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Emotion Regulation Deficits in Intermittent Explosive Disorder

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Abstract

Intermittent explosive disorder (IED) is a psychiatric disorder characterized by repeated acts of affective aggression. Despite the diagnostic emphasis on the failure to control aggressive impulses, there is little research on affective processes and emotion regulation in IED; however, this research suggests possible dysfunctions in experiences of emotional intensity and lability. The hypothesis in the present study was that compared to individuals with other psychiatric disorders, and psychologically healthy individuals, individuals with IED experience greater negative affect intensity and emotional lability. Participants (N = 373) consisted of 202 individuals diagnosed with IED, 68 non-IED psychiatric controls (PC), and 103 healthy volunteers (HV). Emotion regulation was assessed using the General Behavior Inventory, the Affective Lability Scale, and the Affect Intensity Measure. Results showed that IED participants reported greater negative affect intensity and greater emotional lability across several emotion domains (e.g., anger, anxiety, depression) than PC and HV participants. These findings suggest that IED is characterized by more global emotion regulation deficits than those associated with anger alone.

Keywords

emotion regulation; intermittent explosive disorder; affect lability

Intermittent explosive disorder (IED) is characterized by several discrete episodes of failure to resist aggressive impulses resulting in assaults or destruction of property, with the degree of aggression being disproportionate to the provocation (American Psychiatric Association, 2000, 2013). IED has a high incidence rate, occurring in 4–7 percent of the population (Coccaro, Posternak, & Zimmerman, 2005; Kessler et al., 2006; Ortega, Canino, & Alegria, 2008) and is associated with considerable impairment in social and occupational functioning (Kessler et al., 2006), including occupational, relationship, and legal difficulties (McElroy, Soutullo, Beckman, Taylor, & Keck, 1998; McCloskey, Berman, Noblett, & Coccaro, 2006) as well as health problems (McCloskey, Kleabir, Berman, Chen, & Coccaro, 2010).

Despite the diagnostic emphasis on affective aggression and the failure to control aggressive impulses, there is little research on affective processes, and emotion regulation

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and dysregulation in IED. According to Linehan, Bohus, and Lynch (2007), emotion dysregulation refers to the inability to change or regulate emotional cues, experiences, actions, verbal, and behavioral responses. Emotion dysregulation is characterized by frequent negative emotional experiences, an inability to regulate intense physiological arousal, difficulty orienting attention away from emotional stimuli, cognitive distortions and difficulty with information processing, and insufficient control of impulsive behavior related to strong emotions (Linehan et al., 2007). One cause of emotion dysregulation according to this model is a greater vulnerability to high emotionality, which consists of heightened sensitivity to emotional stimuli, intense reactions to these stimuli, and a slow, delayed return to an emotional baseline (Linehan et al., 2007). Thus, a vulnerability to high emotionality may be reflected through the tendency of emotions to shift independently of an emotional trigger (affective lability), through the strength of one's emotional response to a stimulus (emotional reactivity), and through a generally heightened emotional intensity.

Studies comparing individuals with IED to other Axis I and Axis II psychiatric comparison groups showed that individuals with IED have higher levels of trait anger and greater affective lability associated with anger (McCloskey et al., 2006; McCloskey, Lee, Berman, Noblett, & Coccaro, 2008). Research also suggests possible dysfunctions of affective processing of a more global nature, reflected by the high degree of comorbidity associated with IED (McCloskey et al., 2006). For example, IED is frequently associated with major depression (Lejoyeux, Arbaretaz, McLoughlin, & Adès, 2002), and aggressive individuals overall tend to be more depressed and anxious than non-aggressive individuals (Galovski & Blanchard, 2002). However, the specific nature of potential deficits in affective processing in IED remains unclear due to the heterogeneity in clinical presentations of Axis I disorders and related symptoms.

Furthermore, a previous study showed some evidence of general affective lability in IED, with physically aggressive IED participants endorsing a greater tendency to vacillate from a depressed mood to an anxious or hypomanic mood and from a euthymic mood to an anxious mood than a personality-disordered comparison group (McCloskey et al., 2008). However, this study had a relatively small sample (group *N*s = 21 to 24) and divided IED participants into physically and non-physically aggressive, limiting the generalizability of the results. The present study therefore sought to replicate and expand these findings to comparisons between individuals with IED and individuals with either other psychopathology or no history of psychological problems to expand the generalizability of this effect.

In addition to a potentially greater tendency to shift emotional states, individuals with IED may also have a vulnerability to experiencing more intense negative emotions. This would be expected with respect to anger and, in fact, individuals with IED reported greater levels of hostility and anger than healthy control participants or other psychiatric control groups (McCloskey et al., 2006, 2008). The tendency of individuals with IED to interpret social information through a more hostile lens may be related to experiences of childhood trauma, as well as other familial and genetic factors (see Coccaro, 2012, for a review).

Furthermore, neuroimaging studies suggest a possible neurobiological vulnerability for intense emotional reactions and emotion dysregulation due to a dysfunction in the

amygdala-orbitofrontal cortex network in individuals with IED, a network involved in affective experiences and emotion regulation processes. Individuals with IED exhibited amygdala hyper-reactivity and orbitofrontal cortex (OFC) hypo-activation compared to healthy controls in response to ecologically-valid social threat signals (i.e., angry faces; Coccaro, McCloskey, Fitzgerald, & Phan, 2007; McCloskey, Phan, Angstadt, Fettich, & Coccaro, 2014). Since the amygdala plays a critical role in emotion processing (Sergerie, Chochol, & Armony, 2008) and the OFC is involved in the processing of rewards and punishments (Kringelbach & Rolls, 2004) as well as decision-making (Rolls & Grabenhorst, 2008), this pattern of activation indicates the possibility that individuals with IED are more vulnerable to experiencing intense negative emotions and an inability to regulate these emotions compared to healthy individuals. Although the strongest evidence for increased emotional intensity in IED is for anger, the psychiatric comorbidity among IED participants with affective and anxiety disorders would suggest that individuals with IED experience other negative emotions more intensely; however, this has yet to be directly assessed.

The present study is the first to examine emotion dysregulation with respect to both affective lability and affective intensity in a large (n = 202) sample of individuals with IED in comparison with both psychiatric and non-psychiatric controls. Our study aimed to replicate previous research with respect to affective lability in individuals with IED, and improve the generalizability of these findings through a larger sample size and control groups covering a broader spectrum of clinical symptoms. The present study is also the first to address the question of affective intensity in individuals with IED as it relates not only to anger, which is most often associated with aggressive behavior, but also to a wider range of affective experiences, thus contributing to a better understanding of the broader emotional impact of IED. We predicted that, compared to PC and HV, individuals in the IED group would exhibit greater affective intensity and greater affective lability particularly in relation to anger, anxiety and depression.

Method

Participants

Participants consisted of 373 individuals (175 men and 198 women) between the ages of 18 and 65 years (M = 35.46, SD = 10.15) recruited via public service announcements and advertisements for healthy volunteers and individuals with emotional / anger problems as a part of larger ongoing studies of anger and personality at the University of Chicago. The University of Chicago Institutional Review Board approved the protocol. All participants provided written informed consent prior to enrollment in the study. By study exclusion criteria, no subject had a life history of mania/hypomania, schizophrenia, or delusional disorder.

Participants were predominately Caucasian (61.7%) and were relatively well educated (87.7% had some college education or above). The median family income range was $25,000–34,999. The 373 participants were categorized into three diagnostic groups. The IED group [IED, n = 202] was comprised of individuals meeting DSM-V IED criteria (APA, 2013). Individuals who did not meet criteria for IED, but did meet DSM-IV criteria for another Axis I or Axis II disorder, comprised the psychiatric control group [PC, n

= 68]. Finally, healthy volunteers [HV, n = 103] denied any lifetime Axis I or Axis II psychopathology. Group assignment was based on the results of a psychiatric interview.

Diagnostic assessment.—The diagnosis of intermittent explosive disorder was made according to DSM-V criteria using information from the Intermittent Explosive Disorder Module (IED-M; Coccaro, unpublished instrument). This is a 20- to 30-minute structured diagnostic interview assessing quantitative information about current and lifetime acts of aggression (verbal and physical) to determine IED integrated research criteria, as well as DSM-IV and DSM-5 criteria. In addition to quantitative information, qualitative descriptions of aggressive episodes are also gathered (e.g., “What was the provocation?”), as well as data related to the developmental time course of these forms of behavior, including age of onset, level of distress, and impact on personal relationships.

Other psychiatric diagnoses were made according to DSM-IV criteria (APA, 2000) using information from: (a) the Structured Clinical Interview for DSM Diagnoses (SCID-I; First, Spitzer, Gibbon, & Williams, 1996) for Axis I disorders, and the Structured Interview for the Diagnosis of DSM Personality Disorders (SID-P; Pfohl, Blum, & Zimmerman, 1997) for Axis II disorders; (b) clinical interview by a research psychiatrist; and, (c) review of all other available clinical data. The diagnostic interviews were conducted by individuals with a master’s, or doctorate degree in clinical psychology. All diagnostic raters went through a rigorous training program that included lectures on DSM diagnoses and rating systems, videos of expert raters conducting SCID-I/SID-P interviews, and practice interviews and ratings until the raters were deemed reliable with the trainer. This process resulted in good to excellent inter-rater reliabilities (mean kappa of .84 ± .05; range: .79 to .93) across IED, mood, anxiety, substance use, and personality disorders. Final diagnoses were assigned by team best-estimate consensus procedures (Leckman, Sholomskas, Thompson, Belanger, & Weissman, 1982; Klein, Ouimette, Kelly, Ferro, & Riso, 1994) involving research psychiatrists and clinical psychologists as previously described (Coccaro, Kavoussi, Sheline, Lish, & Csernansky, 1996). This methodology has previously been shown to enhance the accuracy of diagnosis over direct interview alone (Kosten & Rounsaville, 1992).

Measures

The Affect Intensity Measure (AIM; Larsen & Diener, 1987) is a 40-item self-report measure designed to assess the multiple dimensions of emotional experience including positive and negative affectivity, reactivity, and affective intensity. The AIM has good internal consistency (α = .90 to .94) and test–retest reliability at 1–3 month intervals (r = .80 to .81; Larsen & Diener, 1987). Construct validity has been shown to be adequate (Coccaro, Berman, & Kavoussi, 1997). Included in the present study were the following scales based on the three-factor model identified by Bryant, Yarnold, and Grimm (1996): positive and negative reactivity scales (e.g., “If I complete a task I thought was impossible, I am ecstatic” and “Sad movies deeply touch me”) to determine the strength of an emotional response to a triggering stimulus, and the negative intensity scale (e.g., “My emotions tend to be more intense than those of most people”) to determine the strength of negative emotional reactions in general.

The Affective Lability Scale (ALS; Harvey, Greenberg, & Serper, 1989) is a 54-item scale in which people rate their agreement with statements regarding the tendency of their mood to shift between what they consider normal mood to the affective domains of anger, depression, elation, and anxiety as well as their tendency to oscillate between depression and elation and between depression and anxiety. Items were created to measure subjective experiences (e.g., “One minute I can be feeling OK and then I feel tense, jittery, and nervous”), physiological perceptions (e.g., “There are times when I’m so mad that my heart is pounding and then shortly afterwards I feel quite relaxed”), and behavior (e.g., “My sleeping patterns frequently shift from times when I have difficulty falling asleep to times when I don’t have much of a desire to sleep at all”) using six subscales (Depression, Hypomania, Biphasic, Anxiety, Anger, and Anxiety/Depression). Internal consistency and test-retest reliability for this measure have been shown to be acceptable (α = .76 to .86; r = .56 to .79; Harvey et al., 1989). Unlike emotional reactivity, affect lability refers to the tendency of mood to shift between euthymic and dysthymic in the absence of a clearly identifiable trigger stimulus.

The General Behavior Inventory (GBI; Depue & Klein, 1988) is a 73-item questionnaire that assesses chronic-intermittent forms of affective disorder rather than low-frequency episodes of affective lability. From the GBI, a total score and subscale scores for Depression and Hypomania can be derived. The GBI reliably identifies both unipolar and bipolar chronic affective disorders (Depue & Klein, 1988).

The Life History of Aggression – Aggression Scale (LHA-AS; Coccaro et al., 1997) is a 5-item retrospective semi-structured clinical interview of the frequency and severity of lifetime aggressive behavior. The LHA-AS has high internal consistency (α = .87), excellent inter-rater reliability (ICC = .95), and good test–retest reliability (r = .80; Coccaro et al., 1997).

The Buss–Perry Aggression Questionnaire (BPAQ; Buss & Perry, 1992) is a self-report measure of trait aggressiveness. The BPAQ consists of 29 items each scored using a four-point Likert-type scale. The BPAQ consists of four scales: physical aggressiveness, verbal aggressiveness, anger, and hostility (i.e., suspiciousness and resentment). The BPAQ has well-known psychometric properties (e.g., physical aggressiveness α = .85, verbal aggressiveness α = .72; anger α = .83, hostility α = .77; Buss & Perry, 1992).

Procedure

Participants were recruited at the University of Chicago and completed a 3–4 hour diagnostic interview conducted by trained graduate-level diagnosticians who were blind to the study hypotheses. Presence of personality and Axis I disorders were assessed using the SID-P and the SCID-I respectively. IED was diagnosed using the IED-M. Diagnoses were confirmed using a best-estimate procedure in which the diagnostic report was reviewed by a committee of psychiatrists, psychologists, and diagnosticians (Klein et al., 1994). Following informed consent and the diagnostic interview, subjects completed a battery of self-report measures, including the GBI, ALS, LHA, and AIM.

Data Analysis

All analyses were conducted two-tailed at the .05 level of significance. For each outcome measure a one-way (group: IED, PC, HV) MANOVA was conducted, followed by ANOVAs when the multivariate group effect was significant, and post-hoc mean comparisons [Tukey HSD] when the univariate group effect was significant. Effect sizes are provided using partial eta squared ($\acute{\eta}_p^2$) for analyses of variance. For $\acute{\eta}_p^2$, values of .01, .06 and .14 are considered small, medium and large effect sizes, respectively (Cohen, 1988). To control for multiple comparisons in these analyses only significant multivariate effects were probed. Secondary analyses consisted of only two logistic regressions.

Of the 373 participants, four failed to complete the AIM, 11 participants failed to complete the ALS, and all but one HV completed the GBI. In addition, data were missing from 13 IED, 4 PC and 6 HV participants. Missing data were deleted list-wise within each analysis, and imputation was not used.

Results

Preliminary Analyses

Demographic variables.—The groups differed with regard to age, $F(2, 370) = 10.04$, $p < .001$. Post-hoc analyses showed that the HV group was significantly, though only modestly, younger than the IED group, with no other age differences between groups. Race was dummy coded to indicate minority status. The groups were not found to differ with regard to minority status [χ^2 (2, $N = 373$) = 5.45, $p = .07$], gender [χ^2 (2, $N = 373$) = 3.01, $p = .22$] or education [χ^2 (2, $N = 373$) = 5.48, $p = .07$] (see Table 1 for demographic variable means and percentages). Correlation analyses (Table 2) indicate that with the exception of the relationship between AIM positive reactivity and GBI depression, all measures were significantly correlated with each other.

Psychiatric disorders.—As Table 3 shows, PC and IED groups did not differ in the proportion of participants with a lifetime (non-IED) Axis I diagnosis ($p = .19$). A greater proportion of IED participants relative to PC participants had a lifetime major mood disorder ($p < .001$), anxiety disorder ($p = .02$), substance dependence ($p = .02$), and personality disorder ($p < .001$). Overall, participants in the IED group had more non–IED disorders than PC participants ($p < .001$).

Aggression variables.—Preliminary analyses were conducted to compare groups on measures of aggression (LHA-AS, BPAQ). A main effect of group was observed for the LHA-AS [$F(2, 347) = 370.95$, $p < .001$, $\acute{\eta}_p^2 = .68$], where subjects with IED reported being significantly more aggressive than PC subjects, who in turn were significantly more aggressive than HV subjects (see Table 4 for means). Similarly, a one-way MANOVA on the four BPAQ scales revealed a significant multivariate effect of group, F (8, 650) = 25.05, $p < .001$. Univariate analyses showed a main effect of group for physical aggression, verbal aggression, anger, and hostility, Fs (2, 329) = 37.93 to 91.58, $p < .001$, $\acute{\eta}_p^2$=.19 to .36. Post-hoc contrasts showed that IED participants reported higher scores than PC or HV groups on physical and verbal aggression, anger, and hostility (see Table 4 for means).

Primary Analyses (see Table 5 for a list of means)

Affect intensity.—A Hotelling's Trace omnibus multivariate GLM found significant differences between groups ($F = 23.71$, $p < .001$, $\acute{\eta}_p^2 = .16$). Subsequent ANOVAs found significant effects for negative affect intensity ($F = 63.37$, $p < .001$, $\acute{\eta}_p^2 = .26$), reactivity to negative affect ($F = 4.93$, $p = .08$, $\acute{\eta}_p^2 = .03$) and reactivity to positive affect ($F = 3.26$, $p = .04$, $\acute{\eta}_p^2 = .02$). Post-hoc tests revealed that IED participants reported experiencing significantly greater negative affect intensity than PC participants, who reported experiencing greater negative affect intensity than HV participants. IED participants did not differ from PC participants on negative affect reactivity, but both IED and PC groups reported significantly greater negative affect reactivity compared to HV participants. On measures of positive affect reactivity, no statistically significant between-groups differences were found.

Affect lability.—A Hotelling's Trace omnibus multivariate GLM found significant differences between groups ($F = 38.01$, $p < .001$, $\acute{\eta}_p^2 = .39$). Subsequent ANOVAs found significant effects for depression ($F = 90.43$, $p < .001$, $\acute{\eta}_p^2 = .34$), hypomania ($F = 74.34$, $p < .001$, $\acute{\eta}_p^2 = .29$), biphasic lability ($F = 72.57$, $p < .001$, $\acute{\eta}_p^2 = .29$), anxiety ($F = 81.87$, $p < .001$, $\acute{\eta}_p^2 = .31$), anger ($F = 213.80$, $p < .001$, $\acute{\eta}_p^2 = .54$), and anxiety/depression ($F = 84.45$, $p < .001$, $\acute{\eta}_p^2 = .32$). Post-hoc analyses revealed a similar pattern of results on all lability subscales: the IED group reported greater lability on all scales than the PC group, who reported greater lability on all scales compared to the HV group.

General behavior inventory.—A Hotelling's Trace omnibus multivariate GLM found significant differences between groups ($F = 32.18$, $p < .001$, $\acute{\eta}_p^2 = .15$). Subsequent ANOVAs found significant effects for the depressive items scale ($F = 60.78$, $p < .001$, $\acute{\eta}_p^2 = .25$), and the hypomania and biphasic items scale ($F = 51.65$, $p < .001$, $\acute{\eta}_p^2 = .22$). Post-hoc tests found that IED subjects scored higher than subjects in the PC group on the depressive items scale and the hypomania/biphasic items scale, and subjects in the PC group scored higher than the HV group on both scales.

Secondary Analyses

Because HV and IED subjects differed significantly on age, primary analyses were also conducted with age as a covariate. All effects maintained significance even after controlling for age, except that IED and PC participants now differed significantly on AIM negative reactivity ($F = 3.77$, $p = .011$). Similarly, secondary analyses controlling for differences in the number of non-IED diagnoses[1] between PC and IED groups did not change the results

[1]To further assess for comorbidity issues within the IED group that may not have been captured by controlling for the total number of non–IED diagnoses, we conducted a series of hierarchical regression analyses for each outcome measure (i.e., each AIM, ALS and GBI scale - 11 analyses total) among members of the IED group. For each of these analyses we included the overall number of non-IED disorders in step 1 and four types of diagnostic comorbidity in step 2 [i.e., presence of a lifetime (a) substance dependence disorder, (b) mood disorder, (c) anxiety disorder and (d) personality disorder]. Of the 11 regression analyses, step 2 (specific diagnostic comorbidities) contributed significant variance above step 1 (the number of non-IED diagnoses) for only one scale, the GBI Depression scale [R^2 change = .056, $p < .05$]. An examination of the individual predictors in step 2 showed that, not surprisingly, only the presence of a lifetime mood disorder significantly predicted GBI Depression scores over and above the total number of non-IED diagnoses, $\beta = .21$, $p < .01$. For the other 10 regression analyses the addition of specific comorbidities in step 2 did not add significant variance beyond the total number of non-IED diagnoses.

of the primary analyses, except that IED and PD participants again differed significantly on AIM negative reactivity ($F = 3.07$, $p = .048$).

To assess which of the emotion regulation factors examined in the primary analysis independently predicted IED diagnostic status, we conducted two exploratory logistic regressions, one with IED vs. PC as the criterion and one with IED vs. HV as the criterion. For each regression all AIM, ALS, and GBI scales that were significantly different between IED and either PC (logistic regression 1) or HV (logistic regression 2) groups were entered as the predictor variables.

The first logistic regression predicting IED vs. PC status was significant for the overall model ($\chi 2$ [10] = 104.46, $p < .001$), with greater ALS anger (B = .48, $p < .001$, Wald = 40.99, odds ratio [OR] = 1.62) and at a trend level lower ALS anxiety (B = −.19, $p = .057$, Wald = 3.63, OR = .83) independently predicting IED status. The second regression predicting IED vs. HV status was also significant for the overall model ($\chi 2$ [10] = 287.66, $p < .001$), with lower ALS anxiety (B = −.59, $p < .01$, Wald = 8.13, OR = .56), higher ALS anger (B =.85, $p < .001$, Wald = 22.62, OR = 2.34) and higher GBI depression (B = .72, $p < .02$, Wald = 5.77, OR = 2.05) all independently predicting IED status.

Discussion

We predicted that individuals with IED would report both a greater tendency to experience shifts in affect (particular to anger, depression and anxiety) as well as a greater intensity of negative affect relative to individuals with either other psychiatric disorders or psychologically healthy individuals. The data supported these hypotheses. Individuals with IED reported experiencing negative emotions more intensely than individuals with other disorders and psychologically healthy subjects. Subjects with IED were also found to report more mood shifts between normal mood and anger, depression, and anxiety, and were more likely to endorse items related to unipolar and bipolar depression.

These findings are in line with previous studies suggesting that individuals with IED are more likely to experience problems with anger, depressed mood and anxiety (Lejoyeux et al., 2002; McCloskey et al., 2006), and add to the literature by showing that emotion lability in IED is not restricted to these three dimensions, but rather represents a more global feature. Individuals with IED, compared to subjects with other psychological disorders and healthy volunteers, reported not only more mood shifts from normal mood to anger, depression and anxiety, but also to feelings of elation, and more oscillations between depression and elation and depression and anxiety. This more global impairment in emotion regulation compared to other psychiatric disorders, particularly as it relates to affective intensity and lability, is not captured by the diagnostic definition of IED and has not been previously assessed empirically. These findings suggest that aggression, while the dominating symptom of this disorder, is a manifestation of a more generalized dysfunction in affect regulation. Further research is needed to begin to understand why this dysfunction manifests as aggression in some but not in others, but findings from exploratory regressions in this study seem to indicate a possible emotional pattern characterized by greater anger and lesser anxiety.

This affective lability influencing several emotional domains in IED may indicate a lack of emotional clarity. Recent research on a community sample suggests that emotional lability is negatively associated with emotional clarity as defined by the ability to understand, discriminate between, and label one's own emotions (Thompson, Dizén, & Berenbaum, 2009). However, further research is needed to examine the relationship between affective lability and emotional clarity in individuals with IED compared to subjects with other psychiatric disorders and psychologically healthy subjects.

Although subjects with IED reported greater affect lability and negative affect intensity than subjects with other psychiatric disorders, the two groups differed on negative affect reactivity only when factors such as age and number of non-IED Axis I diagnoses were controlled. However, both groups reported experiencing greater negative affect reactivity than healthy volunteers. Thus, in the presence of a clear stressor, individuals with IED experience similar pathological levels of negative emotional reactivity to emotional triggers as individuals with other disorders, but further research is needed to clarify the relationship between negative emotion reactivity, age, and comorbidity in psychiatric populations.

Although subjects with IED reported greater affect lability and negative affect intensity than subjects with other psychiatric disorders, the two groups did not differ on negative affect reactivity, with both groups reporting greater negative affect reactivity than healthy volunteers. Thus, in the presence of a clear stressor, individuals with IED experience similar pathological levels of negative emotional reactivity to emotional triggers as individuals with other disorders; however, they experience these negative emotions at a greater intensity. What appears to differentiate individuals with IED from those with other psychiatric disorders is that individuals with IED (1) are more likely to experience a negative mood shift in the absence of a clearly identifiable negative emotional trigger, and (2) will have a more intense negative emotional response in the presence of a clear stressor. The pattern of pathological negative affect lability and emotional intensity may result in more frequent or prolonged experiences of negative affect. Given the distressing nature of negative affect, individuals with IED, who experience more intense and frequent anger, likely also experience a correspondingly greater urge to engage in behavior potentiated by the negative affect such as aggression. Individuals with IED are more likely than other psychiatric groups to often refer to their anger and aggression as "out of control" (Kulper, Kleiman, McCloskey, Berman & Coccaro, 2014).

These findings are consistent with the view that individuals with IED experience more global emotion processing and regulation deficits that involve experiencing negative affect as more intense, and emotions as less stable than individuals with other Axis I and Axis II disorders overall. As expected, exploratory analyses found that when controlling for group differences in emotion regulation variables, greater labile anger predicted IED status from individuals with other psychopathology. However, a marginally significant effect was also found for lower labile anxiety independently predicting IED status from other psychopathology. When predicting IED status using HV as a reference group, greater self-reported labile anger and lower labile anxiety both significantly predicted IED status, as did greater self-reported unipolar depression. Although further research is needed to replicate and confirm these exploratory findings, our data suggest that individuals with IED

have an elevated and generalized emotional lability compared to a healthy control and a psychopathology control group, and within this generalized lability exhibit a pattern of greater labile anger and lower labile anxiety, which may differ from the pattern observed in individuals with other psychological disorders and in healthy individuals.

Because anger is conceptualized as an approach-oriented emotion and anxiety as an avoidance-oriented emotion (Carver & Harmon-Jones, 2009), this pattern may reflect functional deficits in physiological pathways related to approach and avoidance in IED, which may be associated with more frequent aggressive actions, particularly in response to anger. This hypothesis is supported by our data suggesting that subjects with IED scored higher on measures of aggression, anger and hostility (i.e., LHA-AS, BPAQ). In addition to anger and anxiety-related affective lability acting as predictors for IED status, the GBI unipolar depression index also emerged as a significant predictor differentiating between IED and healthy controls, highlighting the fact that the impairment associated with IED extends beyond the domains of anger and aggression and is associated with greater depressed mood.

Although this study addresses an important gap in the literature, these findings should be considered together with some limitations. In measuring emotional intensity and lability, this study relied exclusively on self-report measures, making it susceptible to mono-method bias. Additionally, the majority of IED subjects also had comorbid Axis I and/or II disorders, which may obscure the effect of emotion regulation in non-comorbid IED. However, using the comorbid IED group increases the ecological validity of these findings, as IED is frequently comorbid with other Axis I (e.g., depression, anxiety) and Axis II disorders (Coccaro et al., 2005).

This study is one of the first to focus on emotion processing and regulation in IED. The results suggest that emotion dysregulation may be a more global problem in IED than previously thought, and further research is needed to clarify whether the identified deficits in emotion processing and regulation function as a risk factor for IED and/or as a pathway to impulsive aggression. These findings further suggest that individuals with IED may benefit from treatments that focus not only on aggressive behavior and impulse control, but also on emotion regulation and coping strategies.

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Table 1

Demographic Variables as a Function of Diagnostic Group (N = 373)

Demographic Variable	HV (*n* = 103)	PC (*n* = 68)	IED (*n* = 202)
*** Age Mean (SD)[a]	31.90 (9.08)	34.96 (10.40)	37.24 (10.07)
Gender N (%)			
Female	52 (50%)	31 (46%)	115 (57%)
Male	51 (50%)	37 (54%)	87 (43%)
Race N (%)			
AA / Other	35 (34%)	20 (29%)	88 (44%)
Caucasian	68 (66%)	48 (71%)	114 (56%)
Education N (%)			
College	96 (93%)	61 (90%)	170 (84%)
No College	7 (7%)	7 (10%)	32 (16%)

Note: AA = African American; HV = Healthy Volunteer; PC = Psychiatric Control; IED = Intermittent Explosive Disorder.

*** $p < .001$,

[a] IED significantly different from HV.

Table 2

Inter-Correlations Among Outcome Measures (N = 373)

Measure	AIM-NR	AIM-PR	ALS-D	ALS-H	ALS-B	ALS-Anx	ALS-Ang	ALS-Anx/Dep	GBI-Dep	GBI-Hyp
AIM-NI	.52***	.44***	.65***	.59***	.59***	.67***	.60***	.67***	.57***	.55***
AIM-NR		.41***	.32***	.29***	.30***	.31***	.16**	.34***	.25***	.23***
AIM-PR			.26***	.42***	.32***	.26***	.21***	.19***	.07	.28***
ALS-D				.88***	.91***	.87***	.75***	.86***	.65***	.63***
ALS-H					.87***	.78***	.70***	.73***	.53***	.61***
ALS-B						.86***	.73***	.85***	.67***	.67***
ALS-Anx							.79***	.89***	.66***	.66***
ALS-Ang								.76***	.62***	.62***
ALS-Anx/Dep									.75***	.65***
GBI-Dep										.82***

Note:

** $p < .01$,

*** $p < .001$

AIM-NI = AIM Negative Intensity; AIM-NR = AIM Negative Reactivity; AIM-PR = AIM Positive Reactivity; ALS-D = ALS Depression; ALS-H = ALS Hypomania; ALS-B = ALS Biphasic; ALS-Anx = ALS Anxiety; ALS-Ang = ALS Anger; ALS-Anx/Dep = ALS Anxiety/Depression; GBI-Dep = GBI Depression; GBI-Hyp = GBI Hypomania

Table 3

Number and Percent of Participants with Lifetime Psychopathology as a Function of Diagnostic Group and Mean Number of Diagnoses by Group (n = 270)

Diagnoses	PC (*n* = 68)	IED (*n* = 202)	χ^2 / *t*-value
Any non-IED (%)	68 (100%)	196 (98%)	1.72
Mood Disorder (%)	23 (34%)	118 (58%)	12.31***
Anxiety Disorders (%)	14 (21%)	73 (36%)	5.63**
Substance Disorders (%)	18 (27%)	85 (42%)	5.25*
Personality Disorders (%)	26 (38%)	128 (63%)	13.43***
Mean Number of Diagnoses (SD)	1.58 (1.72)	3.00 (2.49)	4.33*

Note: PC = Psychiatric Control; IED = Intermittent Explosive Disorder;

* $p < .05$;

** $p < .01$;

*** $p < .001$

Table 4

Aggression Measures as a Function of Diagnostic Group (N = 373)

Measure	HV (*n* = 103)	PC (*n* = 68)	IED (*n* = 202)
LHA-AS[a,b,c]	5.00 (3.34)	8.08 (5.13)	18.21 (4.18)
***BPAQ – physical aggression[a,b]	16.68 (7.93)	18.47 (8.19)	27.13 (9.35)
*BPAQ – verbal aggression[a,b,c]	12.61 (3.63)	14.67 (4.04)	17.33 (4.67)
***BPAQ – anger[a,b,c]	12.83 (5.50)	15.76 (7.33)	23.91 (7.15)
**BPAQ – hostility[a,b,c]	14.53 (6.40)	19.55 (7.61)	23.98 (7.62)

Note: HV = Healthy Volunteer; PC = Psychiatric Control; IED = Intermittent Explosive Disorder; LHA-AS = Life History of Aggression – Aggression Scale; BPAQ = Buss–Perry Aggression Questionnaire;

*$p < .05$,

**$p < .01$,

***$p < .001$,

[a]IED significantly different from PC,

[b]IED significantly different from HV,

[c]PC significantly different from HV

Table 5

Emotion Regulation Measures as a Function of Diagnostic Group (N = 373)

Measure	HV (*n* = 103)	PC (*n* = 68)	IED (*n* = 202)
Affect Intensity Measure			
***Negative Intensity[a,b,c]	13.81 (4.73)	18.75 (6.95)	21.51 (5.56)
**Negative Reactivity[b,c]	20.65 (4.78)	22.58 (4.88)	22.43 (5.20)
*Positive Reactivity[b]	51.17 (12.02)	54.20 (12.68)	55.16 (13.43)
Affective Lability Scale			
***Depression[a,b,c]	15.44 (4.73)	20.98 (7.25)	25.75 (6.66)
***Hypomania[a,b,c]	17.40 (5.66)	22.75 (7.63)	27.63 (7.23)
***Biphasic[a,b,c]	11.45 (3.27)	15.52 (5.95)	19.45 (6.17)
***Anxiety[a,b,c]	8.35 (2.56)	11.56 (5.05)	14.80 (4.49)
***Anger[a,b,c]	7.89 (1.99)	10.63 (4.32)	17.96 (4.94)
***Anxiety/Depression[a,b,c]	9.22 (2.58)	13.39 (5.90)	17.44 (5.93)
Global Behavior Inventory			
***Depressive[a,b,c]	0.29 (0.80)	6.47 (10.32)	12.72 (11.22)
***Hypomania/Biphasic[a,b,c]	0.51 (1.46)	3.06 (5.16)	6.36 (5.74)

Note: HV = Healthy Volunteer; PC = Psychiatric Control; IED = Intermittent Explosive Disorder;

* $p < .05$,

** $p < .01$,

*** $p < .001$;

[a] IED significantly different from PC,

[b] IED significantly different from HV,

[c] PC significantly different from HV