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Latent structure of intermittent explosive disorder in an epidemiological sample

Anthony O. Ahmed [a,*], Bradley A. Green [a], Michael S. McCloskey [b], Mitchell E. Berman [a]

[a] Department of Psychology, University of Southern Mississippi, 118 College Drive, #5025 Hattiesburg, MS 39406, United States
[b] Department of Psychology, Temple University, 1701 N., 13th Street, Philadelphia, PA 19122, United States

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ABSTRACT

Intermittent explosive disorder (IED) is characterized by distinct periods of impulsive aggression marked by assaultive acts or destruction of property. However, impulsive aggression is also a feature of other disorders, all of which are viewed in diagnostic nomenclature as qualitatively distinct from IED. This state of affairs is problematic for categorical models unless it is demonstrated empirically that IED-related impulsive aggression is qualitatively distinct from impulsive aggression observable in other axis I and II disorders. The current study addresses this question using taxometric methods to examine the latent structure of IED. Participants were respondents on the Collaborative Psychiatric Epidemiological Surveys, which obtained data on a range of disorders including intermittent explosive disorder ($N = 20{,}013$) and a range of psychological variables. Indicator variables used were drawn from the survey items and submitted to select taxometric methods (MAMBAC and MAXEIG) to determine the relative fits of a taxonic versus dimensional model. The results of taxometric analyses provided support for a taxonic, rather than dimensional, structure for IED symptoms in the epidemiological sample. Taxon group membership was associated with treatment seeking, family history of anger attacks, lower age of onset of anger attacks, and male biological sex, providing strong support for the validity of the IED taxon.

1. Introduction

With the publication of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) imminent, the question of whether specific mental disorders are better conceptualized as classes rather than an extremes on continua remains a key issue (for discussions see Eysenck, 1986; Widiger and Trull, 2007). This question is of particular relevance to the study of aggression, a feature of multiple diagnoses in the current DSM. Intermittent explosive disorder (IED) is the diagnosis used to classify individuals who engage in repeated acts of impulsive aggression that are disproportionate to any provocation, and not better accounted for by the effects of a substance, medical condition, or other psychological disorder (American Psychiatric Association, 2000). In short, IED is a disorder of impulsive aggression; the only such disorder in the DSM-IV. However, impulsive aggression is also a key feature of antisocial behavior in children, adolescents, and adults and may serve as a reliable indicator of oppositional defiant disorder (ODD), conduct disorder (CD), and antisocial personality disorder (ASPD). Impulsive other-directed aggression has also been associated with borderline personality disorder (BPD) (Critchfield et al., 2008), bipolar disorder (Garno et al., 2008), substance use disorders (Eronen et al., 1996; Swanson et al., 1991) paranoid, and passive aggressive features in axis II disorders (Berman et al., 1998). Given that impulsive aggression is a feature of many disorders it may be of interest to determine whether aggressive symptoms in IED are qualitatively distinct from those manifested in other psychopathology and non-pathology.

The latent structure of aggressive symptoms in IED is central to the appropriateness of a categorical diagnostic model, such as that presented in the DSM manual. Detractors of categorical models argue that diagnostic boundaries are often arbitrary and incorrectly situated (Widiger and Clark, 2000), whereas others argue that categorical models should endeavor to reflect the true discontinuities in the latent structure of disorders to be considered practical (Hinshaw et al., 1993). Evidence of a taxonic structure for IED symptoms may lend some support to the current DSM categorical framework for symptoms of aggression in IED. Although evidence of a taxonic structure may not reconcile whether IED is correctly grouped as an "impulse-control disorder not elsewhere classified," it could serve as a springboard to further clarification of its boundary with other impulse-control disorders, and other clinical syndromes associated with aggression, including mood, anxiety, substance use, and personality disorders (Coccaro et al., 2005, 2004; Kessler et al., 2006). If empirical research confirms that a categorical model is appropriate, this may also serve as an impetus for further delineation of a taxonic structure, such as investigating the performance of the current IED research criteria at correctly identifying members of an IED taxon, searching for the existence of

* Corresponding author. Tel.: +1 240 506 3809.
E-mail address: anthony.o.ahmed@gmail.com (A.O. Ahmed).

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possible IED subtypes, and illuminating the nature of its comorbidity with other clinical syndromes.

In addition, deciphering the true structure of the underlying distribution of aggressive behavior has both clinical and methodological ramifications. The goal of assessment in clinical practice is often to determine whether an individual meets diagnostic criteria for IED. Treatment decisions are then made depending on whether diagnostic criteria are met. However, categorical decision making may be misguided if the underlying distribution of IED is dimensional rather than discontinuous; as it may result in the neglect of subthreshold presentations that may otherwise be experiencing clinically significant impairment in functioning. The practice of dichotomizing scores measured on continuous scales (e.g., the Buss–Perry Aggression Questionnaire) or making diagnostic (yes/no) decisions based on such scales is also only appropriate if the underlying distribution of aggressive behavior is categorical and cutting scores adequately approximate the point separating IED taxon and non-taxon members. In clinical research, classifying cases to IED versus non-IED categories based on a median split or any cutting score when IED symptoms are dimensional will likely reduce the variance of the otherwise continuous measure and attenuate the size or magnitude of the relationship among the variables being investigated. Dichotomizing a dimension can lead to loss of statistical power and inflate type II error rate even when sample sizes and effect sizes are large, and have even more profound results when *N* is relatively small and effect sizes are minimal to moderate. However, if IED symptoms are categorically distributed, then cutting scores that approximate the boundaries between IED and non-IED groups would perform adequately at sustaining statistical power. The structure of IED symptoms may also be relevant to sampling strategies for clinical research. If IED symptoms are taxonic, then aggression studies should use sampling strategies that increase the likelihood of obtaining taxon group members, sampling individuals who likely meet diagnostic criteria (i.e., clinical samples), as it may be inappropriate to generalize the results of investigations conducted on student samples to clinical populations (for detailed discussions, see (Meehl, 1992; Ruscio et al., 2006).

Taxometric analyses are used to determine the latent structure of constructs; that is, whether constructs are best viewed as taxonic or dimensional, and whether psychiatric diagnoses are better conceived as distinct categories (qualitatively different from non-pathological functioning), or if a disorder is a pathological end of a continuum of normal behavior (i.e., dimensional). Taxometric analyses have suggested some psychiatric disorders, such as post-traumatic stress disorder (Forbes et al., 2005), social phobia (Kollman et al., 2006), generalized anxiety disorder (AM Ruscio et al., 2001), antisocial personality disorder (Marcus et al., 2008), and borderline personality disorder (Rothschild et al., 2003) are dimensional, but other disorders such as schizotypy appear to be categorical (Fossati et al., 2007; Haslam, 2003). A finding that IED is taxonic would suggest that individuals with IED represent an unique class that is qualitatively different from individuals with non-pathological (i.e., non-IED) levels of aggression. Such a finding would also provide evidence that IED is distinct from ASPD, a disorder associated with impulsive violence, which has been shown to be dimensional in nature (Marcus et al., 2008), and other disorders that have been shown to be associated with impulsive aggression. Further, the outcomes of a taxometric study may help evaluate the validity of current DSM criteria, serve as a catalyst to understanding the most distinctive features of an IED putative taxon, and confirm the prevalence rate of IED. Though initially thought to be rare (American Psychiatric Association, 1994), recent studies have shown IED to be an under-diagnosed disorder existing in over 6% of the population (Coccaro et al., 2005; Kessler et al., 2006).

The present study examined the latent structure of IED using a large epidemiological sample and taxometric methods. Conducting taxometric analysis with a large epidemiological sample confers a number of technical advantages. First, epidemiological samples, due to their large sample sizes, provide clearer resolution of taxometric plots (Meehl and Yonce, 1994), allowing statistics computed in sliding slabs (i.e., consecutive subsamples) to be computed with adequate cases per subsample. Second, a nationally representative epidemiological sample allows some confidence that members of the conjectured taxon group are represented in the sample, minimizing the possibility of obtaining false dimensional results due to under-representation of taxon members. Finally, the base rate of the taxon group, if found, can be compared to the prevalence rate of IED in the epidemiological sample, which may be informative in terms of confirming prevalence rates, refining IED diagnostic criteria, and determining optimal thresholds for classifying individuals into IED positive and negative categories (Ruscio et al., 2006).

2. Method

2.1. Participants

The dataset used was the 2001–2003 Collaborative Psychiatric Epidemiological Surveys (CPES; Inter-university Consortium for Political and Social Research [ICPSR], 2003). The CPES dataset ($N = 20{,}013$) includes data from three nationally representative surveys of people residing in the United States who are 18 years or older and who are not institutionalized. The surveys are the National Comorbidity Survey Replication (NCS-R), National Survey of American Life (NSAL), and the National Latino and Asian American Study (NLAAS). The CPES dataset was created by combining the three surveys using a sample weighting method described by Kessler et al. (2004) and using race/ancestry grouping that breaks down the sample into 12 ethnic groups. Because the three surveys specifically sampled groups of individuals with the NSAL and the NLAAS over-sampling specific ethnic groups, the CPES sampling procedure was able to use apportioned weights to define each individual's ethnic category as well as each individual's geographical location. Using this procedure to estimate the population sample characteristics, the final CPES sample comprised Vietnamese ($n = 527$), Filipino ($n = 525$), Chinese ($n = 619$), Other Asian ($n = 613$), Cuban ($n = 625$), Puerto Rican ($n = 654$), Mexican ($n = 1442$), Other Hispanic ($n = 899$), Afro-Caribbean ($n = 1492$), African American ($n = 4746$), and White and Other ($n = 7871$) participants.

2.2. Measures

The CPES surveys were derived from the World Mental Health Composite International Diagnostic Interview (WMH-CIDI; Kessler and Üstün, 2004). The WMH-CIDI was itself derived partly from the Diagnostic Interview Schedule (Robins et al., 1995) and partly from an earlier version of the CIDI. All three surveys collected data on several constructs, such as criteria for affective disorders, anxiety disorders, personality pathology, substance-related disorders, childhood disorders and other psychiatric conditions. The IED-related portion of the survey comprised 73 questions assessing: frequency of explosive episodes, types of destructive and assaultive behaviors, treatment seeking, family history of anger attacks, IED diagnosis disqualifiers, perceptions of controllability and proportionality of expressed aggression, and functional impairment. The data were collected by the Survey Research Center (SRC) of the Institute for Social Research at the University of Michigan using computer assisted personal interviewing. Data collection began in early 2002 and was completed by December of 2003.

2.3. Data analysis

To determine the latent structure of IED symptoms, we used two of the taxometric procedures developed by Meehl and his colleagues to analyze responses on the CPES survey – Mean Above Minus Mean Below A Cut (MAMBAC; Meehl and Yonce, 1994) and Maximum Eigenvalue (MAXEIG; Waller and Meehl, 1998).[1] The MAMBAC procedure requires at least two valid indicators of the conjectured taxon with one of the indicators designated as the input indicator and another designated as an output indicator. At least one of the indicators used for the analysis must be a continuous variable. In MAMBAC, if all indicators are continuous, then all indicators used for the analysis are paired in every possible input/output combination. After an input indicator is designated, the cases are sorted in ascending order using the input indicator; successive cuts are made on the input indicator, with the cuts sliding across the distribution of scores on this indicator, from lowest to highest. At each successive cut on the input indicator, the mean of scores of the output indicator, falling below the cut on the input indicator, are subtracted from the output scores falling above the cut. A graph of the mean difference at each cut point is plotted. According to Meehl and Yonce (1994) taxonic situations will yield a graph that is characteristically "concave down," whereas dimensional situations will yield curves that are "concave up." Considering the size of the sample as well as the base rate of the conjectured taxon (the prevalence rate of IED diagnosis in the sample is 7.00%, whereas the point prevalence was estimated at 1.62%), 2000 evenly-spaced cuts were made along the input indicator to ensure that the analyses were fine enough to detect such a low base rate construct. We configured the taxometric program to exclude 20 cases from each end of the distribution of the input indicator to minimize the effect of extreme responses on the MAMBAC plots. The program was also configured to resort the input indicator data ten times for each analysis, recalculate the mean differences, and compute the average of the results as the final result. This is implemented to control for random sorting effects of ties scores that may arbitrarily fall on different sides of MAMBAC cuts.

The MAXEIG procedure uses three or more valid indicators of the construct of interest that are relatively independent within the taxon and complement groups (i.e., indicators possessing relatively low covariance within groups, or low nuisance covariance). MAXEIG proceeds by assigning one of the indicator variables as an input indicator and the remaining indicators as the output indicators. As with MAMBAC, the cases are sorted on the input variable from lowest to highest. Overlapping windows (portions of the distribution) are created on the input indicator, and at each window MAXEIG assesses the first eigenvalue among the output indicators, moving successively across the scores on the input variable from beginning to end of the distribution. A graph of the first eigenvalues is plotted with the *y*-axis representing the eigenvalues for the output indicators and the input variable represented on the *x*-axis. Each possible permutation of indicators is examined with each indicator serving as an input variable in the MAXEIG analysis while the other indicators serve as outputs, yielding one MAXEIG graph for each indicator. According to Waller and Meehl (1998), taxonic situations will yield a graph with a characteristic "hump" near the hitmax point (the point on the input indicator that maximally differentiates between taxon and complement group members); however, dimensional situations will yield a graph that is relatively flat, varying around the mean eigenvalue. Again, considering the size of the sample and the base rate of the conjectured taxon, the taxometric program was configured to create 2000 overlapping windows with successive windows sharing about 90% of the cases within their intervals. With this number of windows, the taxometric plot is parsed finely enough to detect a low base rate taxon. With 2000 overlapping windows, each window contained 100 cases. We also configured the program to randomly resort the input indicator data ten times for each analysis, recalculate the eigenvalues, and compute the average of the results as the final result. This procedure reduces the effects of random ordering of the cases when multiple cases possess the same value on the input indicator.

The data were analyzed using Ruscio's taxometric programs (Ruscio et al., 2006) running on the R 2.6.1 statistical program (R Foundation for Statistical Computing, 2007). Ruscio's program produces graphs for each input–output indicator configuration, and also creates an average curve representing the average of the individual curves. Interpretation of the graphs is aided by comparison to taxonic and dimensional graphs obtained from simulated data possessing the idiosyncratic distribution features of the research data including sample size, means, standard deviations, skew, and kurtosis. We configured the program to generate ten samples of taxonic and dimensional data for each analysis (Ruscio et al., 2006). This method allows us to compare the average graphical output obtained from the research data with the output obtained from the simulated taxonic and dimensional comparison data. For the generation of taxonic comparison data, we used the taxometric program defaults, which assigns cases to groups based on the mean taxon base rate from each analysis. Ruscio's program also computes a comparison curve fit index (CCFI) as a numerical aid to determine whether the research data more closely fit the simulated taxonic or dimensional data. CCFI values range from 0 to 1, with values closer to 0 suggesting dimensional results, values closer to 1 lending support to a taxonic structure, and values around .50 suggesting ambiguous results. Ruscio (2007) found that when MAMBAC and MAXEIG analyses produced CCFI values that were either both less than .50 or both greater than .50, taxonic and dimensional data sets were correctly identified with 99% accuracy.

2.4. Construction of candidate indicators

The 73 IED-related items in the dataset assessed DSM-IV diagnostic criteria for IED including criterion C, the disqualifier criterion (anger not explained by some other disorder or circumstances), various forms of disturbance in the domains of interpersonal and occupational functioning, specific assaultive and destructive acts carried out during explosive episodes, and a number of items assessing onset of anger episodes, treatment seeking, and family history of anger attacks. Only a subset of the items were judged appropriate for the investigation given that some items were conceptually redundant, and others were better conceived as external validity variables rather than symptoms of IED. We selected 28 IED-related items, which we summed into five scales labeled anger attack, out of proportion/control, aggression consequences, aggression forms, and aggression duration. The items were combined into composite indicators rather than individual items because composite indicators appear to be more reliable and tend to produce more stable taxometric results (Beauchaine, 2003; Cole, 2004). Moreover, item-level taxometric

[1] We also ran two additional taxometric procedures – MAXCOV (MAXimumCOVariance; Meehl and Yonce, 1996) and L-Mode (Latent Mode Factor Analysis; Waller and Meehl, 1998), the results of which are not reported here due to space but available upon request. The results of both procedures were consistent with those of MAMBAC and MAXEIG. The MAXEIG procedure is conceptually and mathematically similar to the more widely used MAXCOV and is a multivariate extension of MAXCOV. The main difference between the two procedures is that MAXEIG plots conditional eigenvalues whereas MAXCOV plots conditional covariances on the y-axis. In Ruscio's program, both procedures can be implemented by varying program parameters (e.g., overlapping or non-overlapping windows, indicator triplets versus composite indicators etc.). The L-Mode procedure distinguishes between taxonic and dimensional constructs by plotting the factor score probability density function of the indicators and examining the distribution for evidence of unimodality (dimensional) or bimodality (taxonic).

Table 1
DSM-IV Criteria for Intermittent Explosive Disorder (American Psychiatric Association, 1994, 2000, p. 667).

A. Several discrete episodes of failure to resist aggressive impulses that result in serious assaultive acts or destruction of property
B. The degree of aggressiveness expressed during the episode is grossly out of proportion to any precipitating psychosocial stressors
C. The aggressive episodes are not better accounted for by another mental disorder (e.g., Antisocial Personality Disorder, Borderline Personality Disorder, Psychotic Disorder, a Manic Episode, Conduct Disorder, or Attention Deficit/Hyperactivity Disorder) and are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., head trauma or Alzheimer's disease)

analyses of 20,013 cases would exceed the computing power of most computers. We created the composite indicators using a rational approach, with the goal of matching candidate items with DSM or IED research criteria, which are reproduced in Tables 1 and 2. Table 3 summarizes all of the items used, provides descriptive statistics for each item used, and indicates the scale subsuming the item.

The aggression frequency scale was composed of six items assessing the number of times in life respondents engaged in particular aggressive behavior, indicating discrete episodes of impulsive anger. It should be noted that the items presented under the subscale appear redundant because the descriptives presented correspond to each individual survey included in the CPES (i.e., the NCS-R, the NSAL, and the NLAAS). The items included in this scale elicited numerical responses from respondents that ranged from 0 to 9997, clearly extreme at the high end of the distribution and highly skewed (skew = 17.31). In addition, numerical responses tended to cluster around certain values (e.g., 5, 10, 20, 100, 500, 1000, etc.). To correct for these potential problems in the variables, we recoded responses on the items into a 7-point Likert scale with 0 = 0, 1–5 = 1, 6–10 = 2, 11–20 = 3, 21–50 = 4, 51–100 = 5, and 101 to 9997 = 6. Recoding the items this way reduced the size of the skew (skew = 3.32), while still minimally violating the stochastic ordering of the raw scores. Conceptually, the Aggression Frequency scale corresponded to Criterion A of the DSM, by assessing discrete episodes of aggressive impulses. The internal consistency alpha of data from this scale was minimal (alpha = −.19), no doubt due to the fact that only pairs of items belonged to the same survey. For example, as indicated in Table 3, the first and the fourth items on the scale were drawn from the NCS-R, whereas the second and the fifth were drawn from the NSAL survey.

The out of proportion/control scale was composed of five items assessing whether respondents viewed their anger attacks as uncontrollable and out of proportion to the cause of anger. The first three items in this scale conceptually match Criterion B of both DSM and IED research criteria, given that they directly query the proportionality of anger to the stressor. The two additional items were included because of the conceptual relatedness of out of control to proportionality. Data from this scale possessed weak internal consistency (alpha = .59).

The aggression consequences scale included seven items assessing the impact of anger episodes on domestic, interpersonal, occupational, and emotional functioning. Although this scale has no conceptual match in current DSM criteria, it conceptually matches Criterion D of the IED research criteria by directly assessing difficulties in emotional and social functioning. The Aggression Consequences scale data demonstrated excellent internal consistency (alpha = .91).

The aggression forms scale combined nine items assessing types of aggressive behaviors that respondents demonstrated including breaking things, destroying property, burning things, injuring or torturing animals, threatening others, and physically injuring others. The items in this scale conceptually matched Criterion A of the IED research criteria, assessing both physical and verbal aggression, as well as less severe forms of impulsive aggression (e.g., throwing clothes etc.). This scale possessed weak internal consistency (alpha = .60).

The aggression duration scale was a single item assessing the number of years respondents have experienced anger attacks. The aggression duration variable was conceptually distinct from the other indicators and varied sufficiently in response to serve as a one item scale drawn from the response to "number of different years in lifetime having at least one anger attack." Although this item does not directly match formal diagnostic criteria, we included it as a candidate indicator because it potentially quantifies the DSM requirement that there be "several discrete episodes," indirectly indicating the frequency of anger attacks.

2.5. Designation of external variables for construct validation of the conjectured taxon

We also created a scale partly matching Criterion C of the IED DSM criteria and Criterion E of IED research criteria (see Tables 1 and 2), which we labeled a disqualifier scale. We did not include this scale as a candidate indicator for the taxometric analysis because it only serves to rule out IED and is not a direct indicator of IED. We reserved the items of this scale for a post hoc analysis of the characteristics of our conjectured taxon. The disqualifier scale included items assessing whether anger symptoms occurred due to substance use, physical illness, or depression (e.g., anger attack was due to substance use). Some of the items were reversed scored (i.e., anger attack was not due to…), so the scoring was changed to make a positive value (endorsement) indicate denial of the disqualifier for all items on that scale. The disqualifier scale possessed adequate internal consistency (alpha = .77).

Prior to analyses we also designated four items assessing age of onset, treatment seeking, family history of aggression, and biological sex as external variables related to construct validity. We included biological sex and a family history variable in the analysis as external variables because of the literature suggesting differential prevalence of IED symptoms in men versus women (e.g., Coccaro et al., 1998, 2005; Kessler et al., 2006), and the heritability

Table 2
Research criteria for intermittent explosive disorder (Coccaro et al., 1998).

A. Recurrent incidents of aggression manifest as verbal or physical aggression towards other people, animals, or property occurring twice weekly on average for one month
B. The degree of aggressiveness expressed is grossly out of proportion to the provocation or any precipitating psychosocial stressors
C. The aggressive behavior is generally not premeditated and is not committed in order to achieve some tangible objective such as money, power, intimidation or other
D. The aggressive behavior causes either marked distress in the individual or impairment in occupational or interpersonal functioning
E. The aggressive behavior is not better accounted for by another mental disorder, general medical condition or direct physiological effects of a substance

Table 3
Items used and descriptive statistics.

Survey item	*M*	*SD*	Skew	Kurtosis
Aggression frequency				
# attacks in life lost control/broke things/hurt or threaten	0.14	0.64	5.93	40.13
# attacks in life lost control/broke things/hurt or threaten	0.04	0.32	10.39	133.29
# attack in life lost control/broke or smash something	0.06	0.34	8.24	94.99
# attack in life lost control/hurt or threaten to hurt someone	0.08	0.44	6.33	51.48
# attacks lost control/hurt or threaten to hurt someone	0.09	0.42	7.30	72.46
# attack lost control/threatened to hurt someone	0.06	0.33	8.71	106.04
Out of proportion/control				
Anger attacks occurred without good reason	0.05	0.21	4.33	16.76
Anger occurs in situations most people wouldn't have attack	0.02	0.14	6.86	45.06
Much more angry than most during attack	0.03	0.16	5.76	31.16
Unavoidable impulse to blow-up before attack	0.05	0.22	4.01	14.08
Anger out of control during typical attack	0.26	0.88	3.32	9.62
Aggression consequences				
Extent anger attacks interfere w/work/social life/relations	0.11	0.47	4.95	26.48
Unable to carry out daily activities due to anger	0.11	0.49	5.09	27.85
Guilty/embarrassed/regret after anger attack	0.21	0.78	3.84	13.79
Extent severe attacks interfere home management	0.11	0.81	8.53	79.05
Extent severe attacks interfere with ability to work	0.10	0.79	9.56	98.65
Extent severe attacks interfere with forming/maintaining relations	0.14	0.92	7.72	64.14
Extent severe attacks interfere social life	0.13	0.90	8.07	69.64
Aggression forms				
# anger attacks hurt someone that needed med attention	0.07	1.50	51.66	3173.20
During attack in week-slam door/kick chair/throw clothes	0.14	1.35	20.52	593.62
During attack in week-break something	0.05	0.66	40.63	2324.46
During attack in week-broke several things	0.03	0.40	32.17	1553.96
During attack in week-set fire or destroyed property	0.01	0.17	34.80	1444.76
During attack in week-injured/tortured animal	0.00	0.10	44.43	2069.37
During attack in week-threatened someone	0.05	0.77	44.63	2853.14
Attack in week-hurt someone that needed med attention	0.00	0.13	45.58	2684.50
Attack in week-hurt someone didn't need medical attention	0.01	0.21	21.04	569.17
Aggression duration				
# different years in lifetime having at least 1 anger attack	0.72	3.60	7.89	79.28
Disqualifiers				
Anger attacks usually occur because of drinking/drug use	0.07	0.26	3.24	8.51
Anger attacks ever occur when not drinking/using drugs	0.01	0.10	9.31	84.78
Anger attacks ever result of physical illness or meds	0.08	0.27	3.13	7.79
Anger attacks ever not result of physical illness or meds	0.00	0.06	16.70	276.95
Anger attack occur when sad/depressed	0.05	0.21	4.37	17.07
Anger attacks ever occur when not sad/depressed	0.02	0.14	6.77	43.84
Other external variables				
Talked to health care professional about anger attacks	0.02	0.14	6.73	43.26
Approx age 1st attack	15.49	8.58	1.18	1.21
Close relatives had repeated anger attacks	0.05	0.22	4.05	14.41
Biological sex				

Notes: $N = 20{,}013$.

of IED (Coccaro, 2003; McElroy et al., 1998). If our analyses reveal an IED taxon, taxon group membership should also be expected to be associated with the designated external variables. If the analysis reveals a latent dimension, then the severity of IED symptoms may be expected to be associated with our designated external variables.

Taxometric analyses require that the indicators used for the analyses possess adequate validities for distinguishing between taxon and complement group members. Thus, evaluating prospective indicator variables for adequate validity is a standard practice in taxometric analyses. Meehl (1992) recommended that indicators used for analyses separate taxon and complement group members by at least 1.25 standard deviations units. We submitted all five candidate indicators to an initial MAMBAC run to evaluate their respective validities. All five indicators demonstrated adequate validities. Thus, we carried out our final MAXEIG and MAMBAC runs with the five indicators to determine the latent structure of IED in the sample. Table 4 provides descriptive statistics for the five scales used in the taxometric analyses.

Table 4
Descriptive statistics of indicator variables.

Indicator variable	*M*	*SD*	Skew	Kurtosis
Aggression frequency	0.40	0.89	3.32	13.63
Out of proportion/control	0.41	1.32	3.09	7.96
Aggression consequences	0.91	4.26	6.65	51.57
Aggression forms	0.36	3.13	18.63	503.89
Aggression duration	0.72	3.60	7.89	79.28

Note: $N = 20{,}013$.

3. Results

3.1. MAXEIG analyses

In MAXEIG analysis, the indicator validities were strong and the nuisance covariance was acceptable. Nuisance covariance is defined as the correlation between indicators within the taxon and complement groups. The indicator validities for the five indicators

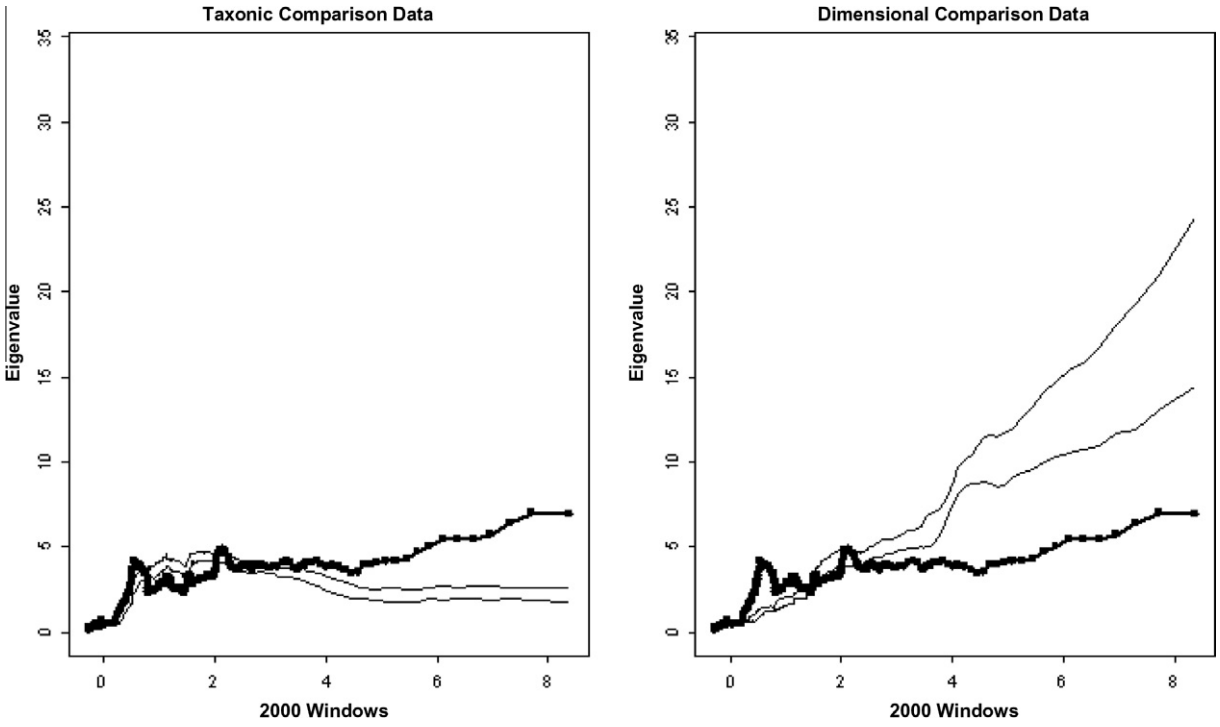

Fig. 1. Averaged MAXEIG curve for the research data, superimposed on the taxonic comparison simulations in the left panel and dimensional comparison simulations in the right panel, for the five indicators of IED drawn from the CPES database. The dark line represents the actual data and the lighter lines represent one standard deviation above and below the average for each type of comparison data.

ranged from 2.31 to 5.09 (M = 3.90, SD = 1.10). The average nuisance covariance in the taxon group was low (r = .06, SD = .20) and in the complement group was acceptable (r = .35, SD = .28). For computing indicator validities and nuisance covariances, MAXEIG assigned 1122 cases as taxon members and 18,891 cases as complement members. The average correlation among indicators in the full sample was estimated as r = .51.

Fig. 1 depicts the average MAXEIG curve superimposed over the simulated taxonic and simulated dimensional plots. Visual inspection of the average curve indicates a function that appears to be peaking at the high end of the distribution, quite consistent with a low base rate taxon with positively skewed indicators (Ruscio and Ruscio, 2004; Ruscio et al., 2004, 2006; Waller and Meehl, 1998). Examining the average curve as well as the outputs of the simulated comparison data, there is clear indication that the average curve matches the simulated taxonic data and is clearly inconsistent with the simulated dimensional data. The MAXEIG analysis yielded a CCFI estimate of .70, providing further support for a taxonic latent structure. The base rate estimates for each indicator ranged from .013 to .071 (M = .038, SD = .029), and the averaged MAXEIG curve produced a base rate estimate of .020. Table 5 summarizes base rate estimates, validity estimates, and nuisance covariance for each indicator in MAXEIG and MAMBAC analyses.

Table 5
Base rate and validity estimates.

	Base rate	Validity
MAXEIG		
Aggression frequency	0.01	3.29
Out of proportion/control	0.07	5.09
Aggression consequences	0.01	4.59
Aggression forms	0.07	2.31
Aggression duration	0.03	4.24
M	0.04	3.90
SD	0.03	1.11
NC_{tax}	.13 (.21)	
$NC_{comp.}$	.14 (.22)	
MAMBAC		
Aggression frequency	0.10	3.27
Out of proportion/control	0.12	10.51
Aggression consequences	0.07	3.10
Aggression forms	0.05	1.34
Aggression duration	0.12	2.78
M	0.09	4.20
SD	0.03	3.61
NC_{tax}	.06 (.20)	
$NC_{comp.}$	.35 (.28)	

Note: NC = nuisance covariance.

3.2. MAMBAC analyses

In MAMBAC analysis, the indicator validities were again strong. The MAMBAC analysis yielded indicator validities ranging from 1.34 to 10.51 (M = 4.20, SD = 3.61). The average nuisance covariance of indicators within the taxon (r = .13, SD = .21) and complement (r = .14, SD = .22) groups were acceptable. To compute indicator validities and nuisance covariance, MAMBAC assigned 1848 cases as taxon members and 18,165 cases as complement members.

Fig. 2 depicts the average MAMBAC curve superimposed over the outputs for the simulated comparison taxonic and dimensional data. Again visual inspection of the average curve suggests that it is consistent with a low base rate taxon, demonstrating the J-Shaped distribution that may be expected in situations of low base rate and high positive skew (Meehl and Yonce, 1994; Ruscio and Ruscio, 2004; Ruscio et al., 2004, 2006). It is also clear that the average curve obtained from the research data more closely fits the simulated taxonic output and is also clearly incongruent with the simulated dimensional output. So high is the fit between the research

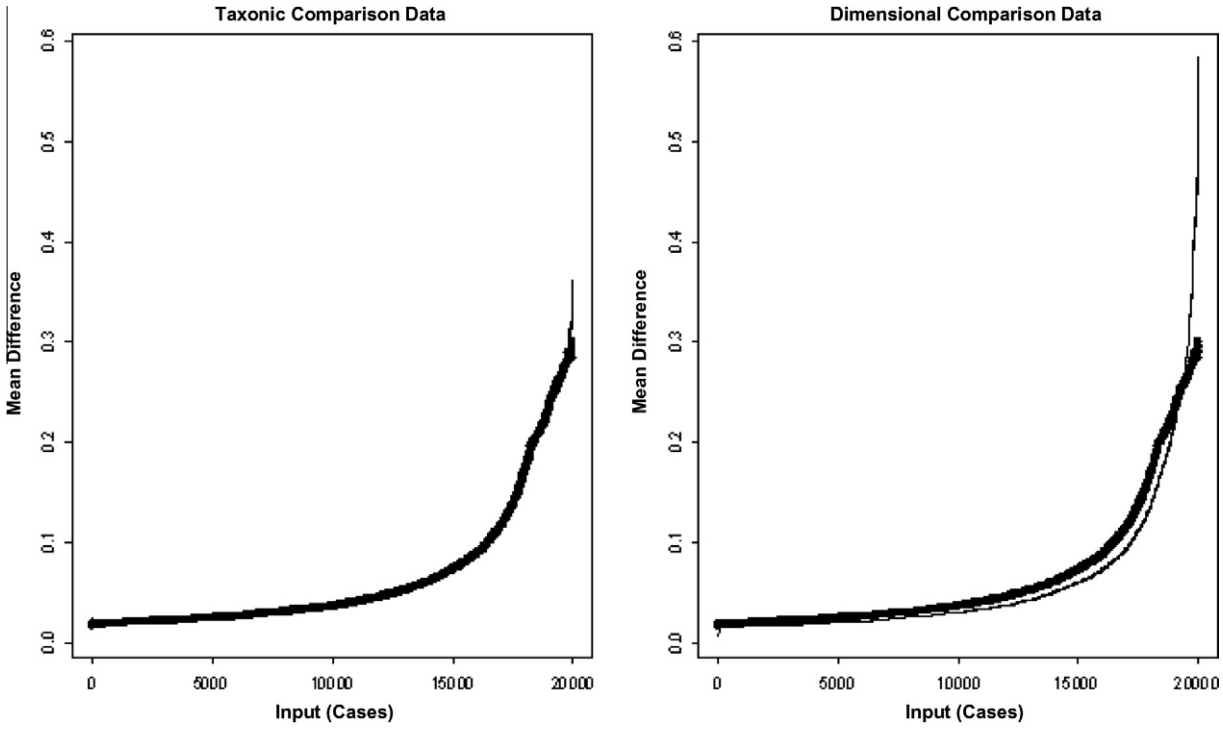

Fig. 2. Averaged MAMBAC curve for the research data along with taxonic and dimensional comparison data. The dark line represents the actual data and the lighter lines represent one standard deviation above and below the average for each type of comparison data.

data and the simulated taxonic data that when the graphical output of the research data was overlaid on that of the simulated taxonic data, there was only a small region of non-overlap. MAMBAC analysis yielded a CCFI estimate of .84, providing strong support for a taxonic latent structure. MAMBAC produced base rate estimates ranging from .019 to .241 (M = .092, SD = .063) across curves, and base rate estimates across indicators ranging from .052 to .12 (M = .092, SD = .031). The base rate of the average curve was .069.

The results of MAXEIG and MAMBAC analyses appear consistent with a low base rate taxon[2]; however, the base rate estimates obtained from both analyses were divergent (MAXEIG – 3.8% and 2.0%; MAMBAC – 9.2% and 6.9%). Both analyses produced base rates that clearly surpassed the point prevalence (1.62%) of IED in the epidemiological sample but that were close to its lifetime prevalence of about 7% (Allegria et al., 2003).[3]

3.3. Construct validation of the presumptive IED taxon

We sorted the cases in order of likelihood of belonging to the taxon group by creating a total severity indicator – the sum of the five scaled scores used in the taxometric analyses. Guided by the average of our base rate estimate from the MAMBAC and MAXEIG analyses (5.5%), we selected 1101 cases from the high end of the distribution of total severity scores and assigned these as taxon group members. These represented cases that obtained total scores that essentially put them in the top 5.5% of the distribution. We assigned the remaining 18,912 cases as complement group members. An alternative method of case assignment would have been to use Bayes's Theorem with MAXEIG; however, both methods of case assignment are comparable in accuracy even when base rate estimates contained substantial error (Ruscio et al., 2006). We examined the association between taxon group membership and our designated external variables. We also examined the association when the 1101 taxon group members are compared to 1101 cases drawn from the high end of the complement group in order to compare the apparent IED taxon to complement members who report severe symptoms. This removes the inflation of the observed effect size that may be caused by a significant number of cases in the complement group that reported little or no anger episodes. Our results remained virtually unchanged with this comparison (see Table 6).

First we evaluated whether taxon versus complement group members tended to meet disqualifier criterion for drinking/drug use, physical illness/medications, and sadness/depression to determine whether any of these three variables may account for taxonicity. Meeting disqualifier criteria implies that an individual's symptoms were not better accounted for by the variable.

All taxon group members (100%) successfully met disqualifier criterion for drinking/drug (i.e., none were disqualified due to drinking/drug use) compared to only 43.48% of complement members who met the same criterion, χ^2 (1, N = 301) = 124.36, p < .001, φ = .76. That is, it was apparent that their symptoms were not accounted for by drinking/drug use. Taxon group members (63.74%) were also more likely to meet the disqualifier criterion for physical illness/medications (i.e., only 36.24% of taxon members had symptoms that could have been due to physical illness/medications) when compared to complement group members (39.39%), χ^2 (1, N = 124) = 5.86, p < .05, φ = .22. Taxon group members (56.45%) were also more likely to meet the disqualifier criterion for sadness/depression than complement group members (39.63%), χ^2 (1, N = 791) = 17.83, p < .001, φ = .15 (i.e., 43.55% of taxon members had symptoms that could have been due to depression/sadness).

Next we compared taxon and complement members on the age of onset of anger attacks. It should be noted that this information was not available for all cases in the epidemiological sample given

[2] MAXCOV and L-Mode also produced CCFIs supporting a taxonic latent structure – whereas MAXCOV produced a CCFI estimate of .701, L-Mode produced a CCFI of .631.

[3] It is remarkable that the point prevalence rate is also lower than the base rate estimates produced by MAXCOV and L-Mode runs. L-Mode produced base rate estimates of .078 based on the location of the left and right modes and .05 based on the classification of cases. MAXCOV produced a base rate estimate of .054 both across curves and across indicators, but .024 for the average curve.

Table 6
Association between class membership and designated external variables.

External variable	Taxon	Comp.	Test	*p*	Top of comp.[a]	Test	*p*
Drinking/drug disq	100%	43.48%	χ^2 (1, *N* = 301) = 124.36	<.001	52.29%	χ^2 (1, *N* = 272) = 96.14	<.001
Illness/meds disq	63.74%	39.39%	χ^2 (1, *N* = 124) = 5.86	<.05	37.50%	χ^2 (1, *N* = 123) = 6.65	<.05
Sadness/depression disq	56.45%	39.63%	χ^2 (1, *N* = 791) = 17.83	<.001	40.93%	χ^2 (1, *N* = 767) = 13.94	<.001
Family history of anger attacks	71.17%	3.46%	χ^2 (1, *N* = 20,013) = 9047.01	<.001	2.18%	χ^2 (1, *N* = 2200) = 408.13	<.001
Treatment sought	28.34%	0.50%	χ^2 (1, *N* = 20,013) = 3957.82	<.001	8.17%	χ^2 (1, *N* = 2200) = 149.98	<.001
Male biological sex	57.22%	41.88%	χ^2 (1, *N* = 20,013) = 100.09	<.001	50.59	χ^2 (1, *N* = 2200) = 9.74	<.001
Age of first attack	14.23 (8.48)	17.68 (8.38)	*t* (389) = −3.89	<.001	17.76 (8.35)	*t* (375) = −3.85	<.001

[a] *Note*: comparison of taxon group with the highest scoring complement group members of equal size.

that some respondents did not report age of onset. Thus, the age analyses represent results of respondents who were able to provide such information. Taxon group members reported an earlier age of their first anger episode (14.23 years) than complement group members (17.68 years), [t (389) = −3.89, p < .001, d = .26].

Finally, we compared taxon and complement members on the remaining three external variables – treatment seeking, family history of anger attacks, and biological sex. Taxon group members (28.34%) were more likely to have sought treatment for anger episodes than complement group members (.5%), χ^2 (1, N = 20,013) = 3957.82, p < .001, φ = .45. Taxon group members (71.17%) were more likely to report that they had a close relative who had repeated anger attacks than complement group members (3.46%), χ^2 (1, N = 20,013) = 9047.01, p < .001, φ = .67. Taxon group members were also more likely to be men (57.22%) than women (42.78%), whereas complement group members were more likely to be women (58.12%) than men (41.88%). The observed distribution of gender across the taxon and complement groups departed from what may be expected by chance alone χ^2 (1, N = 20,013) = 100.09, p < .001, φ = .07. Overall, results provided strong support for the validity of the presumptive IED taxon.

4. Discussion

The objective of the present study was to determine whether IED symptoms are best viewed as representing a taxonic or dimensional construct. The results of the MAXEIG and MAMBAC analyses clearly converged on an IED taxon characterized by frequent anger attacks that are experienced as uncontrollable and out of proportion to provocation, and that result in social/psychological problems. It is noteworthy that our base rate estimates were mostly convergent across analysis; conforming to the consistency of base rate estimates that Meehl and colleagues suggested should characterize taxonicity (Meehl, 1992; Meehl and Yonce, 1994, 1996). It is noteworthy that significant deviations from normality, low base rates, and level of nuisance covariance can result in biased base rate estimates and make it difficult for us to compare with certainty; our base rate estimates from taxometric analyses to the prevalence rate of IED in the epidemiological sample (see Ruscio, 2007). Notwithstanding, the average of our base rate estimates obtained from the MAMBAC and MAXEIG analyses (5.5%) fell between the point prevalence and lifetime prevalence of IED in the sample, and the relative closeness of estimates obtained from taxometric analyses to the prevalence rates does provide some support to the validity of our taxonic findings. However, if our taxon group members are truly individuals who are currently experiencing clinically significant IED symptoms, then comparing our base rate estimates to the point prevalence of IED in the sample (1.62%), it is possible that current DSM diagnostic criteria may be underestimating the point prevalence of IED in the population. We created candidate indicators for our taxometric analyses guided by both DSM and IED research criteria; however, diagnosis in the original epidemiological survey was carried out using only DSM criteria. Thus, it may be that DSM criteria are overly restrictive and should undergo revision guided by IED research criteria. This assertion is however mitigated by the possibility that the characteristics of our research data (e.g., positive skew) increase our risk of obtaining biased base rate estimates, which could be higher or lower than they would have been in the absence of positive skew and a low base rate.

The current study represents the first attempt to investigate the latent structure of IED or any impulse-control disorder using taxometric methods. The findings from this study support a categorical model of impulsive aggression with IED symptoms producing a distinct class. One limitation of the current study is that we did not directly evaluate whether taxon group members met disqualifier status for ASPD and BPD, both of which are also characterized by features of impulsive aggression. However, given the dimensional status of both disorders in the taxometric literature (Marcus et al., 2008; Rothschild et al., 2003); it is unlikely that they are responsible for our taxonic findings. On the other hand, it may be argued that the dimensional status of ASPD and BPD, and our taxonic findings for IED, rules out any possibility of overlap between IED-related impulsive aggression and impulsive aggression observable in both personality disorders at the latent level. However, we do not evaluate this issue directly in the current study, and we perceive this as a meaningful follow-up to the results of the current study. Impulsive aggression in ASPD, BPD, mood, and substance-related disorders may stem from influences that would disqualify an IED diagnosis. The taxometric method is not conducive to directly evaluating the boundaries between disorders; thus it does not directly answer the question of whether the co-occurrence and overlap of IED symptoms with those of other axis I and II conditions is due to a shared etiological relationship or problematic reification of diagnostic categories. That is, does IED co-occur with and share features of aggression with disorders such as ASPD and BPD because they are etiologically related (e.g., lack of constraint contributing to all three) or because the boundaries between disorders are poorly conceived in the DSM (e.g., are aggression and anger not definitive of ASPD and BPD)? Nonetheless, a meaningful next step to understanding the nature of the co-occurrence or association between IED and other axis I and II conditions can be accomplished through taxometrics by examining the relationship between IED taxon group membership, and class membership when the co-occurring disorder of interest is taxonic, or severity status when the co-occurring disorder is dimensional (Ruscio and Ruscio, 2004). The existence of an association between IED taxon group membership, and class membership or severity status of the co-occurring disorder, would indicate that the co-occurrence and symptom overlap is likely due to an etiological relationship rather than poor reification of diagnostic boundaries.

Our uncovering of an IED taxon may engender questions about the possibility of additional boundaries within the IED taxon (or perhaps within the complement category). One limitation of taxometric models is that they are only adaptable to situations in

which the researcher is comparing the relative fits of a one-versus-two class model for a single latent class variable (i.e., distinguishing between two putative groups at a time). Although there are currently no theories in the IED literature about the possibility of IED subtypes, it is understood that there are subtypes of aggression in relation to other disorders characterized by impulsive aggression (e.g., antisocial behavior; Coie and Dodge, 1998). It is conceivable that within the IED taxon, meaningful subtypes may emerge differing in perhaps the predominant type of aggression (e.g., verbal versus physical) or the target of aggression (humans versus animals/objects). It is also conceivable that unveiled subtypes may differ in severity rather than types or targets of aggression. Although these questions may be answered in a taxometric framework, the family of latent variable mixture models, specifically, factor mixture models (e.g., Muthén, 2006) may be more adaptable to such comprehensive investigations.

It is interesting to consider our taxonic findings for IED in light of the growing support for dimensional views of psychiatric disorders (e.g., Carson, 1991; Widiger and Trull, 2007) and the preponderance of taxometric studies that support dimensional views for many disorders (see Haslam, 2003 for review). Although some have called for replacing the DSM categorical system with a dimensional representation of psychiatric disorders, we favor an empirical realist approach to the categorical versus dimensional debate and suggest that nosological systems reflect taxa when they are empirically supported (Meehl, 1992). One implication of our taxonic findings for IED is that there are taxonic constructs out there in the world of psychiatric disorders that are identifiable with taxometric analyses, and categorical conceptions are at least appropriate for some disorders and should not be completed discarded. Future editions of the DSM would benefit from both categorical and dimensional representations of psychiatric disorders, given the relative merits of each approach.

A particular strength of the current study is that we took preliminary steps towards establishing the construct validity of the apparent IED taxon with theoretically relevant external indicators following Waldman and Lilienfeld (2001) recommendation that the taxonic status of a construct be considered provisional while its construct validity is being established. Taxon group membership was associated with all of our designated external variables including meeting disqualifier criterion, lower age of onset, treatment seeking, family history of anger episodes, and biological sex. Taxon group members were more likely to report anger episodes not due to substance use, physical illness/medication, or sadness/depression than complement members. Our taxon group members were also more likely to have sought treatment for anger problems, report having family members with anger attacks, and were more likely to be men than women.

It is of interest that the average age of onset of reported anger problems in the taxon group was in adolescence (approximately 14 years), which is congruent with the mean age of onset (15 years) of IED symptoms reported by McElroy et al. (1998). However, the age of onset for complement group members was 17 ½ to 18 years old. The mean differences of about three to four years may reflect a meaningful difference in brain maturation and cognitive development that may be relevant to understanding the pathophysiology of the IED taxon. The results of our study suggest that family history of anger attacks, early age of onset, and male biological sex are key predictors of IED taxon group membership. One limitation of our family history data is that we were unable to investigate exact family relationships (i.e., first-degree, second-degree etc.) and their relation to the IED taxon, given that this was not available in the survey data. Differential associations between IED taxonic status and anger episodes in first and second-degree relatives would have provided more compelling support for the validity of our IED taxon. Future studies should further elucidate the role of family history (genetic and/or environmental), as well as male biological sex, on risk for developing IED symptoms.

One implication for treatment is the need to understand the relationship between taxon group membership and response to treatment modalities. It may be of interest to know if IED taxon group members possess characteristics that may make them less likely to respond to a particular form of treatment or that may worsen their overall prognosis. Recent data suggest that cognitive-behavioral therapy may be efficacious in treating IED symptoms (McCloskey et al., 2008). However, studies have generally not considered the moderating effects of characteristics that may be found in taxon group members, for example, an earlier age of onset, family history of IED, male biological sex, paternal lineage, etc. The early age of onset of IED symptoms in the taxon group (about 14 years) also suggests that treatment programs should include early identification of high risk individuals and development of preventative measures to reduce risk for becoming future taxon members or high scoring complement group members.

Further establishment of the validity of the IED taxon is required, including replication of the results of this study in other epidemiological samples or similarly large datasets. Obtaining taxonic results and similar taxon membership (same participants in the taxon at multiple points) would further lend credence to the existence of an IED taxon. Finally, the construct validation of the IED taxon has to be treated as an ongoing process (see Cronbach and Meehl, 1955) as the nomological network of IED continues to be established. As additional correlates of IED are identified through research, the ongoing process of validating the IED taxon would include determining if taxon group membership is associated with the IED correlates identified through research.

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Conflict of interest statement

The authors have no personal or financial conflicts of interest.

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