.1927
Intercept	23.65	7.85	3.01	0.0047
Fear	0.26	0.15	1.77	0.0845
Playfulness	**−0.67**	**0.17**	**−3.99**	**0.0003**
Social Anhedonia	**0.54**	**0.15**	**3.60**	**0.0010**

Results in bold are significant predictors ($p < .05$)

Discussion

The goal of the current study was to achieve a better characterization of social motivation deficits and affective dysregulation in ASD. To do so, we used subjective measures inspired by research in both social motivation theories of ASD and affective neuroscience (Chevallier et al. 2012b; Panksepp 2006). We first confirmed that participants with ASD are socially anhedonic (Berthoz et al. 2013; Chevallier et al. 2012a), and that this held true for most of our ASD sample (80 %). This result in a French speaking sample of adults with ASD is similar to that obtained by Berthoz and colleagues (Berthoz et al. 2013) who found 74 % of social anhedonics in their sample of ASD adult participants.

Second, our study also demonstrated that ASD was not only related to high levels of negative affectivity (i.e.

Table 3 Correlation matrices for the ASD and control groups

	AQ Scores				EQ Scores			
	Patients		Controls		Patients		Controls	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*
Social anhedonia	**0.59**	**0.0064**	**0.47**	**0.0346**	**−0.49**	**0.0296**	−0.41	0.0718
Seeking	0.07	0.7541	0.12	0.6262	0.29	0.2208	**0.46**	**0.0390**
Playfulness	**−0.76**	**0.0001**	**−0.69**	**0.0007**	**0.70**	**0.0006**	**0.53**	**0.0173**
Caring	**−0.59**	**0.0065**	**−0.45**	**0.0470**	**0.50**	**0.0249**	**0.55**	**0.0121**
Anger	−0.04	0.8696	0.05	0.8421	0.15	0.5161	−0.13	0.5763
Fear	0.04	0.8663	0.09	0.7037	−0.10	0.6596	0.42	0.0628
Sadness	0.14	0.5531	−0.04	0.8590	0.13	0.5821	0.30	0.2003

Results in bold are significant correlations ($p < .05$)

ANPS Negative super-order factor), but also to low levels of positive affectivity (i.e. ANPS Positive super-order factor). More specifically, we found that the PLAYFULNESS and FEAR systems were both related to ASD (negatively for the former and positively for the latter). The effect size was particularly large for PLAYFULNESS, where each increase of one point in the total score corresponded to a 0.72 time decrease in the likelihood of having an ASD diagnosis. The effect size was medium for FEAR, and an increase of one point in the FEAR score corresponded to a 1.28 time increase in the likelihood of having an ASD diagnosis. These results are in line with Schwartz et al.'s (2009) study where parents described their children with ASD as having a temperament comprising more negative affectivity and fear, and less pleasure.

More broadly, our results fit well with a growing number of studies on negative affectivity in ASD (Hallett et al. 2013) frequently highlighting comorbid anxiety and depression disorders (Hofvander et al. 2009; Lugnegård et al. 2011; Sterling et al. 2008; Stewart et al. 2006). Depression and anxiety are even considered as secondary disorders in ASD (Schultz 2005), and the impact of psychopharmacological interventions targeting anxiety and mood disorders in individuals with ASD is now being developed (Boyd et al. 2011). Here, the effect observed for the FEAR system could correspond to tension, worry or rumination. Interestingly, manifestations of fear are extremely marked in patients with ASD, especially in response to changes in the environment and to social approaches, and these manifestations of fear might in turn lead to increased need for sameness (i.e. compulsive adherence to routine, and stereotyped, repetitive behaviors) (Gotham et al. 2013; Prior and Macmillan 1973), which is one of the core symptoms of autism, as described by Kanner (1943).

Unexpectedly, however, whereas we replicated previous well-known results that ASD subjects report low empathy scores (using the EQ-short; large effect size), we found no major difference between the groups for the CARING system. Considering its suggested role in social bonding (Panksepp and Panksepp 2013; Baron-Cohen and Wheelwright 2004; Panksepp 2011), this result may seem counterintuitive but our interpretation is that the ANPS CARING scale actually encompasses quite a wide range of "caring situations" which may not all be conceptually related: caring for the ill ("Caring for a sick person would be a burden for me"), maternal care or child-centered concerns ("I like taking care of children"), but also caring and empathy for pets ("I love being around baby animals"). Unexpectedly too, SEEKING did not significantly predict diagnosis, despite a medium effect size difference between the ASD and the control group (Baron-Cohen et al. 2009; Ben-Sasson et al. 2009; Grove et al. 2013; Waterhouse et al. 1996). Here again, beyond a potential lack of statistical power due to the sample size, our interpretation is that the SEEKING scale encompasses a wide range of situations that may be conceptually distinct: exploratory behaviors ("I really enjoy looking forward to new experiences"), seeking behaviors ("I enjoy anticipating and working toward a goal almost as much as achieving it"), as well as curiosity situations ("Almost any little problem or puzzle stimulates my interest"). These issues could be clarified in further studies, notably by using a shortened version of the ANPS, which would only comprise items that are maximally relevant for the assessment of human motivation and behavior (e.g., CARING: "I often feel a strong need to take care of others"; SEEKING: "My curiosity often drives me to do things") (Barrett et al. 2013; Pingault et al. 2012a).

Finally, we found group differences in self-reported depression (as assessed by the BDI-13) but not in SADNESS scores. This discrepancy could be explained by the fact that the BDI-13 specifically taps current dysphoric affects and relates to distress on an intra-personal level. However, the SADNESS dimension assesses inter- rather than intra-personal distress by focusing on feelings of loneliness and personal-distress associated to the loss of relationships (e.g.: "I tend to think about losing loved ones often"). These inter-personal situations may not be particularly distressing for individuals with ASD who do not appear to place as much emphasis on the concern for others (Chevallier et al. 2012c; Grove et al. 2013; Kohls et al. 2012). Interestingly, ASD participants AQ scores were positively associated with BDI-13 scores ($r = 0.59$, $p < 0.006$) but not with SADNESS scores (see Table 3), which is consistent with the suggestion that dysphoric affects emerge secondarily to ASD (Schultz 2005).

Our results, which suggest critical implications of the PLAYFULNESS and FEAR systems, relative to the CARING and SADNESS systems, also speak to a recent debate regarding a proposed revision of Panksepp et al.'s model (Toronchuk and Ellis 2013). In this updated model, two meta-systems are distinguished in the primary neuroaffective emotional and motivational systems, based on individual and social needs. Within social needs, three systems or 'life forces' are further defined (Lewis et al. 2010; Toronchuk and Ellis 2013): 'reproduction', 'group cohesion, bonding and development', and 'group function, regulating conflict' (Toronchuk and Ellis 2013, p. 4). In this framework, CARING, SADNESS and PLAYFULNESS are considered to be crucial systems for group cohesion and social bonding. Here, as outlined above, SADNESS was not related to ASD diagnosis or to the level of autistic traits as measured by the Autism Spectrum Quotient. Moreover, in the overall sample, despite the negative correlations between CARING and Autism Spectrum Quotient scores, only PLAYFULNESS and

social anhedonia (SAS scores) emerged as significant predictors of the level of autistic traits. These two scores were also predictive of the participant's diagnostic category, which is consistent with the idea that social interplay and its hedonic consequences contribute to the identification and diagnosis of ASD (Aitken 2008; Baron-Cohen et al. 1992; Jordan 2003).

At this point of the discussion, however, it is important to note a number of limitations of the present research. First, we did not have detailed information about comorbidities. Further studies including clinical tools such as the MINI (Lecrubier et al. 1997) are therefore needed. Second, our results should also be confirmed among a larger sample in order to limiting the risk of Type I errors as well as allowing the computation of more complex logistic models of analysis, and so that various subtypes of ASD can be studied independently. Third, the present study was deeply rooted in the field of affective neurosciences but we only resorted on self-report data. It would thus be interesting to combine questionnaire data with neurophysiological data (neurochemical, anatomical structure, and neural networks) in future work. In particular, atypicalities in the PLAYFULNESS system in ASD might be linked to opioid and oxytocin impairments (Dölen et al. 2013; Panksepp 1993, 2007; Panksepp et al. 1984; Sahley and Panksepp 1987). Finally, further studies including healthy and ASD-related familial cohorts and conducting cross-syndrome comparisons will also be required to ascertain that primary traits such PLAYFULNESS can be construed as endophenotypes [i.e. "measurable components unseen by the unaided eye along the pathway between disease and distal genotype" (Gottesman and Gould 2003, p. 636)].

Despite these limitations, however, we believe that our results point to a particularly important role of the PLAYFULNESS system in the psychopathology of ASD. Our results thus echoe Panksepp's original insight: "Perhaps humans with the most severe deficiencies in play circuitry will be diagnosed as schizophrenic, perhaps autistic" (Panksepp et al. 1984, p. 23). From a developmental point of view, the PLAYFULNESS system works together with other systems and allows for the development of "imaginative play" skills and symbolic representations (Ellis and Toronchuk 2005; Panksepp 1998; Toronchuk and Ellis 2013). As such, it is construed as an essential building block for the development of social cognition (i.e. cognitive learning, language acquisition, Theory of Mind, empathy and understanding of humor) (Panksepp 2006; Toronchuk and Ellis 2013; Waterhouse 2012). Indeed, social play and, more generally, the tendency to seek and enjoy social interactions, might relate to deficits in the PLAYFULNESS system, possibly upstream from impaired ToM. Recent research indeed indicates that deficits in social processing are better explained by differences in spontaneous attention and exploration of social cues rather than by impairments in social cognition (Chevallier 2012; Jemel et al. 2006).

More broadly, the PLAYFULNESS system has been described as underlying creativity and "rough-and-tumble" play, particularly in children (Davis and Panksepp 2011). This physical component of the PLAYFULNESS dimension ("I like all kinds of games including those with physical contact") might be especially involved in the "embodiment" impairments that have recently been reported in ASD (Cheslack-Postava and Jordan-Young 2012; Eigsti 2013).

From a clinical stance, diagnostic tools currently focus more on the lack of joint attention than on the lack of playfulness and shared enjoyment. The dimension of shared enjoyment is indeed present in the diagnostic criteria for ASD in the DSM IV or 5 (i.e. "reduced sharing of interests, emotions, or affect; failure to initiate or respond to social interactions") and ICD10 but it is not part of the main diagnostic instruments (e.g. ADI-R, ADOS or CARS). Interestingly, social play is one of the dimensions assessed by the Preaut scale, which also includes items looking at the presence of early active behaviors that encourage being gazed at or kissed by the mother or sharing joy with her. Interestingly, a recent study has shown that infants diagnosed with a West syndrome were 38 times more likely to receive an ASD diagnosis later in life if they had scored high on the Preaut scale at 9 months (Ouss et al. 2013).

From a therapeutic perspective, the involvement of the PLAYFULNESS system in ASD suggests that a better understanding of the neurobiological basis of social play behaviors might contribute to the development of novel treatments for psychiatric conditions in which social play and appetency for social bonding are altered (e.g. autism, early-onset schizophrenia or ADHD). More specifically, socio-emotional development is related to brain maturation (Dawson 2008; Dawson et al. 2012; Johnson et al. 2005) and socio-emotional development therefore has the power to impact neural development (Brent et al. 2013; Sallet et al. 2011). Acting on the development of the PLAYFULNESS system early on might thus have a positive impact on the development of the social brain.

In addition, relying on the PLAYFULNESS system for socio-affective skills enhancement may provide an effective therapeutic lever for certain early intervention programs (Panksepp 2007), such as applied behavior analysis (ABA), where the participation of the child in cognitive tasks is rewarded, or Floortime or Denver approaches, where shared enjoyment through play activities is a main objective of the treatment (Pajareya and Nopmaneejumruslers 2011; Warren et al. 2011). Therefore, it would be particularly interesting to assess whether early markers of the PLAYFULNESS system are correlated with both the

socio-affective and cognitive development trajectory of the child, and with the efficacy of the treatment.

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Conflict of interest The authors declare that they have no conflict of interest.

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