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Emotion attribution in intermittent explosive disorder

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ABSTRACT

Background: Accurate recognition of the emotions of others is an important part of healthy neurological development and promotes positive psychosocial adaptation. Differences in emotional recognition may be associated with the presence of emotional biases and can alter one's perception, thus influencing their overall social cognition abilities. The present study aims to extend our collective understanding of emotion attribution abnormalities in individuals with Intermittent Explosive Disorder (IED).
Methods: Two-hundred and forty-two adults participated, separated into groups of those diagnosed with IED according to DSM 5 criteria, Psychiatric Controls (PC), and Healthy Controls (HC). Participants completed a modified version of the Emotional Attribution Task wherein they attributed an emotion to the main character of a short vignette.
Results: Participants with IED correctly identified anger stories and misattributed anger to non-anger stories significantly more often than PC and HC participants. They were also significantly less likely than HC participants to correctly identify "sad stories."
Limitations: We utilized self-report assessments in a community-recruited sample. Replication in a clinical is suggested.
Conclusions: Findings from this study support the validity of IED as a diagnostic entity and provide important information about how individuals with psychiatric disorders perceive and experience emotional cues.

1. Introduction

Accurate recognition of the emotions of others is an important part of healthy neurological development and promotes positive psychosocial adaptation [1–7]. Correctly identifying others' non-verbal cues (e.g., facial expressions) facilitates successful social interactions and provides information about others' emotional experience [8]. This understanding can help lay the foundation for intimate and meaningful relationships. However, individual and group differences exist in the ability to accurately recognize and identify others' emotions. For example, evidence suggests that adults with Attention-Deficit Hyperactivity Disorder (ADHD) demonstrate an impaired ability to identify emotions expressed via facial, vocal, or facial-vocal combination stimuli when compared to healthy adults [9,10]. Some differences in emotional recognition are associated with the presence of emotional biases, which alter one's perception and influence their overall social cognition abilities.

Emotional bias is the asymmetric approach with which humans respond to emotionally charged stimuli [11]. This process may take the form of responding with increased intensity to negative stimuli (referred to as negativity bias) or demonstrating an enhanced reaction to positive stimuli, known as positivity offset [11]. These biases are evident in everyday human behaviors; such as seeking out food an individual finds appealing while encouraging them to avoid large spiders or other sources of possible danger.

Emotional biases differ as a function of trait characteristics and are also malleable. For example, one study of healthy individuals showed that heightened levels of empathy correlated with an elevated level of response bias for sad and fearful stimuli using a facial expression recognition paradigm [12]. Moreover, short-term administration of citalopram and reboxetine to healthy individuals lowered participants' recognition of fearful and angry facial expressions [13]. Studies with healthy older adults have also shown positive emotional memory [14] and vividness for positive imagery scenarios [15] can be significantly increased through an encoding task and positive imagery training, respectively. In sum, the dynamic nature of healthy emotion recognition indicates that biological, personological, and environmental factors contribute to how one labels another person's emotional experiences, known more specifically as emotion attribution.

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Many studies have examined abnormalities in emotion attribution in individuals with various psychiatric disorders, including borderline personality disorder [16], anorexia nervosa [17], depressive disorders [18], obsessive-compulsive disorder [19], body dysmorphic disorder [20], anxiety disorders [21], conversion disorder [22], and post-traumatic stress disorder [23]. In each of these studies, study participants characterized by a psychiatric disorder differed from healthy controls with respect to their interpretation of other's expressed emotions. This small but growing body of knowledge can be an important tool that helps us to better understand the social and functional impairments that individuals with various psychiatric disorders manifest.

The present study aims to extend our collective understanding of emotion attribution abnormalities to individuals with Intermittent Explosive Disorder [IED; 24,25]. IED is the sole psychiatric disorder for which impulsive aggression is the central feature, and it is characterized by recurrent, problematic, impulsive aggressive behaviors that lead to a variety of complications in social, occupational, financial, and legal spheres of life [24]. In the US, at least 4.0% of adults, and 8.9% of adolescents, meet DSM-5 criteria for IED [26]. In addition, another group of about equal size, demonstrate recurrent impulsive aggressive behavior but, otherwise, fail to fully meet the DSM-5 criteria for IED. Accordingly, IED and recurrent impulsive aggressive behavior is quite common among the US population. Of note, the diagnostic criteria for IED requires that aggressive behavior outbursts be driven by impulsivity or anger, rather than by financial gain or another secondary incentive (e.g., power) [24].

Individuals diagnosed with IED demonstrate significantly decreased psychosocial functioning when compared to healthy and psychiatric control groups [27], as clearly evidenced by allied criminological works [28,29]. For instance, results from a study of federal correctional clients indicated that the presence of an IED diagnosis was significantly related to accruing chronic assault-related arrests and several other crimes, including murder, attempted murder, and aggravated assault [28]. A similar study among juvenile offenders revealed that those in the IED group displayed significantly more aggression and were significantly more likely to have committed a violent crime than both the non-IED psychopathology and healthy control groups [29]. This altered psychosocial functioning exhibited by those with IED may stem from a deficit in identifying their own emotions [30,31]. However, there is also biological evidence that suggests these difficulties extend and present as differential responses when perceiving others' emotions [32]. For example, fMRI neuroimaging evidence suggests that amygdala responses to anger stimuli are greater in individuals diagnosed with IED than in healthy controls [33,34].

The present study extends our understanding of the social information processes in IED by examining how IED individuals evaluate others' expected emotional experiences in social situations compared to individuals (a) with no psychiatric diagnosis, or (b) a psychiatric diagnosis other than IED. It was hypothesized that individuals with IED would be specifically attuned to social situations that typically elicit anger and would be more likely to attribute anger as the correct response in other social situations that typically do not elicit anger. This information has potential implications for understanding how individuals with IED navigate their social worlds, including how they may misinterpret social cues that could lead to aggressive acts. The results could also provide information to guide the development of psychosocial interventions for this disorder.

2. Methods

2.1. Study participants

Participants included 242 physically healthy adults recruited as part of a larger study developed to investigate correlates of impulsive aggression and other personality-linked human behaviors. All participants provided written informed consent and were systematically assessed regarding aggression and other behaviors. Study recruitment was conducted via public service announcements, newspapers, and other media, with advertisements calling for individuals who: a) reported psychosocial difficulty associated with anger or, b) had minimal evidence of psychopathology. The study protocol received approval from the Institutional Review Board at the University of Chicago.

2.2. Diagnostic assessment

DSM-5 criteria were used to determine psychiatric diagnoses [24] based on information gathered from: (a) the Structured Clinical Interview for DSM Diagnoses [SCID-1; 35] for syndromal (formerly Axis I) disorders and the Structured Interview for the Diagnosis of Personality Disorder [SIDP; 36] for personality (formerly Axis II) disorders; (b) a clinical interview with a research psychiatrist; and, (c) a review of all other available clinical data. Research diagnostic interviews were administered by a master's or doctorate-level degree holder in Clinical Psychology. All diagnostic raters underwent a rigorous training program including lectures on DSM diagnoses and rating systems, videos of expert raters conducting SCID/SIDP interviews, and practice interviews and ratings until raters' performance was deemed reliable with the trainer. These procedures resulted in good to excellent interrater reliabilities (mean kappa of 0.84 ± 0.05; range: 0.79–0.93) spanning anxiety, mood, substance use, impulse control, and personality disorders. Final diagnoses were assigned by a team best-estimate consensus protocol involving research psychiatrists and clinical psychologists [37]. Although information used in assigning syndromal diagnoses was obtained using the SCID-I, clinical data were available to update assignments from DSM-IV diagnoses to DSM-5 diagnoses. Diagnoses for personality disorders based on the SIDP remain consistent between the DSM-IV and the DSM-5. Participants were excluded from the study based on having a current substance use disorder or a life history of bipolar disorder, schizophrenia (or other psychotic disorder), or an intellectual disability, because, by definition, IED participants cannot possess such comorbidities.

After diagnostic assignment, 63 participants demonstrated no evidence of a psychiatric diagnosis (Healthy Controls: HC); 41 participants fulfilled criteria for a lifetime diagnosis of a syndromal psychiatric disorder or personality disorder other than IED (Psychiatric Controls: PC), and 138 participants met criteria for a current DSM-5 diagnosis of intermittent explosive disorder. Of the 179 PC and IED participants, most ($n = 138$, 77%) participants endorsed having a prior formal psychiatric evaluation or treatment (65%) or having a behavioral disturbance during which they did not seek mental health services but others thought they should have (12%).

2.3. Assessment of aggression, anger, and impulsivity

Aggression was measured using the Aggression subscale of the Life History of Aggression assessment [LHA; 38] and the Aggression (Physical and Verbal) subscale from the Buss-Perry Aggression questionnaire [BPA; 39]. The LHA evaluates history of observable aggressive behaviors and BPA evaluates aggressive tendency as a personality trait. Anger was measured with the Anger subscale from the BPA and the Trait Anger score from the State-Trait Anger and Expression of Anger Inventory [STAXI-2; 40]. Impulsivity was determined using the Life History of Impulsive Behavior [LHIB; 41] and Barratt Impulsivity Scale [BIS-11; 42]. The LHIB evaluates the number of times a person has demonstrated impulsive behavior while the BIS-11 evaluates the individual's tendency to act impulsively as a personality trait. All measures have good to excellent psychometric properties.

2.4. Assessment of emotional attribution

A modified version of the Emotional Attribution Task (EAT) developed by Heims et al. [43] was used to assess emotion attributions in

social situations. This task presents the study participant with short vignettes describing emotional scenarios and requires them to provide an emotional label for how the central character might feel in that situation. The sentences were constructed to evoke ascriptions of happiness, sadness, fear, anger, disgust, or embarrassment. The adaptation contained fifty-four emotion stories with a priori emotional labels for "happy" (e.g., "Harry just found out he got a pay raise"), "sad" (e.g., "Margaret has just found out that her mother has died"), "fear" (e.g., Jake hears the sound warning of an imminent bomb attack"), "angry" (e.g., "Simon finds a man trying to steal his wallet from his bag"), disgust" (e.g., Rebecca is taking out the Sunday lunch out of the fridge when she sees that it is crawling with maggots"), and "embarrassed" (e.g., Ed is in the café when he slips on some grease and falls straight over; everyone in the café stares at him"). Ten emotion stories each represented "happy," "sad," "anger," and "embarrassment," with nine for "fear," and five for "disgust"; scores for the latter two sets of emotion stories were adjusted to place each set of scores on the same scale. A pilot study of twenty-five HC study participants (each of whom is included in the dataset described below) confirmed that each set of emotional stories elicited the targeted emotional label and did so compared to each other emotional label at $p < .0001$ (Fig. 1).

2.5. Other assessments

The Hollingshead approach was used to assess Socioeconomic status [SES; 44], and the Global Assessment of Function scale (GAF) was used during the diagnostic assessment to determine highest level of psychosocial functioning from the previous year [45].

2.6. Statistical analyses

Between-group data were analyzed and compared using both parametric and non-parametric methods. Diagnostic (IED and PC) group comparisons were analyzed using Chi-Square (χ^2) tests to examine differences in sex, ethnicity, and psychiatric diagnosis prevalence, with post-hoc single df χ^2 as needed. Multivariate analyses of variance and covariance (MANOVA/MANCOVA; i.e., accounting for age, sex, ethnicity, and socioeconomic score) assessed other demographic, psychometric, and task variables. A two-tailed alpha (α) value of 0.05 was used to denote statistical significance for all analyses, unless otherwise noted.

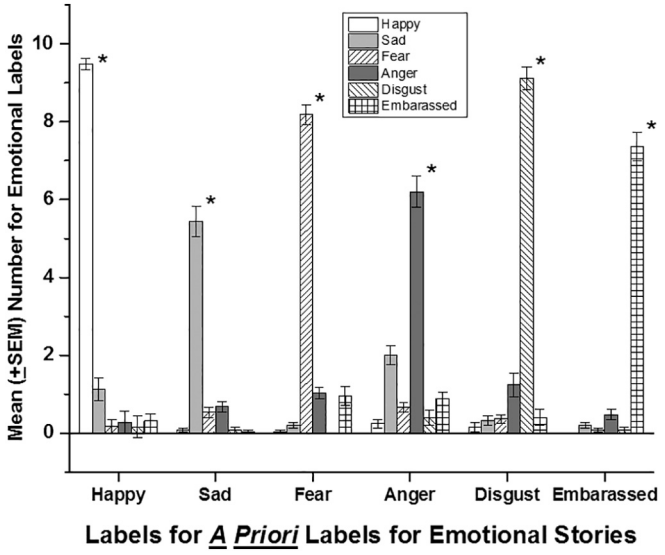

Fig. 1. Marginal means (± SEM) for MANCOVA for correct a priori emotion labels compared to other emotion labels in the pilot group of Healthy Controls. The single asterisk (*) indicates p value < .001.

3. Results

3.1. Sample characteristics

Participant groups differed modestly, but significantly, on age and distribution of sex variables, but did not differ in socioeconomic score or in distribution of ethnicity (Table 1). Age was lowest for HC compared to both PC and IED groups. The proportion of men was highest in the HC group, and lowest among IED participants. Therefore, subsequent analyses accounted for these demographic characteristics. IED participants also demonstrated lower GAF scores compared to PC participants, with HC study participants demonstrating the highest GAF scores of the three groups. Measures of aggression (LHA/BPA), anger (BPA/STAXI-2), and impulsivity (LHIB/BIS-11) produced the reverse pattern, with IED study participants exhibiting the highest scores on average on these measures compared to individuals in the HC and PC groups. IED and PC diagnostic groups also significantly differed in the presence of personality disorders and lifetime depressive disorders. None of the other comorbidity rates between IED and PC diagnostic groups differed significantly. See Table 2 for the prevalence of syndromal and personality disorder diagnoses within this sample.

3.2. Correct (a priori) emotional attribution among study participants

A MANCOVA was conducted using the number of correct emotion attributions (maximum score of 10) for all types emotions as the dependent variables in the model. Group served as the independent variable, with age, sex, ethnicity, and SES score as covariates. This analysis determined that IED study participants were more accurate than HC or PC study participants regarding anger stories and less accurate than HC in response to sad stories (see Fig. 2). Effect sizes (d) for PC and IED contrasts for "Happy", "Sad", and "Anger" were 0.38, 0.23, and 0.43, respectively. Because the presence of comorbid depressive disorder or personality disorder (Table 2) was greater in those with IED than in those without IED, we performed a subsequent analysis to determine if this effect was due to these comorbidities. MANCOVA using lifetime depressive disorder (Wilks $\lambda = 0.98$, $F[6229] = 0.91$, $p = .491$) and any personality disorder (Wilks $\lambda = 0.97$, $F[6229] = 1.01$, $p = .417$) as group variables found no effect for either on correct attribution variables.

3.3. Misattributed emotional labeling among study participants

A MANCOVA conducted with number of emotion labels endorsed other than the a priori label for all emotions as the dependent variable, group as the independent variable, and age, sex, ethnicity, and SES score as covariates, demonstrated that IED study participants were more likely to attribute anger to non-anger stories (Fig. 3; d = 0.34 for the PC vs. IED contrast). Similar MANCOVA substituting lifetime depressive disorder (Wilks $\lambda = 0.99$, $F[6229] = 0.21$, $p = .973$) and any personality disorder (Wilks $\lambda = 0.96$, $F[6229] = 1.67$, $p = .128$) as group variables found no effect for either on non a priori emotional label endorsements.

3.4. Correlates of correct anger endorsement with measures of aggression, anger, and impulsivity

Partial correlations (age, sex, ethnicity, SES) of correct anger endorsement with measures of aggression, anger, and impulsivity revealed moderate sized correlations with LHA Aggression ($r = 0.33$, $p < .001$), BPA Aggression ($r = 0.23$, $p < .001$), STAXI-2 Anger ($r = 0.33$, $p < .001$), and LHIB Impulsivity ($r = 0.32$, $p < .001$). The correlation with BIS-11 scores was lower ($r = 0.17$, $p < .011$) but not statistically significant following corrections for the number of comparisons. An analysis regressing correct anger endorsement on LHA, BPA, STAXI-2, LHIB, and BIS-11 scores revealed that only LHA Aggression scores

Table 1
Study participant demographic, functional, and psychometric characteristics.

	HC ($N = 63$)	PC ($N = 41$)	IED ($N = 138$)	P^*	Group Differences
Demographic variables					
Age	32.2 ± 10.0	35.0 ± 10.9	37.6 ± 10.1	<0.001	HC < PC = IED[a]
Sex (% Male)	60.3%	53.7%	42.0%	=0.017	HC > PC > IED[b]
Race (% White)	68.3%	70.7%	58.0%	=0.494	HC = PC = IED[b]
SES Score	42.2 ± 11.2	39.4 ± 13.4	39.4 ± 12.7	=0.303	HC = PC = IED[a]
Psychosocial functioning					
GAF Score	83.6 ± 4.6	63.9 ± 12.2	57.5 ± 9.7	<0.001	HC > PC > IED[a]
Psychometric variables					
Aggression: LHA	5.4 ± 3.3	9.1 ± 5.0	18.0 ± 4.4	<0.001	IED > PC > HC[a]
Aggression: BPA	30.1 ± 10.8	33.4 ± 10.7	42.5 ± 12.4	<0.001	IED > PC = HC[a]
Anger: BPA	13.5 ± 5.9	16.0 ± 7.1	23.0 ± 7.4	<0.001	IED > PC = HC[a]
Anger: STAXI-2	12.8 ± 2.6	16.8 ± 6.1	25.9 ± 7.2	<0.001	IED > PC > HC[a]
Psychometric variables					
Impulsivity: LHIB	24.5 ± 17.9	38.6 ± 20.6	54.8 ± 17.6	<0.001	IED > PC > HC[a]
Impulsivity: BIS-11	56.1 ± 8.2	60.9 ± 11.1	69.2 ± 11.1	<0.001	IED > PC > HC[a]

[a] by ANOVA, b by Chi-Square.

Table 2
Syndromal and personality disorder prevalence among study participants.

	PC ($N = 41$)	IED ($N = 138$)	P
Current syndromal disorders:			
Any Depressive Disorder	5 (12.2%)	31 (22.5%)	=0.186
Any Anxiety Disorder	4 (9.8%)	33 (23.9%)	=0.051
Stress and Trauma Disorders	3 (7.3%)	21 (15.2%)	=0.296
Obsessive-Compulsive Disorders	1 (2.4%)	5 (3.6%)	=0.999
Eating Disorders	2 (4.9%)	7 (5.1%)	=0.999
Somatoform Disorders	0 (0.0%)	4 (2.9%)	=0.575
Non-IED Impulse Control Disorders	0 (0.0%)	0 (0.0%)	=0.999
Lifetime syndromal disorders:			
Any Depressive Disorder	5 (12.2%)	27 (19.6%)	<0.001**
Any Anxiety Disorder	5 (12.2%)	46 (33.3%)	=0.010
Any Substance Use Disorder	15 (36.6%)	74 (53.6%)	=0.075
Stress and Trauma Disorders	8 (19.5%)	34 (24.6%)	=0.675
Obsessive-Compulsive Disorders	3 (7.3%)	7 (5.1%)	=0.698
Eating Disorders	4 (9.8%)	19 (13.8%)	=0.604
Somatoform Disorders	0 (0.0%)	5 (3.6%)	=0.590
Non-IED Impulse Control Disorders	0 (0.0%)	5 (3.6%)	=0.590
Personality disorders:			
Any Personality Disorder	26 (63.4%)	125 (90.6%)	<0.001**
Cluster A (Odd)	1 (2.4%)	21 (15.2%)	=0.029
Cluster B (Dramatic)	12 (29.3%)	68 (49.3%)	=0.031
Cluster C (Anxious)	10 (24.4%)	42 (30.4%)	=0.558
PD-NOS	9 (22.0%)	40 (29.0%)	=0.430

*$p < .05$ after correction for multiple comparisons (uncorrected $p < .0025$).

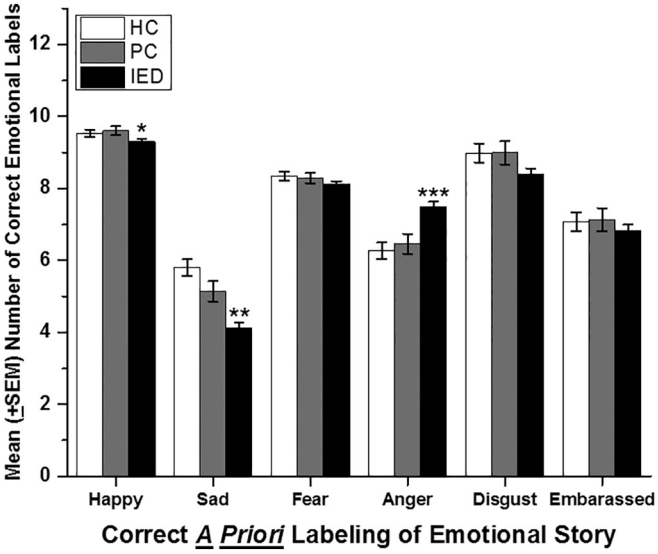

Fig. 2. Marginal means (± SEM) for MANCOVA for correct a priori emotion labels as a function of group. A single asterisk (*) indicates p value < .05 between IED and HC study participants, a double asterisk (**) indicates $p < .001$ between IED and HC study participants, a triple asterisk (***) indicates p value ≤ .001 between IED compared to PC and to HC.

uniquely predicted anger endorsement in this model ($F[9181] = 4.20$, $p < .001$; $\beta = 0.22$, $p = .001$ for LHA Aggression).

4. Discussion

Previous studies have shown the presence of negative emotional biases, particularly anger biases, in a number of disorders, including depression [46], social anxiety disorder [47], borderline personality disorder [48], and generalized anxiety and panic disorders [49], among others. In each of these studies, participants with psychiatric disorders demonstrated a significant bias towards anger stimuli compared to their healthy control counterparts. However, this is the first study to demonstrate a unique emotion attribution and recognition profile among individuals diagnosed with IED in comparison to both psychiatric controls (PC) and healthy controls (HC).

As expected, results confirm that participants with IED were more likely than PC and HC study participants to correctly identify anger stories and to misattribute anger to non-anger stories. They were also less likely than HC participants to correctly identify "sad stories." Correct anger endorsement was positively associated with the LHA, BPA, STAXI-2, and LHIB scales in bivariate analyses, with LHA Aggression uniquely predicting correct anger endorsement when these variables were considered simultaneously in a regression model. MANCOVAs were also conducted to ensure outcomes could not be attributed to the existence of a comorbid depressive or personality disorder. These analyses indicated there was no effect of comorbidity on the IED performance data.

Results from the current study provide evidence to support the validity of IED as a diagnostic entity. Specifically, IED individuals, by definition, are expected to show heightened sensitivity to provocative or threatening stimuli. Indeed, results of the study align with the idea that IED individuals are more attuned to social situations that would be expected to elicit an anger response, and are more likely to misinterpret a situation as anger-eliciting than individuals without IED. Previous literature had linked provocation to aggression [50–52], and it logically follows that the heightened sensitivity to provocation in IED, as demonstrated in this study, may meaningfully contribute to the impulsive aggression characteristic of IED as a clinical disorder [24,33,34,53,54].

A strength of this study was the use of a relatively large sample of well-characterized individuals with IED. However, future studies

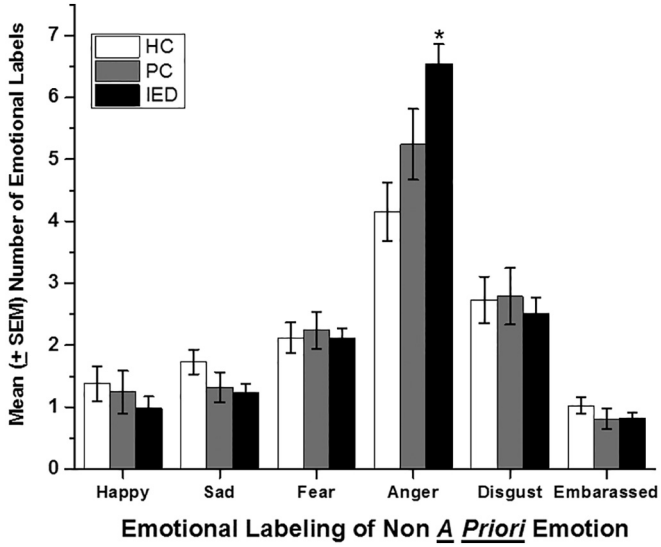

Fig. 3. Marginal means (± SEM) for MANCOVA for labeling emotions for non-a priori emotional stories (e.g., emotional label given as "sad" when a priori emotional label was "anger") as a function of group. A single asterisk (*) indicates p value <.001 between IED compared to PC and to HC.

might include larger sample sizes to confirm and refine the observed results. A limitation of the study is that the three groups significantly differed in age and sex composition, with age being lowest in the HC group and percent of men being lowest in the IED group. Future studies should aim to achieve more representative distributions of age and sex based on the general population to increase the accuracy and external validity of results. Another limitation is that most of the measures used in evaluating anger, aggression, and impulsivity consisted of self-report. Although self-report measures have clear benefits, impression management and memory biases can influence respondents' report on these measures.

Studies prior to ours have focused on facial affect recognition tasks in subjects with psychiatric disorders. This study is unlike previous studies in that it measured emotion attribution bias with respect to discrete social scenarios and anecdotes. In our age of decreasing in-person, face-to-face communication and increasing written and social media-based communication, developing an understanding of how individuals with psychiatric disorders perceive and experience emotional cues in anecdotal social interactions via social media or otherwise has the potential for providing a greater breadth of information beyond facial emotion recognition tasks alone. Additionally, the use of emotionally based vignettes provide more context to participants than facial expressions alone and future data may be used to discern how these individuals approach emotion attribution with and without a social component involved. Furthermore, emotion attribution in response to social scenario paradigms can be easily delivered to large groups of study participants via the Internet, which could lead to population-based community studies of psychopathology in the future.

Disclosures

Dr. Coccaro reports being a member and consultant to the Scientific Advisory Boards of Azevan Pharmaceuticals, Inc. and of Avanir Pharmaceuticals, Inc., and being a current grant recipient of an award from the NIMH. Dr. Berman and Ms. Patoilo have nothing to disclose.

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References

[1] Ekman P. Basic Emotions. Handb. Cogn. Emot.; 2005; 45–60. https://doi.org/10.1002/0470013494.ch3.
[2] Gottman J, Gonso J, Rasmussen B. Social interaction, social competence, and friendship in children. Child Dev. 1975;46:709–18. https://doi.org/10.1111/j.1467-8624.1975.tb03372.x.
[3] Pollak SD, Tolley-Schell SA. Selective attention to facial emotion in physically abused children. J Abnorm Psychol. 2003;112:323–38. https://doi.org/10.1037/0021-843X.112.3.323.
[4] Thomas LA, De Bellis MD, Graham R, LaBar KS. Development of emotional facial recognition in late childhood and adolescence: REPORT. Dev Sci. 2007;10:547–58. https://doi.org/10.1111/j.1467-7687.2007.00614.x.
[5] Jackson HF, Moffat NJ. Impaired emotional recognition following severe head injury. Cortex. 1987;23:293–300. https://doi.org/10.1016/S0010-9452(87)80039-4.
[6] Adolphs R, Tranel D, Damasio H, Damasio A. Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. Nature. 1994;372:669–72. https://doi.org/10.1038/372669a0.
[7] Rodger H, Vizioli L, Ouyang X, Caldara R. Mapping the development of facial expression recognition. Dev Sci. 2015;18:926–39. https://doi.org/10.1111/desc.12281.
[8] Lambrecht L, Kreifelts B, Wildgruber D. Gender differences in emotion recognition: impact of sensory modality and emotional category. Cognit Emot. 2014;28:452–69. https://doi.org/10.1080/02699931.2013.837378.
[9] Zuberer A, Schwarz L, Kreifelts B, Wildgruber D, Erb M, Fallgatter A, et al. Neural basis of impaired emotion recognition in adult attention deficit hyperactivity disorder. Biol Psychiatry Cogn Neurosci Neuroimaging. 2020. https://doi.org/10.1016/j.bpsc.2020.11.013.
[10] Bisch J, Kreifelts B, Bretscher J, Wildgruber D, Fallgatter A, Ethofer T. Emotion perception in adult attention-deficit hyperactivity disorder. J Neural Transm. 2016;123:961–70. https://doi.org/10.1007/s00702-016-1513-x.
[11] Yuan J, Tian Y, Huang X, Fan H, Wei X. Emotional bias varies with stimulus type, arousal and task setting: meta-analytic evidences. Neurosci Biobehav Rev. 2019;107:461–72. https://doi.org/10.1016/j.neubiorev.2019.09.035.
[12] Chikovani G, Babuadze L, Iashvili N, Gvalia T, Surguladze S. Empathy costs: negative emotional bias in high empathisers. Psychiatry Res. 2015;229:340–6. https://doi.org/10.1016/j.psychres.2015.07.001.
[13] Harmer CJ, Shelley NC, Cowen PJ, Goodwin GM. Increased positive versus negative affective perception and memory in healthy volunteers following selective serotonin and norepinephrine reuptake inhibition. Am J Psychiatry. 2004;161:1256–63. https://doi.org/10.1176/appi.ajp.161.7.1256.
[14] Gui W jun, Wang P yun, Lei X, Lin T, Horta M, Liu X yi, et al.. Sleep facilitates consolidation of positive emotional memory in healthy older adults. Memory. 2019;27:387–96. https://doi.org/10.1080/09658211.2018.1513038.
[15] Murphy SE, Clare O'Donoghue M, Drazich EHS, Blackwell SE, Christina Nobre A, Holmes EA. Imagining a brighter future: the effect of positive imagery training on mood, prospective mental imagery and emotional bias in older adults. Psychiatry Res. 2015;230:36–43. https://doi.org/10.1016/j.psychres.2015.07.059.
[16] Veague HB, Hooley JM. Enhanced sensitivity and response bias for male anger in women with borderline personality disorder. Psychiatry Res. 2014;215:687–93. https://doi.org/10.1016/j.psychres.2013.12.045.
[17] Manuel A, Wade TD. Emotion regulation in broadly defined anorexia nervosa: association with negative affective memory bias. Behav Res Ther. 2013;51:417–24. https://doi.org/10.1016/j.brat.2013.04.005.
[18] Auerbach RP, Stewart JG, Stanton CH, Mueller EM, Pizzagalli DA. Emotion-processing biases and resting EEG activity in depressed adolescents. Depress Anxiety. 2015;32:693–701. https://doi.org/10.1002/da.22381.
[19] Daros AR, Zakzanis KK, Rector NA. A quantitative analysis of facial emotion recognition in obsessive-compulsive disorder. Psychiatry Res. 2014;215:514–21. https://doi.org/10.1016/j.psychres.2013.11.029.
[20] Mier D, Lis S, Zygrodnik K, Sauer C, Ulferts J, Gallhofer B, et al. Evidence for altered amygdala activation in schizophrenia in an adaptive emotion recognition task. Psychiatry Res Neuroimaging. 2014;221:195–203. https://doi.org/10.1016/j.pscychresns.2013.12.001.
[21] Waters AM, Henry J, Mogg K, Bradley BP, Pine DS. Attentional bias towards angry faces in childhood anxiety disorders. J Behav Ther Exp Psychiatry. 2010;41:158–64. https://doi.org/10.1016/j.jbtep.2009.12.001.
[22] Kozlowska K, Brown KJ, Palmer DM, Williams LM. Specific biases for identifying facial expression of emotion in children and adolescents with conversion disorders. Psychosom Med. 2013;75:272–80. https://doi.org/10.1097/PSY.0b013e318286be43.
[23] El Khoury-Malhame M, Reynaud E, Soriano A, Michael K, Salgado-Pineda P, Zendjidjian X, et al. Amygdala activity correlates with attentional bias in PTSD. Neuropsychologia. 2011;49:1969–73. https://doi.org/10.1016/j.neuropsychologia.2011.03.025.
[24] Amercian Psychiatric Association. Diagnostic and statistical manual of mental disorders; 2013. https://doi.org/10.1016/B978-0-12-809324-5.05530-9.
[25] Coccaro EF. Intermittent explosive disorder as a disorder of impulsive aggression for DSM-5. Am J Psychiatry. 2012;169:577–88. https://doi.org/10.1176/appi.ajp.2012.11081259.
[26] Coccaro EF, Lee RJ. Disordered aggression and violence in the United States. J Clin Psychiatry. 2020;81. https://doi.org/10.4088/JCP.19m12937.
[27] Rynar L, Coccaro EF. Psychosocial impairment in DSM-5 intermittent explosive disorder. Psychiatry Res. 2018;264:91–5. https://doi.org/10.1016/j.psychres.2018.03.077.
[28] DeLisi M, Elbert M, Caropreso D, Tahja K, Heinrichs T, Drury A. Criminally explosive: intermittent explosive disorder, criminal careers, and psychopathology among

Federal Correctional Clients. Int J Forensic Ment Health. 2017;16:293–303. https://doi.org/10.1080/14999013.2017.1365782.
[29] Shao Y, Qiao Y, Xie B, Zhou M. Intermittent Explosive Disorder in Male Juvenile Delinquents in China. Front Psychol. 2019;10:485. https://doi.org/10.3389/fpsyt.2019.00485.
[30] Fahlgren MK, Puhalla AA, Sorgi KM, McCloskey MS. Emotion processing in intermittent explosive disorder. Psychiatry Res. 2019;273:544–50. https://doi.org/10.1016/j.psychres.2019.01.046.
[31] Garofalo C, Velotti P, Zavattini GC. Emotion regulation and aggression: the incremental contribution of alexithymia, impulsivity, and emotion dysregulation facets. Psychol Violence. 2018;8:470–83. https://doi.org/10.1037/vio0000141.
[32] Coccaro E, Fanning J, Keedy S, Lee RJ. Social cognition in intermittent explosive disorder and aggression. J Psychiatr Res. 2016;83:140–50.
[33] Coccaro EF, McCloskey MS, Fitzgerald DA, Phan KL. Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. Biol Psychiatry. 2007;62:168–78. https://doi.org/10.1016/j.biopsych.2006.08.024.
[34] McCloskey MS, Phan KL, Angstadt M, Fettich KC, Keedy S, Coccaro EF. Amygdala hyperactivation to angry faces in intermittent explosive disorder. J Psychiatr Res. 2016;79:34–41. https://doi.org/10.1016/j.jpsychires.2016.04.006.
[35] First Spitzer, Gibbon Williams, Davies Borus J, et al. The structured clinical interview for dsm-iv axis i disorders-patient edition; 1995.
[36] Pfohl B, Blum N, Zimmerman M. Structured interview for DSM-IV personality: Sidp-IV. Washington DC: American Psychiatric Press; 1997.
[37] Coccaro EF, Nayyer H, McCloskey MS. Personality disorder-not otherwise specified evidence of validity and consideration for DSM-5. Compr Psychiatry. 2012;53:907–14. https://doi.org/10.1016/j.comppsych.2012.03.007.
[38] Coccaro EF, Berman ME, Kavoussi RJ. Assessment of life history of aggression: development and psychometric characteristics. Psychiatry Res. 1997;73:147–57. https://doi.org/10.1016/S0165-1781(97)00119-4.
[39] Buss AH, Perry M. The aggression questionnaire. J Pers Soc Psychol. 1992;63:452–9. https://doi.org/10.1037/0022-3514.63.3.452.
[40] Spielberger CD. State-trait anger expression inventory: STAXI professional manual. Psychol Assess Resour. 1999 (Lutz, FL).
[41] Coccaro EF, Schmidt-Kaplan CA. Life history of impulsive behavior: development and validation of a new questionnaire. J Psychiatr Res. 2012;46:346–52. https://doi.org/10.1016/j.jpsychires.2011.11.008.
[42] Patton JH, Stanford MS, Barratt ES. Factor structure of the barratt impulsiveness scale. J Clin Psychol. 1995;51:768–74. https://doi.org/10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1.
[43] Heims HC, Critchley HD, Dolan R, Mathias CJ, Cipolotti L. Social and motivational functioning is not critically dependent on feedback of autonomic responses: neuropsychological evidence from patients with pure autonomic failure. Neuropsychologia. 2004;42:1979–88. https://doi.org/10.1016/j.neuropsychologia.2004.06.001.
[44] Hollingshead A. Four factor index of social status; 1975.
[45] Jones SH, Thornicroft G, Coffey M, Dunn G. A brief mental health outcome scale. Br J Psychiatry. 1995;166:654–9. https://doi.org/10.1192/bjp.166.5.654.
[46] Leyman L, De Raedt R, Schacht R, Koster EHW. Attentional biases for angry faces in unipolar depression. Psychol Med. 2007;37:393–402. https://doi.org/10.1017/S003329170600910X.
[47] Kim SY, Shin JE, Lee YI, Kim H, Jo HJ, Choi SH. Neural evidence for persistent attentional bias to threats in patients with social anxiety disorder. Soc Cogn Affect Neurosci. 2018;13:1327–36. https://doi.org/10.1093/scan/nsy101.
[48] Lobbestael J, McNally RJ. An empirical test of rejection- and anger-related interpretation bias in borderline personality disorder. J Personal Disord. 2016;30:307–19. https://doi.org/10.1521/pedi_2015_29_194.
[49] Ashwin C, Holas P, Broadhurst S, Kokoszka A, Georgiou GA, Fox E. Enhanced anger superiority effect in generalized anxiety disorder and panic disorder. J Anxiety Disord. 2012;26:329–36. https://doi.org/10.1016/j.janxdis.2011.11.010.
[50] Chermack ST, Berman M, Taylor SP. Effects of provocation on emotions and aggression in males. Aggress Behav. 1997;23:1–10. https://doi.org/10.1002/(SICI)1098-2337(1997)23:1<1::AID-AB1>3.0.CO;2-S.
[51] Stadler C, Rohrmann S, Steuber S, Poustka F. Effects of provocation on emotions and aggressionen experimental study with aggressive children. Swiss J Psychol. 2006;65:117–24. https://doi.org/10.1024/1421-0185.65.2.117.
[52] Taylor SP. Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. J Pers. 1967;35:297–310. https://doi.org/10.1111/j.1467-6494.1967.tb01430.x.
[53] Coccaro EF, Lee R, Gozal D. Elevated plasma oxidative stress markers in individuals with intermittent explosive disorder and correlation with aggression in humans. Biol Psychiatry. 2016;79:127–35. https://doi.org/10.1016/j.biopsych.2014.01.014.
[54] Coccaro EF, Solis O, Fanning J, Lee R. Emotional intelligence and impulsive aggression in intermittent explosive disorder. J Psychiatr Res. 2015;61:135–40. https://doi.org/10.1016/j.jpsychires.2014.11.004.