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Subtypes of aggression in Intermittent Explosive Disorder

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Abstract

Research in aggression has distinguished two major subtypes of aggressive behavior: hostile and instrumental. Previous research has examined these subtypes in healthy individuals and forensic samples but not in intermittent explosive disorder (IED), a disorder characterized by recurrent and severe aggressive behavior. We examined aggression subtypes in individuals with IED, healthy subjects, and psychiatric control subjects. We also considered the relationship between aggression subtypes and measures of trait anger and impulsivity to evaluate whether the hostile/instrumental dichotomy adequately captures the heterogeneity of aggressive behavior in this sample. Finally, we consider the implications of these results for research on aggression, including neurobiological research on aggression

Keywords

impulsive aggression; premeditated aggression; intermittent explosive disorder; Research Domain Criteria (RDoC); instrumental aggression

Introduction

Aggression is a destructive behavior that imposes a considerable burden on individuals and society. In clinical settings, recurrent problematic aggression is identified by intermittent explosive disorder (IED) which is the categorical psychiatric construct for pathological aggression (American Psychiatric Association, 2013). IED has a lifetime prevalence of around 7% (Kessler et al., 2006). Investigations of IED have revealed associated biological abnormalities in central neurotransmitter function (Coccaro, Fanning, Phan, & Lee, 2015; Coccaro, Lee, & Vezina, 2013), peripheral biological markers (Fanning, Lee, Gozal, Coussons-Read, & Coccaro, 2015), and brain structure and function (Yang & Raine, 2009). However, a growing literature supporting the dimensionality of many constructs in psychopathology has shifted the paradigm for studying psychiatric disorders away from categorical diagnostic models and toward modeling psychopathology along transdiagnostic dimensions of behavior. This model of psychopathology has been adopted by the National Institute of Mental Health (NIMH), a key source of funding for mental health research in the United States (Cuthbert, 2014). The Research Domain Criteria (RDoC) initiative by the

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NIMH furthers the movement toward adopting dimensional models of behavioral dysfunction.

There is a long tradition in the aggression literature of distinguishing among subtypes of aggression. Most subtyping schemes dichotomize aggression into two forms, which are distinguished by the motivation for the aggressive behavior. Reactive (or hostile) aggression is considered to: (a) be driven by a proximal desire to hurt the target; (b) occur in the presence of anger; and (c) involve little forethought or planning (Bushman & Anderson, 2001; Dodge & Coie, 1987; Dodge, 1991; Evans, 1961; Feshbach, 1964; Kempes, Matthys, De Vries, & Van Engeland, 2005). Instrumental (or premeditated) aggression is: (a) motivated proximally by a reinforce or goal (e.g., to obtain a reward, establish one's image, or restore justice); (b) not primarily driven by anger; and (c) planned (Evans, 1961; Bushman & Anderson, 2001). Note that in the case of instrumental aggression, planning does not have to be lengthy and may constitute a snap decision to use aggression to achieve some goal, for example, upon spotting a vulnerable target, suddenly pushing someone to steal their property. Indeed, psychopathic individuals have been described as engaging in behavior that is "impulsively instrumental" (Hart & Dempster, 1997). The notion that subtypes of aggression occur in different situational contexts, and have distinct antecedents and psychological correlates supports the rationale for the subtyping approach (Dodge & Coie, 1987; Dodge, 1991; Raine et al., 2006).

Several self-report measures assess aggression subtypes in adults. These measures support the subtyping approach by showing differential correlations across subtypes. For example, researchers using the Reactive Proactive Questionnaire (RPQ; (Raine, et al., 2006) have found that reactive aggression is more closely related to anxiety, hostility, and impulsivity, while proactive aggression is more closely linked to callousness, substance use, and delinquency (Cima, Raine, Meesters, & Popma, 2013; Miller & Lynam, 2006). Researchers using the Aggressive Acts Questionnaire (AAQ) have found that participants who rated their aggression as *impulsive* report more anger, hostility, and trait impulsivity, while *premeditated* aggression was negatively associated with anger and hostility and was not associated with trait impulsivity (Smith & Waterman, 2006). Factor analytic studies further support the subtyping of aggression into separate constructs (Barratt, Stanford, Dowdy, Liebman, & Kent, 1999; Poulin & Boivin, 2000; Raine, et al., 2006).

A separate viewpoint is that aggressive behavior can have mixed motivations and can thus embody different subtypes simultaneously. This has been argued by Bushman and Anderson (2001; among others; Evans, 1961; Feshbach, 1964), who note that harming a target is a goal for both hostile and instrumental aggression and that aggression that is motivated by anger is not always impulsive (for example, revenge). Others have argued that even aggression which appears to be reactive aggression is actually a form of coercive behavior, and that instrumental aggression can be carried out impulsively with very little planning (Hart & Dempster, 1997; Tedeschi & Felson, 1994). Indeed, reactive and proactive aggression are often highly correlated (e.g., r = 0.41–0.87; Baker, Raine, Liu, & Jacobson, 2008; Dodge & Coie, 1987; Fite et al., 2010; Polman, Orobio De Castro, Koops, Van Boxtel, & Merk, 2007; Poulin & Boivin, 2000; Raine, et al., 2006) and can be difficult to distinguish at the level of the aggressive act (Barratt, et al., 1999).

Although subtyping of aggression has been discussed in the literature for many years, it continues to have relevance today given the emphasis on understanding the role of brain functioning in human behavior. It is reasonable to hypothesize that subtypes of aggression, which are proposed to have distinct motivational dimensions and psychological, social, and emotional correlates, will depend on distinct neural systems. However, the literature on aggression subtypes in adults is small relative to that in children (Dodge & Coie, 1987; Dodge, 1991). Overall, this research supports the existence of at least the two main aggression subtypes in adults. Research shows that adults (e.g., college students, violent-offenders, and partner-violent men) report engaging in both impulsive and premeditated aggressive behavior, but that aggressive behavior in these groups is more often impulsive (i.e., reactive) rather than instrumental (Barratt, et al., 1999). Psychopathic individuals, who are antisocial, impulsive, and lack empathy, may be more prone to instrumental aggression (Cornell et al., 1996; Williamson, Hare, & Wong, 1987). Together these studies show that aggression subtypes are evident in adults and that aggressive behavior is heterogeneous in motivation but is more often reactive in nature.

Little is known about subtypes of aggression in intermittent explosive disorder (IED), a psychological disorder characterized by recurrent aggressive behavior. In the current study, we examine: (a) whether research participants with IED characterize their aggression with respect to impulsive or premeditated subtypes; (b) whether IED subjects differ from healthy individuals and psychiatric control subjects in relative engagement in reactive and instrumental subtypes of aggressive behavior; and (c) the contribution of trait anger and trait impulsivity to each of the aggression subtypes. Finally, we examined the overlap between different subtypes of aggression (impulsive, premeditated, expressive, and instrumental) to test the fit of the two-factor approach to aggression in this sample. We expected that both IED and control subjects would describe their aggression as predominantly impulsive (versus premeditated) and expressive (versus instrumental). We also predicted that trait anger and impulsivity would be more closely related to "reactive" forms of aggression. Finally, we hypothesized that factor analysis of the subtypes would support the hostile/instrumental aggression dichotomy.

Methods

Subjects.

Subjects (n=860) were recruited through public service announcements, newspaper, and other media advertisements seeking out individuals who: (a) reported psychosocial difficulty related to personality disorder traits or aggressive behavior, or (b) had little evidence of psychopathology, to participate in research on the correlates of personality and aggressive behavior. Exclusion criteria included: current substance use disorder, life history of bipolar disorder or psychotic disorder, and significant intellectual disability. All subjects gave informed consent in accordance with procedures approved by the local Institutional Review Board.

Diagnostic Assessment.

Psychiatric and personality disorder diagnoses were made by DSM-5 criteria (American Psychiatric, 2013) using information from: (a) the Structured Clinical Interview for DSM Diagnoses (SCID-I; First, Spitzer, Gibbon, & Williams, 1997) and the Structured Interview for the Diagnosis of DSM Personality Disorder (SIDP; Pfohl, Blum, & Zimmerman, 1997) and (b) clinical interview. Diagnoses of IED were made using a structured clinical interview, which yields a diagnosis of IED equivalent to the DSM-5 diagnostic criteria (Coccaro, Lee, & McCloskey, 2014). The diagnostic interviews were conducted by individuals with a masters or doctorate degree in psychology who completed a rigorous training program on DSM diagnoses. This process resulted in good to excellent inter-rater reliabilities (mean kappa of $.84 \pm .05$; range: .79 to .93) across anxiety, mood, substance use, impulse control, and personality disorders. Final diagnoses were assigned by team best-estimate consensus procedures as previously described (Coccaro, Nayyer, & McCloskey, 2012).

After diagnostic assignment, 284 subjects had no evidence of any psychiatric diagnosis (healthy controls: HC); 311 subjects met criteria for a lifetime diagnosis of a syndromal or personality disorder, but not lifetime IED (psychiatric controls: PC); and 265 subjects met criteria for current (n=128) or lifetime (n=137) IED. Of the 576 subjects with any diagnosis, most (66.8%) reported: (a) a history of formal psychiatric evaluation and/or treatment or, (b) a history of behavioral disturbance during which the subject or others thought they should have sought mental health services. Diagnoses among PC and IED subjects are listed in Table 2.

Measures

Aggressive Acts Questionnaire.—The AAQ assesses the nature of self-reported, overtly aggressive behavioral acts (Barratt, et al., 1999). Subjects were asked to report the number of extreme or inappropriate aggressive acts (including physical and verbal acts) in which they had engaged during the previous six months. Subjects then rated up to four acts on 22 Likert-scaled items assessing the impulsiveness (e.g., "I lacked self-control during the act") and premeditation of the acts (e.g., "the act was planned"; "I profited financially from the act"). Items were scored from 1 ("definitely not") to 5 ("definitely yes"). Factor analysis of the original AAQ revealed four scales including impulsive and premeditated aggressive acts; items reflecting the two other factors (general mood and agitation) were not included in this study. Alpha coefficients for the first act were $\alpha = 0.74$ and $\alpha = 0.54$ and were higher when multiple acts were included.

Expressive and Instrumental Representation of Aggression (ExpAgg).—

Subjects who completed the full AAQ were also given the ExpAgg. ExpAgg data was available in n=252 subjects. The ExpAgg is a 16-item questionnaire that assesses how much the subject views their aggression as representing expressive (e.g., "I believe my aggression comes from my losing my self-control") and instrumental (e.g., "I believe that physical aggression is necessary to get through to some people") motivation. Scores range from 1 ("strongly disagree") to 5 ("strongly agree"). Internal consistencies (a) were 0.79 (expressive) and 0.87 (instrumental).

Trait Aggressive Behavior, Trait Impulsive Behavior, Trait Anger and

Psychopathy.—Life history of aggression and of impulsive behavior was assessed with the aggression score from the Life History of Aggression scale (LHA; Coccaro, Berman, & Kavoussi, 1997) and the impulsivity score from the Life History of Impulsive Behavior scale (LHIB; Coccaro & Schmidt-Kaplan, 2012). LHA Aggression assesses the historical frequency of actual aggressive behavior and has good internal consistency (α = .87) and testretest reliability (r = .80). The 20-item, 5-point ordinal scale LHIB assesses history of actual impulsive behavior (Coccaro & Schmidt-Kaplan, 2012). The LHIB demonstrates good internal consistency (α = .96) and test-retest reliability (r = .88). Trait anger was assessed with the anger subscale of the Buss Perry Aggression Questionnaire (BPAQ; α = 0.83; Buss & Perry, 1992). Psychopathy was assessed using the Psychopathy Checklist Screening Version (PCL-SV; Hart, Cox, & Hare, 1995), administered during the diagnostic assessment interview. PCL-SV scores range from 0 to 24 with scores of 13 and higher suggesting possible psychopathic personality.

Clinical Measures.—Measures of current symptom severity for depression and anxiety were used to control for the impact of current mood symptoms on self-report ratings in multiple regression analyses. Current (past 2-week) depression symptom severity was assessed using the Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). Current anxiety (past week) was assessed using the Beck Anxiety Inventory (BAI; Steer & Beck, 1997).

Statistical Analysis

Analyses were performed using SPSS 22. Between group comparisons were performed using t-test, ANOVA and MANOVA, and Tukey's post hoc test. Correlational analyses were conducted using Pearson correlation, hierarchical multiple regression analysis, and Fisher's exact test (FET). AAQ and ExpAgg scales were evaluated dimensionally and categorically. For categorical analyses of the AAQ, participants were classified as "mostly impulsive", "mostly premeditated", or "equally both" based on whether they endorsed more items (weighted by subscale) as clearly impulsive versus clearly premeditated. ExpAgg scores were treated similarly. Finally, exploratory factor analysis (EFA) was conducted on items from the two measures. To the extent that the four subscales reflect two higher-order aggression subtypes, we expected that items from the AAQ and ExpAgg subscales would load onto two factors reflecting hostile and instrumental aggression. For the AAQ, only items from the first aggressive incident were used in this analysis. A two-tailed alpha value of 0.05 was used to denote statistical significance.

Results

Characteristics of the Sample (Table 3).

Differences between groups are shown in Table 2. Adjusting for these differences did not affect the results so analyses are reported using unadjusted data. As expected, IED subjects had higher aggression, impulsivity, and anger scores (IED > PC > HC).

Group Differences: AAQ (Table 4).

Because serious aggressive acts are relatively rare, most subjects (n = 551) reported no serious aggressive acts on the AAQ during the preceding six months. Among the 309 subjects who reported aggressive behavior, 108 reported and scored four acts, 41 three acts, 65 two acts, and 95 one act (mean \pm sd: 2.5 ± 1.3). Impulsive and premeditated aggression scores were unrelated to the number of aggressive acts reported (Wilks λ = .97, F[6, 608]=1.37, p = .225). IED subjects reported the greatest mean number of serious aggressive acts followed by PC and HC subjects, who did not differ (see Table 4). Current IED subjects reported a greater mean number of serious aggressive acts (19.4 \pm 66.3) compared with past IED subjects (2.7 \pm 6.3), who did not differ from PC and HC subjects.

For subjects who reported at least one aggressive act, MANOVA (Wilks λ =0.85, F[4, 610] = 13.05, p <.001) revealed higher impulsive aggression scores for IED (F[2, 306] = 22.06, p <.001), compared with both HC (p < .05) and PC (p = .053) subjects. ANOVA for premeditated aggression scores revealed only a statistical trend for a difference among the groups (F[306] = 2.56, p = .079). IEDs and HCs rated their aggressive acts as marginally more premeditated compared to PCs.

Next, the patient groups were compared on their categorical designations on the AAQ. Most subjects (n = 275, 89.0%) rated themselves as predominantly impulsively aggressive; 4.5% were predominantly premeditated and 6.5% were both equally. The pattern of scores differed across the three groups (FET p <.001). IED subjects were less likely to be classified as both impulsive and premeditated (2.0%) compared to PC (13.5%) and HC (16.2%) subjects. There was no other differences between groups in the percent classified as mostly impulsive (HC = 78.4%, PC = 83.8%, IED = 92.9%) or mostly premeditated (5.4%, 2.7%, 5.1%).

Relationship Between AAQ Variables and Comorbidity with Syndromal Disorders.

Impulsive aggression scores were higher in IED subjects (IED: 3.4 ± 1.6 ; non-IED: 3.1 ± 1.0) even when controlling for lifetime comorbid syndromal disorders (listed in Table 2; F[1, 293] = 6.58, p = .011) and when controlling for borderline (BPD) and antisocial personality disorder (ASPD), F[1, 305] = 4.99, p = .026. No syndromal or personality disorder predicted impulsive aggression scores when IED diagnosis was accounted for. Premeditated aggression scores did not differ as a function of IED when BPD and ASPD were examined in the same model.

Group Differences: ExpAgg (Table 4).—MANOVA (Wilks λ =0.85, F[4, 486] = 10.50, p < .001) revealed that IED subjects rated their aggression as more expressive (M = 26.7, SE = .35, F[2, 244] = 12.14, p < .001) compared with both HC (M = 20.9, SE = .42) and PC (M = 22.8, SE = .35) subjects. IED subjects also rated their aggression as more instrumental (M = 22.6, SE = .46) than HC (M = 16.8, SE = .43) and PC (M = 17.3, SE = .35) subjects. When categorical scores were examined, the groups differed in their classifications (FET p < .001). IED subjects were less likely to be classified as both expressive and instrumental (HC = 28.8%, PC = 18.4%, IED = 10.4%) and were more likely to be classified as instrumentally aggressive (HC = 11.5%, PC = 11.0%, and IED = 27.1%). The three groups were similarly likely to be classified as mostly expressive (59.6%, 70.6%, and 62.4%).

Relationship Between AAQ and ExpAgg (Table 5).—Correlations between subscales were conducted to assess the similarity of the measures. Impulsive aggression (AAQ) and expressive aggression (ExpAgg) correlated modestly (r=.30), as did premeditated aggression (AAQ) and instrumental aggression (ExpAgg; r=.25). Impulsive aggression did not correlate with either premeditated or instrumental aggression (see Table 5). The correlation between expressive and instrumental aggression scales was high (r=.56). The conceptual overlap between these measures was also assessed using the categorical designations. A 3 × 3 FET was conducted to study whether subjects classified as impulsive were also classified as expressive and whether those classified as premeditated were classified as instrumental. The FET was non-significant (p=.131) indicating that classification on one measure did not predict classification on the other measure. This held true when the scales were treated dichotomously (p=0.12).

Relationship Between Aggression Subtype Scores and Impulsivity, Anger, Aggression, and Psychopathy (Tables 5 and 6).

We examined the relationship between dimensions of aggression and trait impulsivity, anger, aggression, and psychopathy. Based on definitions of impulsive aggression as affect-laden (Evans, 1961) we expected that impulsive and expressive dimensions of aggression would be most closely related to anger and impulsivity (Bushman & Anderson, 2001; Raine, et al., 2006). We also expected that psychopathy scores would correlate with instrumental and premeditated aggression (Porter & Woodworth, 2006; Williamson, et al., 1987). Impulsive aggression correlated moderately with BPAQ anger (r = 0.25) and LHIB impulsivity (r = (0.22) and not with psychopathy (r = -0.01). A similar pattern was observed for expressive aggression (ExpAgg) scores (see Table 5). Premeditated aggression did not correlate significantly with anger, impulsivity, or psychopathy, while ExpAgg instrumental aggression correlated moderately with impulsivity (r = 0.33) and anger (r = 0.41), and non-significantly (r=0.02) with psychopathy. These patterns generally held when multiple regression analyses were used to examine the unique influence of impulsivity and anger on the four aggression subtypes, controlling for the impact of current depression and anxiety on self-ratings. Impulsivity was not a significant predictor (p = .10) of impulsive aggression when considered alongside anger (see Table 6). Both anger and impulsivity significantly predicted expressive and instrumental aggression, and neither anger nor impulsivity predicted premeditated aggression. Current depression and anxiety showed little impact on these relationships.

Factor Analysis of Aggression Items (Table 7).

Items from the four subscales were entered into an EFA using principal components analysis with varimax rotation. Using mean substitution for missing items, data from 729 subjects were available (a ratio of 30 cases per item). Four factors with eigenvalues over one explained 51% of the variance (21%, 12%, 11%, and 7% respectively) following rotation. Eleven items loaded onto Factor 1 (with factor loadings of .4 or greater). These included the eight items from the ExpAgg instrumental aggression subscale and three items from the ExpAgg expressive subscale that cross-loaded (.50 or greater) onto this factor and onto Factor 2 (see Table 7). Factor 2 included the eight items from the ExpAgg expressive aggression subscale. The five items loading onto Factor 3 were from the AAQ impulsive

aggression subscale, while the three items loading onto Factor 4 were from the AAQ instrumental aggression subscale. In general, with the exception of the three cross-loading items, the observed factor structure closely reflects the original four subscales from which the items were drawn, rather than theoretical hostile-instrumental dichotomy.

Discussion

This study examined self-reported subtypes of aggression in healthy individuals, IED, and mixed psychiatric controls. Two measures assessed subjects' aggressive behavior along: (a) *impulsive* and *premeditated* dimensions, and (b) *expressive* and *instrumental* dimensions. Impulsive and expressive dimensions theoretically represent subtypes of hostile aggression, while premeditated and instrumental aggression theoretically reflect instrumental aggression within the hostile-instrumental aggression theoretical framework. Subjects in all three groups considered their aggression to be primarily impulsive and expressive in motivation; however, we did observe heterogeneity in subjects' attributions of their aggressive behavior.

When categorizing past 6-month aggressive acts on the AAQ as impulsive or premeditated, most subjects (close to 90%) described their individual aggressive acts as predominantly impulsive. This pattern held even at the group level. This accords with previous research that respondents describe their aggressive behavior as more reactive than proactive (Baker, et al., 2008; Cima, et al., 2013; Fung, Raine, & Gao, 2009; Raine, et al., 2006; Smith & Waterman, 2006). When categorizing aggression as predominantly expressive versus instrumental (ExpAgg), subjects (across all groups) were more likely to describe their aggression as predominantly expressive (66%) rather than instrumental (22%). IED subjects described their aggression as more instrumental (i.e., outcome-oriented) compared to PC and HC subjects.

On the AAQ, IED subjects described their aggression as dimensionally more impulsive than HC and PC subjects. This finding mirrors an earlier finding that violent offenders described their aggression as more impulsive compared to undergraduate students (Smith & Waterman, 2006). These results were not affected by controlling for comorbid psychiatric disorders, including BPD and ASPD. IED subjects also rated their aggression as more expressive compared to PC and HC subjects. IEDs and HCs rated their aggression as marginally more premeditated than PCs. However, after controlling for comorbid BPD and ASPD, IED was not associated with greater premeditated aggression. Thus, premeditated aggression might only be higher in IED subjects with BPD or ASPD psychopathology.

Borderline and antisocial personality disorders include anger and aggression among the criteria (APA, 2013), and research from this lab shows that a significant proportion of IED subjects recruited from the community have comorbid BPD (22%), ASPD (10%), or both (11%; Coccaro, Shima, & Lee, 2018). However, while the combination of IED and BPD or ASPD is associated a higher degree of aggressiveness, IED alone is associated with significantly more aggression than are BPD and ASPD alone. In contrast, subjects with IED report significantly less impulsivity compared to subjects with BPD and ASPD. In the current study, we found that IED subjects described their aggression as more impulsive than PC and HC subjects, and these differences were not attributable to BPD or ASPD diagnoses.

Subjects with IED have also been shown to have significant comorbidity with other PDs, including narcissistic PD, obsessive compulsive PD, and paranoid PD. Personality disorder traits that are associated with IED include: anger and affective instability (BPD), irritability/ aggressiveness (ASPD), bearing grudges (paranoid PD), arrogance (narcissistic PD), suspiciousness (schizotypal PD), and rigidity/stubbornness (obsessive-compulsive PD). However, IED does not appear to be particularly related to the antisocial PD trait of deceiving or conning others, which might be related to premeditated aggression (Coccaro et al., 2018). Empirical studies have linked BPD, antisocial personality traits, and psychopathy to premeditated aggression (Ostrov & Houston, 2008; Porter & Woodworth, 2006; Stanford et al., 2003). Overall we did not find that clinical variables, including psychopathy, predicted self-reported premeditated aggression. The ability to detect a relationship between premeditated aggression and psychopathy in the current study may be limited by the restricted range of psychopathy in the sample, as evidence of clinically significant psychopathy was exclusionary for study participation. Notably, premeditated aggression was the one form of aggression that did not differ between the IED subjects, healthy controls, and psychiatric controls.

Associations between measures yielded both expected and unexpected findings. As expected, impulsive aggression correlated moderately with expressive aggression, was inversely associated with premeditated aggression, and was not significantly correlated with instrumental aggression. Premeditated aggression correlated moderately with instrumental aggression and was unrelated to expressive aggression. Unexpectedly expressive and instrumental aggression were strongly correlated. As expected, trait anger and impulsivity correlated in the small-to-moderate range with impulsive and expressive aggression, and did not correlate with premeditated aggression scores. Unexpectedly, anger and impulsivity correlated moderately with instrumental aggression. Reactive forms of aggression (i.e., impulsive and expressive aggression) are typically regarded as occurring during intense emotional arousal and as representing a loss of control. However, these results suggest that anger and impulsivity are involved in instrumental aggression as well. This is at odds with the notion that instrumental aggression represents a "cold", calculated, and controlled behavior. It is, however, consistent with models of aggression that posit mixed motivations for aggressive behavior as well as models positing that aggression is a form of coercive influence (Bushman & Anderson, 2001; Tedeschi & Felson, 1994).

Finally, factor analysis of the aggression items was not consistent with two higher order aggression subtypes reflecting the hostile-instrumental dichotomy. Rather, the resulting factors in general reflected the original scale composition, with distinct factors for impulsive, expressive, instrumental, and premeditated aggression. Together with the unexpected strong correlation between instrumental and expressive aggression, and the positive correlations between instrumental aggression, anger, and impulsivity, these findings point to heterogeneity in aggressive behavior that is not fully accounted for by the two-factor model. These findings are consistent with the view of aggressive behavior described by Bushman and Anderson (2001) that aggressive behavior often reflects mixed motivations. The findings are also consistent with observations that instrumental behavior can be impulsive and not strictly in a "cold-blooded" and planned (Hart & Dempster, 1997).

Contemporary neuroscience methods, including functional magnetic resonance imaging (fMRI), are significantly advancing our understanding of the neurobiological basis of aggressive behavior and to address some of the theoretical issues discussed here (Beyer, Münte, Erdmann, & Krämer, 2013; Krämer, Jansma, Tempelmann, & Münte, 2007). The NIMH RDoC initiative, for example, seeks to link transdiagnostic dimensions of behavior to specific underlying biological processes. Neuroimaging studies of clinical populations characterized by impulsive aggression (IED) and premeditated aggression (psychopathy) point to abnormal function in overlapping and distinct neural circuits. For example, research subjects with IED show relatively diminished activity in orbitofrontal cortex (OFC), relative hyperactivity of amygdala, and abnormal OFC-amygdala functional connectivity when viewing threatening (angry) faces (Coccaro et al., 2007; McCloskey et al., 2016). In contrast, clinical populations with psychopathy traits (i.e., callous-unemotional traits), show reduced activity in brain regions (such as amygdala) supporting affective process (e.g., Seara-Cardoso & Viding, 2014). Given ongoing questions as to the nature of aggressive behavior (its factor structure and core dimensions) and evidence of the heterogeneity of the behavior, certain research practices would increase the likelihood that research conducted within the dimensional framework will yield informative, reliable, and meaningful insights into the neurobiology of aggression. First, researchers should be explicit about the model of aggression on which the research is based (e.g., reactive versus instrumental aggression), and whenever possible, evaluate the fit of different models of aggression. Where possible, the proposed model should be supported by state and trait assessments such as self-report and mood rating scales, observer ratings, and psychophysiological measures. Several trait questionnaires, described earlier, are available for assessing aggression subtypes; however, those that assess only two subtypes of aggression may not fully capture the heterogeneity of aggressive behavior. Scales (either individually or combined) should assess emotional/ reactive aggression, instrumental aggression (both premeditated and impulsive) and frustrated aggression, as these dimensions may implicate distinct neurobiological systems. This may warrant the development of new scales (or combining of existing scales) as we are aware of no scales specific to aggression that assess all of these dimensions. Furthermore, as we found here, the hostile-instrumental dichotomy of aggression may be insufficient to capture the heterogeneity of aggressive behavior. In the case of research that assesses individual aggressive acts or laboratory paradigms that simulate aggressive interactions, it would be beneficial to assess subjects' attributions of the aggressive acts along relevant dimensions (hostility, impulsivity, and potential rewards and consequences) and to include these measures in statistical models in order to evaluate these motivations in relation to the biological system being studied. This approach may be particularly informative in light of Bushman and Anderson's (2001) perspective on mixed aggression subtypes, and Barratt's (1999) finding on the difficulty of distinguishing reactive versus instrumental behavior. Finally, given the significant correlations between subtypes, these assessments should be independent (not bipolar) to allow for correlation across dimensions.

Two examples illustrate how including state and trait measures into the research design facilitates identification of the relevant psychological and neurobiological processes underlying aggressive behavior. Verona, Patrick, and Lang (2002) used a laboratory paradigm to simulate an aggressive interaction. They investigated whether participants high

in negative affect (i.e., high NEM) would show greater aggression during a frustrating task and whether this effect would be attributable to increased reactivity to discrete aversive stimuli (a phasic negative affect) or to sustained elevations in negative mood (a tonic negative affect). They assessed trait negative affect, state psychophysiological measures of phasic negative affect (startle blink potentiation) and tonic negative affect (startle sensitization), and subjects' self-reports of hostile versus instrumental motivation during the task. They found that subjects high in NEM were more aggressive overall (delivering more intense shocks to a confederate) and that this result was related to tonic increases in negative affect (following experimental manipulation) in the high NEM group rather than increased emotional reactivity to discrete provoking stimuli. On average, participants rated their motivation as more instrumental than hostile; however, high NEM participants rated their motivation as both more hostile and more instrumental than low NEM participants. Selfreported hostile and instrumental motivations were both related to the intensity of shock delivered (in both groups). In an fMRI study, Beyer and colleagues (2013) examined the relationship between emotional reactivity (i.e., startle potentiation to threatening versus neutral pictures), aggressive behavior, and fMRI BOLD activity in a neural network engaged during a simulated reactive aggressive interaction. Although startle potentiation was unrelated to aggressive behavior in their sample of healthy individuals, it was inversely related to provocation-related activity in the prefrontal cortex and to activity in brain regions that support mentalizing when the subject was interacting with a provocative (compared to neutral) opponent. The authors concluded that greater emotional reactivity to threat is associated with decreased activity in the mentalizing neural network during a provocative interaction.

Over the years many theoretical models of aggressive behavior have been proposed. Some of these proposed broad explanations for aggressive behavior, for example, the original frustration-aggression and social learning theories (Bandura, 1973; Dollard, et al., 1939). Recent theories have, in general, have attempted to account for the heterogeneity of aggressive behavior (e.g., General Aggression Model; Anderson & Bushman, 2002; Berkowitz, Mowrer, & Sears, 1989; Berkowitz, 1990). The subtyping approach to aggression is currently well-accepted in aggression research, with most models reflecting a version of the hostile-instrumental dichotomy. This approach is supported by factor analytic studies and empirical studies showing differential correlates of aggression subtypes. In the current study, we found that research participants provided varied explanations for their aggressive behavior, although reactive explanations predominated. To the extent that these subtypes are valid (distinct and reliable), they should show also distinct neural correlates, particularly where one subtype (premeditated aggression) conceptualizes aggression as a controlled behavior and others (impulsive aggression) as a loss of control due to impulsivity or emotional arousal (Campbell, Muncer, & Coyle, 1992). Research conducted within the transdiagnostic dimensional framework may shed light on a variety of issues in aggression research, including identifying the biological mechanisms supporting aggressive behavior, revealing how healthy and pathologically aggressive individuals differ in their neural processing of threat, and addressing to what extent reactive aggression represents a loss of control (Tedeschi & Felson, 1994). On the other hand, the possibility of multiple and

overlapping subtypes of aggression poses a challenge for researchers pursuing these answers (Barratt, et al., 1999; Bushman & Anderson, 2001).

This study has several limitations worth mentioning. First, this study used a convenience sample of paid volunteers from the community. However, research participants were rigorously evaluated with respect to psychiatric disorders and most reported a history of engaging in or needing treatment for an emotional or behavioral problem. Still, the sample may differ in important ways from subjects seen in psychiatric treatment settings. Second, the lack of a two-factor result of our factor analysis could be due to confounding by measurement type. Although both assessment measures dichotomized aggression into hostile and premeditated forms, one asked subjects to describe the motivation for their aggressive acts while the other asked them to describe their aggressive motivations in general, which may have contributed to the observed factor solution. Finally, the lack of expected relationships between psychopathy and dimensions of aggressive behavior may be due to the restricted range of psychopathy in this sample. Further research in a sample recruited to study psychopathy might yield different results.

In spite of these limitations, the current study provides substantial evidence to support the heterogeneity of aggressive behavior in subjects with IED, healthy subjects, and psychiatric control subjects. The increasing application of advanced neuroscience methods and transdiagnostic dimensional research protocols (as in the NIMH RDoC initiative) have the potential to shed light on the neurobiological systems that support pathological aggressive behavior. Achieving this goal will be advanced by addressing the heterogeneity of aggressive behavior in research designs.

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References

American Psychiatric Association (2013). Diagnostic and Statistical Manual of Mental Disorders (5th ed.). Washington, DC: Author.

Anderson CA, & Bushman BJ (2002). Human aggression. Psychology, 53(1), 27.

Baker LA, Raine A, Liu J, & Jacobson KC (2008). Differential genetic and environmental influences on reactive and proactive aggression in children. Journal of Abnormal Child Psychology, 36(8), 1265–1278. [PubMed: 18615267]

Bandura A (1973). Aggression: A social learning analysis Oxford, England: Prentice Hall.

Barratt ES, Stanford MS, Dowdy L, Liebman MJ, & Kent TA (1999). Impulsive and premeditated aggression: A factor analysis of self- reported acts. Psychiatry Research, 86(2), 163–173. [PubMed: 10397418]

Beck AT, Steer RA, Brown G. Manual for the Beck Depression Inventory-II San Antonio, TX: Psychological Corporation; 1996.

Berkowitz L (1989). Frustration-aggression hypothesis: Examination and reformulation. Psychological Bulletin, 106(1), 59. [PubMed: 2667009]

Berkowitz L (1990). On the formation and regulation of anger and aggression: A cognitive-neoassociationistic analysis. American Psychologist, 45, 494–503. [PubMed: 2186678]

Beyer F, Münte TF, Erdmann C, & Krämer UM (2013). Emotional reactivity to threat modulates activity in mentalizing network during aggression. Social cognitive and Affective Neuroscience, 9(10), 1552–1560. [PubMed: 23986265]

- Bushman BJ, & Anderson C. a. (2001). Is it time to pull the plug on the hostile versus instrumental aggression dichotomy? Psychological Review, 108(1), 273–279. [PubMed: 11212630]
- Buss AH, & Perry M (1992). The aggression questionnaire. Journal of Personality and Social Psychology, 63(3), 452. [PubMed: 1403624]
- Campbell A, Muncer S, & Coyle E (1992). Social representation of aggression as an explanation of gender differences: A preliminary study. Aggressive Behavior, 18, 95–108.
- Cima M, Raine A, Meesters C, & Popma A (2013). Validation of the Dutch Reactive Proactive Questionnaire (RPQ): Differential correlates of reactive and proactive aggression from childhood to adulthood. Aggressive Behavior, 39(2), 99–113. [PubMed: 23386470]
- Coccaro EF, McCloskey MS, Fitzgerald DA, & Phan KL (2007). Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. Biological Psychiatry, 62(2), 168–78. [PubMed: 17210136]
- McCloskey MS, Phan KL, Angstadt M, Fettich KC, Keedy S, & Coccaro EF (2016). Amygdala hyperactivation to angry faces in intermittent explosive disorder. Journal of Psychiatric Research, 79, 34–41. [PubMed: 27145325]
- Coccaro EF, Berman ME, & Kavoussi RJ (1997). Assessment of life history of aggression: Development and psychometric characteristics. Psychiatry Research, 73(3), 147–157. [PubMed: 9481806]
- Coccaro EF, Lee R, & McCloskey MS (2014). Validity of the new A1 and A2 criteria for DSM-5 intermittent explosive disorder. Comprehensive Psychiatry, 55(2), 260–267. [PubMed: 24321204]
- Coccaro EF, Lee R, & Vezina P (2013). Cerebrospinal fluid glutamate concentration correlates with impulsive aggression in human subjects. Journal of Psychiatric Research, 47(9), 1247–1253. [PubMed: 23791397]
- Coccaro EF, Nayyer H, & McCloskey MS (2012). Personality disorder–not otherwise specified evidence of validity and consideration for DSM-5. Comprehensive Psychiatry, 53(7), 907–914. [PubMed: 22520088]
- Coccaro EF, & Schmidt-Kaplan CA (2012). Life history of Impulsive Behavior: Development and validation of a new questionnaire. Journal of Psychiatric Research, 46(3), 346–352. [PubMed: 22212770]
- Coccaro EF, Shima CK, & Lee R (In press). Comorbidity of personality disorder with intermittent explosive disorder. Journal of Psychiatric Research
- Cornell DG, Warren J, Hawk G, Stafford E, Oram G, & Pine D (1996). Psychopathy in instrumental and reactive violent offenders. Journal of Consulting and Clinical Psychology, 64(4), 783–790. [PubMed: 8803369]
- Cuthbert BN (2014). The RDoC framework: Facilitating transition from ICD/DSM to dimensional approaches that integrate neuroscience and psychopathology. World Psychiatry, 13(1), 28–35. [PubMed: 24497240]
- Dodge K (1991). The structure and function of reactive and proactive aggression. In Pepler DJ & Rubin KH (Eds.), The Development and Treatment of Childhood Aggression (pp. 201–218). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.
- Dodge KA, & Coie JD (1987). Social-information-processing factors in reactive and proactive aggression in children's peer groups. Journal of Personality and Social Psychology, 53(6), 1146–1158. [PubMed: 3694454]
- Dollard J, Miller NE, Doob LW, Mowrer OH, & Sears RR (1939). Definitions. In Dollard J (Ed.), Frustration and Aggression (pp 1–26). New Haven, CT: Yale University Press.
- Evans PJ, & Brown. (1961). Anger, hostility and aggression. In Buss A (Ed.) The Psychology of Aggression (pp 1–16). New York, NY: John Wiley and Sons.
- Fanning JR, Lee R, Gozal D, Coussons-Read M, & Coccaro EF (2015). Childhood trauma and parental style: Relationship with markers of inflammation, oxidative stress, and aggression in healthy and personality disordered subjects. Biological Psychology, 112, 56–65. [PubMed: 26423894]

Feshbach S (1964). The function of aggression and the regulation of aggressive drive. Psychological Review, 71(4), 257–272. [PubMed: 14183611]

- First M, Spitzer R, Gibbon M, & Williams J (1997). Stuructured Clinical Interview for DSM-IV Axis I Disorders (SCID-I), Clinical Version Washington DC and London: American Psychiatric Press, Inc.
- Fite PJ, Vitulano M, Wynn P, Wimsatt A, Gaertner A, & Rathert J (2010). Influence of perceived neighborhood safety on proactive and reactive aggression. Journal of Community Psychology, 38(8), 757–768.
- Fung AL-C, Raine A, & Gao Y (2009). Cross-cultural generalizability of the Reactive-Proactive Aggression Questionnaire (RPQ). Journal of Personality Assessment, 91(5), 473–479. [PubMed: 19672753]
- Geen R (2001). Human aggression Philadelphia, PA: Open University Press.
- Hart RD, Cox DN, & Hare RD (1995). The Psychopathy Checklist- Screening Version (PCL-SV) Toronto, ON, Canada: Multi-Health Systems.
- Hart SD, & Dempster RJ (1997). Impulsivity and psychopathy. In Webster CD & Jackson MA (Eds.), Impulsivity: Theory, Assessment, and Treatment (pp 212–232). New York, NY: Guilford Press.
- Kempes M, Matthys W, De Vries H, & Van Engeland H (2005). Reactive and proactive aggression in children: A review of theory, findings and the relevance for child and adolescent psychiatry. European Child and Adolescent Psychiatry, 14(1), 11–19. [PubMed: 15756511]
- Kessler RC, Coccaro EF, Fava M, Jaeger S, Jin R, & Walters E (2006). The prevalence and correlates of DSM-IV intermittent explosive disorder in the National Comorbidity Survey Replication. Archives of General Psychiatry, 63(6), 669–678. [PubMed: 16754840]
- Krämer UM, Jansma H, Tempelmann C, & Münte TF (2007). Tit-for-tat: The neural basis of reactive aggression. Neuroimage, 38(1), 203–211. [PubMed: 17765572]
- Miller JD, & Lynam DR (2006). Reactive and proactive aggression: Similarities and differences. Personality and Individual Differences, 41(8), 1469–1480.
- Ostrov JM & Houston RJ (2008). The utility of forms and functions of aggression in emerging adulthood: Association with personality disorder symptomatology. Journal of Youth and Adolescence, 37(9), 1147–1158.
- Pfohl B, Blum N, & Zimmerman M (1997). Structured Interview for DSM-IV Personality: SIDP-IV: American Psychiatric Pub.
- Polman H, Orobio De Castro B, Koops W, Van Boxtel HW, & Merk WW (2007). A meta-analysis of the distinction between reactive and proactive aggression in children and adolescents. Journal of Abnormal Child Psychology, 35, 522–535. [PubMed: 17340178]
- Porter S, & Woodworth M (2006). Psychopathy and aggression. In Patrick C (Ed.), Handbook of Psychopathy (pp 481–494). New York, NY: Guilfod Press.
- Poulin F, & Boivin M (2000). Reactive and proactive aggression: Evidence of a two-factor model. Psychological Assessment, 12(2), 115–122. [PubMed: 10887757]
- Raine A, Dodge K, Loeber R, Gatzke-Kopp L, Lynam D, Reynolds C, et al. (2006). The reactive-proactive aggression questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys. Aggressive Behaviour, 32, 159–171.
- Seara-Cardoso A & Viding E (2015). Functional neuroscience of psychopathic personality in adults. Journal of Personality, 83(6), 723–737. [PubMed: 25041571]
- Smith P, & Waterman M (2006). Self-reported aggression and impulsivity in forensic and non-forensic populations: The role of gender and experience. Journal of Family Violence, 21(7), 425–437.
- Stanford MS, Houston RJ, Villemarette-Pittman NR, & Greve KW (2003). Premeditated aggression: Clinical assessment and cognitive psychophysiology. Personality and Individual Differences, 34(5), 773–781.
- Steer RA, Beck AT. Manual for the Beck Anxiety Inventory San Antonio, TX: Psychological Corporation; 1997.
- Tedeschi JT, & Felson RB (1994). Violence, aggression, and coercive actions Washngton, DC: American Psychological Association.

Verona E, Patrick CJ, & Lang AR (2002). A direct assessment of the role of state and trait negative emotion in aggressive behavior. Journal of Abnormal Psychology, 111, 249–258. [PubMed: 12003447]

- Williamson S, Hare RD, & Wong S (1987). Violence: Criminal psychopaths and their victims. Canadian Journal of Behavioural Science, 19, 454–462.
- Yang Y, & Raine A (2009). Prefrontal structural and functional brain imaging analysis in antisocial, violent, and psychopathic individuals: A meta-analysis. Psychiatry Research: Neuroimaging, 174, 81–88.

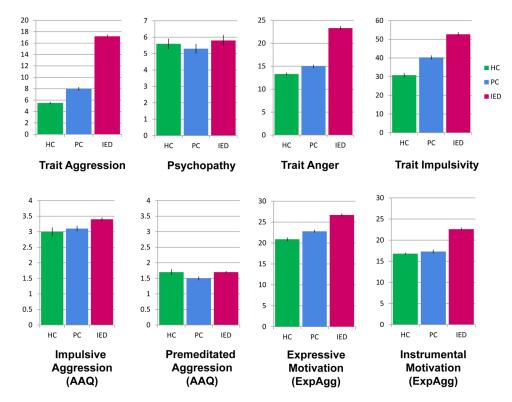


Figure 1.

Means and standard errors for healthy subjects (HC), psychiatric controls (PC), and intermittent explosive disorder (IED). Trait aggression=Life History of Aggression; Psychopathy=Psychopathy Checklist-Screening Version; Trait Anger=Buss Perry Anger Scale; Trait impulsivity=Life History of Impulsivity-20 item; AAQ=Aggressive Acts Questionnaire; ExpAgg= Expressive and Instrumental Representation of Aggression

Table 1

Characteristics of aggression according to the hostile-instrumental dichotomy

Hostile aggression	Instrumental aggression
Impulsive	Planned
Proximal motive: to inflict harm	Goal-directed (material benefit, status, etc)
Driven by anger	Not primarily driven by anger
Includes: reactive aggression, impulsive aggression, retaliatory aggression, affective aggression	Includes: proactive aggression, premeditated aggression

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Table 2
Syndromal and personality disorder diagnoses in the patient sample

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	PC (N = 311)	IED (N = 265)	p =
Current Syndromal Disorders:	,	(,	
Any Depressive Disorder	22 (7.1%)	51 (19.2%)	<.001*
Any Anxiety Disorder	53 (17.0%)	56 (21.1%)	.241
Any Substance Use Disorder	0 (0.0%)	0 (0.0%)	.999
Any Stress and Trauma Disorder	17 (5.5%)	36 (13.6%)	.001*
Any Eating Disorder	4 (1.3%)	14 (5.3%)	.007
Any Obsessive-Compulsive Disorder	4 (1.3%)	8 (3.0%)	.241
Any Somatoform Disorder	1 (0.3%)	4 (1.5%)	.186
Intermittent Explosive Disorder	0 (0.0%)	162 (61.1%)	< .001
Non-IED Impulse Control Disorder	1 (0.3%)	2 (0.8%)	.597
Lifetime Syndromal Disorders:			
Any Depressive Disorder	122 (39.2%)	155 (60.5%)	< .001 *
Any Anxiety Disorder	78 (25.1%)	77 (30.0%)	.219
Any Substance Use Disorder	116 (37.3%)	128 (50.0%)	.003
Any Stress and Trauma Disorder	55 (17.7%)	60 (22.6%)	.144
Any Eating Disorder	18 (5.8%)	25 (9.4%)	.112
Any Obsessive-Compulsive Disorder	8 (2.6%)	11 (4.2%)	.352
Any Somatoform Disorder	1 (0.3%)	5 (1.9%)	.099
Intermittent Explosive Disorder	0 (0.0%)	265 (100.0%)	< .001
Non-IED Impulse Control Disorder	2 (0.6%)	11 (4.2%)	.008
Personality Disorders:			
Any Personality Disorder	107 (34.3%)	211 (79.6%)	< .001 *
Cluster A (Odd)	5 (1.6%)	35 (13.2%)	<.001*
Cluster B (Dramatic)	20 (6.4%)	107 (40.4%)	<.001*
Cluster C (Anxious)	43 (13.8%)	58 (21.9%)	.012
PD-NOS	52 (16.7%)	76 (28.7%)	.001*

^{*} Note: p .0025 (Corrected for 20 unique comparisons).

Table 3

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Demographic characteristics of the sample

	HC (N = 284)	PC (N = 311)	IED (N = 265)	p =
Demographic Variables				
Age	34.4 ± 10.4	35.8 ± 9.7	37.8 ± 10.2	<.001 ^a
Gender (% Male)	49%	42%	48%	.321 ^b
Race (% W / AA / Other)	77 / 14 / 9	83 / 13 / 4	56 / 32 / 12	<.000°
SES Score	48.5 ± 10.9	46.4 ± 12.9	40.4 ± 13.7	<.001 ^d
Psychometric Variables				
LHA Trait Aggressive Behavior	5.5 ± 3.5	8.0 ± 4.9	17.2 ± 4.6	<.001 ^e
LHIB Trait Impulsive Behavior	30.8 ± 17.7	40.3 ± 17.5	52.7 ± 17.9	<.001 ^e
BPAQ Trait Anger	13.3 ± 4.8	15.0 ± 5.3	23.3 ± 7.0	<.001 ^e
PCL-SV Psychopathy	5.6 ± 5.1	5.3 ± 5.3	5.8 ± 5.5	.481

Notes.

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a: ANOVA: IED > PC = HC;

b: Chi-Square;

^C: Chi-Square: IED < PC = HC;

d: ANOVA: IED = PC < HC;

e: ANOVA: IED > PC > HC;

f: IED > PC = HC

Table 4

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AAQ and ExpAgg Scores in the Sample

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	HC (N = 284)	PC (N = 311)	IED (N = 265)	p =
AAQ Variable Scores				
Number of Aggressive Acts	0.2 ± 0.8	0.9 ± 6.0	10.8 ± 47.0	< .001f
Aggressive Acts Examined	1.8 ± 1.1	2.0 ± 1.2	2.9 ± 1.2	<.001 ^f
Raw IA-AAQ	3.0 ± 0.8	3.1 ± 0.8	3.4 ± 0.8	$=.005^{f}$
Raw PA-AAQ	1.7 ± 0.6	1.5 ± 0.5	1.7 ± 0.6	= .079
ExpAgg Variable Scores				
Raw ExpAgg-Expressive	20.9 ± 6.0	22.8 ± 5.5	26.7 ± 5.1	<.001e
Raw ExpAgg-Instrumental	16.8 ± 6.2	17.3 ± 5.5	22.6 ± 6.9	$< .001^{\rm f}$

Bivariate correlations between measures

Measure	1	2	$\frac{1}{2}$ $\frac{2}{3}$ $\frac{3}{4}$ $\frac{4}{5}$ $\frac{5}{6}$ $\frac{6}{2}$	4	5	9	
1. Premeditated (AAQ)	1						
2. Premeditated (AAQ)	18*	ŀ					
3. Expressive (ExpAgg)	.30**	.10	;				
4. Instrumental (ExpAgg)	08	.25**	.56**	1			
5. Trait Aggression (LHA)	.17*	.07	**0**	.46**	ŀ		
6. Trait Impulsivity (LHIB)	.22**	03	.36**	.33**	**84.	;	
7. Trait Anger (BPAQ)	.25**	.01	.38**	.41**	**65"	**74.	1
8. Psychopathy (PCL-SV)	01	.02	01 .0205 .02 .01	.00	.01	.01	.05

Table 5

Table 6

Multiple regression of aggression subtypes

Predictors	Impulsive Aggression (AAQ)	Premeditated Aggression (AAQ)	Expressive Aggression (ExpAgg)	Instrumental Aggression (ExpAgg)
Variance Explained (Model R ²)	0.09, p<0.001	0.005, p=0.860	0.198, p<0.001	0.193, p<0.001
Step 1				
Variance Explained (R ²)	0.021, p<0.076	0.003, p=0.689	0.059, p<0.001	0.065, p<0.001
Betas				
Depression (BDI-II)	06, p=0.419	.05, p=0.488	10, p<0.024	.05, p<0.230
Anxiety (BAI)	.07, p=0.352	.03, p=0.418	.10, p<0.024	.01, p<0.758
Step 2				
Variance Explained (\mathbb{R}^2)	0.069, p<0.001	0.002, p=0.754	0.139, p<0.001	0.128, p<0.001
Betas				
Trait Anger (BPAQ)	.25, p=0.001	.01, p=0.863	.28, p<0.001	.32, p=0.001
Trait Impulsivity (LHIB)	.09, p=0.217	06, p=0.454	.22, p<0.001	.15, p<0.001

a. n=243; b. n=585

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Table 7

Factor analysis of aggression items (N=729)

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	:	Fac	tors	
<u>Items</u>	1	2	3	4
Get through to people(ExpAgg1)	.726	.022	015	.020
Asking for it (ExpAgg3)	.706	.042	082	.030
Made me look bad (ExpAgg5)	.722	.246	.028	.088
Rather hit than cry (ExpAgg7)	.703	.082	.019	.021
People fall in line (ExpAgg9)	.790	012	045	.076
Won't back down (ExpAgg11)	.681	.053	028	008
Prevent future trouble (ExpAgg13)	.677	.271	041	010
Shown up publicly (ExpAgg15)	.678	.106	014	.075
Pushed when under stress (ExpAgg4)	.550	.455	.092	.041
Want acknowledgement (ExpAgg8)	.532	.419	049	025
Aggressive when alone (ExpAgg12)	.506	.434	.027	.018
Feel out of control (ExpAgg2)	.274	.574	.093	.087
Feel drained and guilty (ExpAgg6)	.010	.753	.116	038
Losing self-control (ExpAgg10)	.253	.645	.126	103
Upset and shaky (ExpAgg14)	.253	.701	.059	040
Verbal aggression (ExpAgg16)	050	.494	022	.067
I lacked self-control (AAQ9)	.004	.054	.691	200
I felt guilty after (AAQ16)	161	.175	.720	070
I was confused (AAQ17)	022	.113	.565	.277
Act was impulsive (AAQ18)	.023	.030	.760	142
Act was disproportionate (AAQ19)	.032	011	.755	060
Act was planned (AAQ1)	.045	.048	328	.486
Profited financially (AAQ13)	.039	.000	018	.832
Power and social status (AAQ14)	.098	032	052	.753