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Evidence for a heritable brain basis to deviance-promoting deficits in self-control

James R. Yancey [a], Noah C. Venables [a], Brian M. Hicks [b], Christopher J. Patrick [a,*]

[a] Department of Psychology, Florida State University, Tallahassee, FL, United States
[b] Department of Psychiatry, University of Michigan, Ann Arbor, Michigan, United States

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ABSTRACT

Purpose: Classic criminological theories emphasize the role of impaired self-control in behavioral deviancy. Reduced amplitude of the P300 brain response is reliably observed in individuals with antisocial and substance-related problems, suggesting it may serve as a neurophysiological indicator of deficiencies in self-control that confer liability to deviancy.
Methods: The current study evaluated the role of self-control capacity—operationalized by scores on a scale measure of trait disinhibition—in mediating the relationship between P300 brain response and behavioral deviancy in a sample of adult twins ($N = 419$) assessed for symptoms of antisocial/addictive disorders and P300 brain response.
Results: As predicted, greater disorder symptoms and higher trait disinhibition scores each predicted smaller P300 amplitude, and trait disinhibition mediated observed relations between antisocial/addictive disorders and P300 response. Further, twin modeling analyses revealed that trait disinhibition scores and disorder symptoms reflected a common genetic liability, and this genetic liability largely accounted for the observed phenotypic relationship between antisocial-addictive problems and P300 brain response.
Conclusions: These results provide further evidence that heritable weaknesses in self-control capacity confer liability to antisocial/addictive outcomes and that P300 brain response indexes this dispositional liability.

Introduction

Prominent criminological theories have posited that weaknesses in dispositional self-control contribute to non-normative, law-breaking behavior (Gottfredson & Hirschi, 1990). Consistent with this, various lines of empirical evidence indicate that a strongly heritable disinhibitory propensity contributes to a range of deviant outcomes, including conduct problems in childhood and early adolescence, antisocial behavior in later adolescence and adulthood, and addictive reliance on alcohol and other drugs (Krueger et al., 2002; Young, Stallings, Corley, Krauter, & Hewitt, 2000). Amplitude of the P300 brain potential response has been shown to operate as a neurophysiological indicator of problems of these types (Iacono, Carlson, Malone, & McGue, 2002; Patrick et al., 2006), exhibiting genetic variance in common with such problems (Hicks et al., 2007) and predicting their occurrence prospectively (Iacono, Malone, & McGue, 2003). The present study extended prior published research by directly evaluating, in an adult twin sample, the role of deficient self-control—operationalized in terms of scores on a brief measure of trait disinhibition—in accounting for relations between P300 brain response amplitude and clinical conditions involving salient antisocial/addictive behavior.[1] The use of data from twin participants enabled us to evaluate the contribution of genetic as compared to environmental influences to the observed overlap between trait disinhibition and antisocial/addictive problems, and to their mutual associations with P300 brain response.

Disinhibition proneness, criminality, and psychopathology

Variation in impulsivity vs. behavioral restraint (i.e., disinhibition proneness) has long been recognized as an important dimension of individual differences (James, 1890). Weaknesses in self-control prospectively predict a host of negative outcomes including poor school performance, financial instability, alcohol and drug dependence, and criminal convictions (Moffitt et al., 2011). Criminologists Gottfredson and Hirschi (1990) proposed a general theory of crime in which deficits in self-control capacity predispose an individual to act on immediate needs and desires without appropriate regard for adverse consequences. This predisposition enhances the likelihood that an individual will commit crimes to fulfill immediate goals. This central proposition has been empirically tested and replicated across many studies (Pratt & Cullen, 2000). Gottfredson and Hirschi further proposed that self-control capacity represents a *stable* trait disposition

* Corresponding author at: Department of Psychology, Florida State University, 1107 West Call Street, Tallahassee, FL 32306-4301, United States. Tel.: +1 850 645 9315; fax: +1 850 644 7739.
E-mail address: cpatrick@psy.fsu.edu (C.J. Patrick).

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that accounts for consistency in the expression of antisocial behavior over time.

Consistent with this, available empirical evidence provides support for the temporal stability of self-control capacity during childhood and early adulthood (Beaver & Wright, 2007; Turner & Piquero, 2002; Arneklev, Cochran, & Gainey, 1998;). However, a key question that has received limited attention to date in the criminological literature, is what etiological factors contribute to individual differences in self-control capacity? In this regard, a longitudinal twin study by Beaver, Wright, DeLisi, and Vaughn (2008) points to a prominent role for genetic influence in self-control capacity. These authors replicated prior research findings indicating appreciable stability of self-control scores across time, and demonstrated through twin modeling (biometric) analyses that 52-64 percent of variance in self-control scores was accounted for by genetic influence and that genetic factors accounted almost entirely for the observed temporal stability of self-control.

Intersecting with the aforementioned criminological theories and empirical findings, psychopathology researchers have posited that low dispositional restraint (trait disinhibition) confers liability to antisocial deviance and problematic substance use (Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Sher & Trull, 1994). Considerable empirical evidence has emerged in support of this hypothesis. Symptoms of alcohol problems, drug problems, and antisocial behavior exhibit a high degree of comorbidity, meaning these conditions tend to occur together in individuals and that the presence of one disorder reliably predicts the presence of others (Kessler et al., 1997; Robins & Regier, 1991; Sher & Trull, 1994; Krueger, 1999a; Krueger, Caspi, Moffitt, & Silva, 1998). Factor analytic studies of the comorbidity among mental disorders described in the current fourth edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV; American Psychiatric Association, 2000) and earlier editions of the DSM have revealed two broad factors or dimensions accounting for the systematic covariance among disorders of highest prevalence: an *internalizing* factor, representing the variance in common among fear, anxiety, and unipolar mood disorders; and an *externalizing* factor, representing the shared variance of antisocial personality and substance-related disorders (Kendler, Prescott, Myers, & Neale, 2003; Krueger, 1999a; Krueger, Caspi, Moffitt, & Silva, 1998). These broad factors have been interpreted as phenotypic manifestations of underlying vulnerabilities contributing to disorders of particular types and personality traits known to be associated with these disorders.

Previous research conducted within differing theoretical frameworks has yielded insights into personality traits associated with proneness to impulse-related (externalizing) disorders. Traits such as low Conscientiousness and low Agreeableness specified by the Big Five model of personality are known to be related to antisocial behavior (Lynam, Leukefeld, & Clayton, 2003; Trull, 1992). Relatedly, higher-order factors of Constraint and Negative Emotionality from Tellegen's temperament-oriented personality model (Patrick, Curtin, & Tellegen, 2002; Tellegen & Waller, 2008) are associated negatively and positively, respectively, with antisocial personality disorder and substance dependence, and prospectively predict the occurrence of these conditions (Krueger, 1999b) and criminal delinquency (Caspi et al., 1994). As evidence for the notion that personality traits of these types and symptoms of impulse-related disorders operate as indicators of a common liability factor, Krueger et al. (2002) reported very high (>80%) heritability for a broad externalizing factor reflecting the variance in common among disinhibitory personality traits and DSM antisocial and substance disorders (see also Kendler et al., 2003; Young et al., 2000).

To further characterize dispositional tendencies underlying the externalizing spectrum, Krueger, Markon, Patrick, Benning, and Kramer (2007) utilized traditional and modern psychometric techniques (item response modeling, exploratory/confirmatory factor analysis, hierarchical cluster analysis) to operationalize an integrative model of impulse-related problems and traits in the form of a multi-scale questionnaire measure, the Externalizing Spectrum Inventory (ESI; Krueger et al., 2007), for use with clinical and nonclinical samples. Confirmatory factor analyses demonstrated that all of the ESI's content scales load appreciably on a general externalizing factor reflecting disinhibitory tendencies. Venables and Patrick (2012) evaluated the validity of the ESI in a sample of male offenders and found strong convergence between scores on the general externalizing factor and interview-assessed symptoms of DSM-IV conditions including conduct disorder, adult antisocial behavior, and alcohol and drug dependence. These findings suggest that scores on the general factor of the ESI index weaknesses in self-control capacity that contributes to maladaptive behaviors of various types. As such, the current study used a brief item-based measure of the ESI general factor to test hypotheses regarding the etiologic basis of self-control deficits that confer liability to behavioral deviancy.

Neurophysiological indicators of disinhibitory problems

Considerable evidence indicates that reduced amplitude of the P300 brain potential response represents a neurophysiological indicator of proneness to externalizing problems. The P300 response is a positive brainwave deflection occurring in response to infrequent, task-relevant stimuli within a sequence that is theorized to reflect coordinated activity in multiple brain regions (including frontal, temporal, and parietal; Dien, Spencer, & Donchin, 2003; Polich, 2007) involved in attentional processing and memory updating (Dien et al., 2003; Polich, 2007). Reductions in P300 amplitude have been demonstrated in abstinent alcoholics compared to healthy controls (Porjesz, Begleiter, & Garozzo, 1980). Diminished amplitude of the P300 response has also shown to be associated with risk for the later development of alcohol problems. For example, children of alcoholics exhibit reduced P300 in comparison with negative family-history controls (Begleiter, Porjesz, Bihari, & Kissin, 1984; Elmasian, Neville, Woods, Schuckit, & Bloom, 1982; Hill & Shen, 2002; for review see Polich, Pollock, & Bloom, 1994). Additionally, P300 amplitude reduction in adolescence predicts later development of alcohol problems in adulthood (Berman, Whipple, Fitch, & Noble, 1993; Iacono et al., 2002). Reduced P300 has also been linked to disorders that are often comorbid with alcohol problems, such as drug dependence (Attou, Figiel, & Timsit-Berthier, 2001; Biggins, MacKay, Clark, & Fein, 1997; Branchey, Buydens-Branchey, & Horvath, 1993), child conduct disorder (Bauer & Hesselbrock, 1999a, 1999b; Kim, Kim, & Kwon, 2001), and adult antisocial behavior (Bauer, O'Connor, & Hesselbrock, 1994; Costa et al., 2000). Taken together, available research suggests that reduced P300 amplitude may index the common genetic risk for problems including childhood and adult antisocial behavior and addictions to alcohol and drugs (Iacono et al., 2002, 2003).

In turn, the foregoing lines of evidence raise the possibility that reduced P300 amplitude may serve as an indicator of deficits in self-control capacity that have long been theorized to play a crucial role in criminal deviancy (Gottfredson & Hirschi, 1990). In support of this possibility, Patrick et al. (2006) demonstrated amplitude of P300 response to be inversely related to scores on the broad externalizing factor reflecting the covariance among multiple impulse-related disorders (cf. Krueger et al., 2002), and Hicks et al. (2007) showed that this P300/externalizing relationship was attributable mainly to genetic influence. This finding, together with evidence that genes account substantially for the stability of self-control capacity across time (Beaver et al., 2008) and that low P300 amplitude predicts the emergence of externalizing problems across time, raises the intriguing possibility that reduced P300 represents a neural indicator of deficient self-control capacity as discussed in classic criminological theories.

Current study aims

The current study sought to extend the aforementioned literature by evaluating phenotypic correlations among symptoms of antisocial/

addictive disorders, self-control capacity as operationalized by scores on a trait disinhibition scale, and P300 brain response amplitude, and testing the hypothesis that trait disinhibition scores would account for (i.e., mediate) the relationship between P300 amplitude and symptoms of disinhibitory disorders. The use of data from a genetically informative sample (adult twins; $N = 419$) permitted estimation of genetic and environmental contributions to the observed association between P300 amplitude and antisocial/addictive disorders, and to the mediating role of trait disinhibition in this association. We hypothesized that (1) trait disinhibition would mediate relations between antisocial/addictive disorders and P3 response, and (2) that the variance in common between trait disinhibition and antisocial/addictive disorders accounting for their mutual relations with P300 would largely reflect genetic influences.

Methods

Participants

Participants consisted of 508 (257 female, 251 male) twins (260 monozygotic [MZ], 248 dizygotic [DZ]) recruited from the University of Minnesota Twin Registry and screened for hearing and visual impairments prior to testing. A total of 89 participants were excluded from analyses due to technical issues with electroencephalogram (EEG) recording ($n = 12$), withdrawal from participation ($n = 4$), incomplete clinical data ($n = 31$), or excessive artifact in the EEG recording ($n = 42$; described further below) resulting in 419 participants for analyses (225 MZ, 194 DZ; 199 males, 220 females) with a mean age of 29.4 ($SD = 4.8$; range = 22-38). Procedures for the study were approved by the University of Minnesota's institutional review board (IRB), and all participants provided informed written consent prior to testing. Data presented in this manuscript were collected as part of a larger protocol for which participants received $100 as compensation along with reimbursement for travel expenses.

Diagnostic assessment

Structured clinical interviews for DSM-IV-TR Axis-I Disorders (SCID-I; First, Spitzter, Gibbon & Williams, 2002) and Axis-II Disorders (SCID-II; First, Spitzer, Gibbon, Williams, & Benjamin, 1997)

Participants were administered the SCID-I interview to assess for symptoms of DSM-IV clinical conditions including anxiety, mood, eating, substance use, and psychotic disorders. Participants were also administered the Cluster B module of the SCID-II interview (First et at., 1997) to assess for symptoms of DSM-IV personality disorders involving deficient impulse control. SCID-I and SCID-II interviews and symptom ratings were completed by clinical psychology graduate students under the supervision of Ph.D. psychologists to ensure uniform practices for assigning clinical ratings. The present study focused on symptoms of antisocial behavior and substance use disorders, which have previously been the focus of P300 studies. Conduct disorder and adult antisocial behavior, respectively, were quantified as the number of child and adult symptoms of antisocial personality disorder endorsed by the participant. Substance use problems were quantified as an average of standardized symptom counts for abuse and dependence across alcohol and all illicit drug classes. Lastly, a composite of symptoms of adult antisocial behavior, conduct disorder, and substance use disorders was computed as a general index of externalizing problems. A Blom transformation was applied to individual diagnostic variables to correct for skewness (cf. Krueger et al., 2002).

Trait disinhibition

As part of the study protocol, participants completed a 100-item version of the Externalizing Spectrum Inventory (ESI; Krueger et al., 2007) that included representation of the ESI's 23 content scales. Trait disinhibition was operationalized using available items (=30) from the following ESI subscales, which load most strongly and selectively on the ESI's general externalizing factor: Irresponsibility, Dependability, Problematic Impulsivity, Impatient Urgency, Planful Control, Alienation, and Theft. The 30 items comprising the Trait Disinhibition scale exhibited high internal consistency reliability (Cronbach's $\alpha = .88$).

Experimental stimuli and design

The visual oddball task in the study consisted of a modified version of the two-stimulus 'rotated-heads' paradigm developed by Begleiter, Porjesz, Bihari, and Kissin (1984), with neutral and affective picture stimuli included as a third (novel) stimulus category. Simple oval shapes served as frequent non-target (standard) stimuli, occurring 70% of the time (i.e, on 168 of the 240 task trials). The target stimuli were schematic heads, each consisting of the same oval shape accompanied by a stylized nose and ear (15% of all stimuli presented [$N = 36$ trials]). For each target trial, the task was to press the left or right button on a button-box, with either the left or right hand respectively, to indicate whether the ear was on the left or right side of the head. On 50% of target trials, the nose was pointed up; on the remaining trials, the nose was pointed down, requiring participants to discern that the ear was on the side of the head opposite to its spatial position on the screen. The novel stimuli, comprising 15% ($N = 36$) of task stimuli, consisted of pleasant, neutral, and unpleasant pictures from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008) interspersed randomly through the stimulus sequence. Before commencing the test procedure, participants practiced to a criterion level of 85% accuracy using a version of the task that included only target and standard (oval) stimuli.

Stimulus delivery and recording procedure

During the experiment, participants viewed the stimuli on a 21" computer monitor while seated in a comfortable recliner, and made responses using a serial response box. The monitor was situated 1 m from participants' eyes, and the response box was positioned on their laps. Stimuli were presented using an IBM compatible computer running E-Prime software (Psychology Software Tools). All task stimuli (ovals, target heads, and novel pictures) were displayed within a rectangular frame, filled with dark gray, which appeared against a black background. Stimuli were presented for 100 ms each, with a variable intertrial interval between 4 and 5 s. Picture presentation was counterbalanced across participants using 12 different stimulus orders, in which the presentation of oval, target head, and novel stimuli was randomized with constraints (i.e., no more than four oval stimuli appeared consecutively, no two target types appeared consecutively, and no two novels appeared consecutively).

Physiological measurement and data reduction

A second IBM compatible computer, running Neuroscan Acquire software, was used for physiological data acquisition. EEG activity was recorded from 53 scalp sites positioned according to the 10-20 system using Neuroscan Quik-Caps with sintered Ag-AgCl electrodes. Electrodes were positioned above and below the left eye to monitor vertical electrooculogram (VEOG) activity and adjacent to the outer canthi of the left and right eyes to monitor horizontal electrooculogram (HEOG) activity. All electrode impedances were kept below 10 KOhms. EEG signals were digitized on-line at 1000 Hz during data collection with an analog band pass filter of .05-200 Hz. Data were referenced to electrode site Cz during on-line data collection and arithmetically re-referenced off-line to the average of left and right mastoid electrodes for subsequent processing and analysis. Data epochs from –1000 ms to 2000 ms were extracted from the continuous EEG recordings using Neuroscan EDIT software (version 4.3, Neuroscan Inc.), and corrected for eye movements using the

algorithm developed by Semlitsch, Anderer, Schuster, and Presslich (1986), as implemented within the EDIT software. The segmented and eye-blink corrected EEG data were then imported to Matlab (Mathworks, Inc.) for subsequent processing, including downsampling to 128 Hz using the Matlab resample command, which applies a low pass anti-aliasing filter before downsampling.

Trials in which activity exceeded ±75 μV either in the pre- (-1000 ms to 0) or poststimulus (0 to 2000 ms), were excluded from further processing. Visual inspection of each participant's average waveforms was undertaken to evaluate the effectiveness of the aforementioned criteria. Electrodes deemed to contain excessive artifact were replaced by their nearest neighboring sites. Participants were excluded if more than 25% of trials were rejected due to excessive artifact ($n = 42$; 8.5% of total sample). Using the grand average across participants to guide window selection, target P300 amplitude was calculated as the maximum voltage peak occurring between 297 ms and 602 ms relative to a preceding baseline (i.e., -136 to -8 ms). Prior studies documenting P300/externalizing associations (cf. Iacono et al., 2003; Patrick et al., 2006) have focused on recording site Pz, a parietal location along the midline of the scalp, where target P300 amplitude is maximal. Following this precedent, P300 at Pz was utilized in the reported analyses.

Data analyses

Correlational analyses were first performed to evaluate zero-order associations for target P300 amplitude with symptom variables (adult antisocial behavior, conduct disorder, substance use) and trait disinhibition scores. Following this, regression analyses were conducted to test for unique and shared effects of externalizing disorder symptoms and trait disinhibition on P300 response. As recommended by Preacher and Hayes (2008), an analytic approach entailing normal theory tests, estimation of indirect effects, and bootstrapped confidence intervals was used to formally evaluate the hypothesis that scores on trait disinhibition would account for (i.e., mediate) the relationship between P300 response amplitude and antisocial/addictive problems. Findings from these analyses are reported following the regression analysis results.

Lastly, we leveraged the twin data to evaluate the contributions of genetic and environmental influences to P300 amplitude, a composite index of externalizing disorder symptoms, trait disinhibition scores, and their covariance by fitting standard biometric models (Neale & Cardon, 1992). These models conceptualize the variance of a phenotype or trait to be attributable to three sources: additive genetic (A), shared environmental (C), and nonshared environmental (E) influences. Estimating the relative contributions of these genetic and environmental influences was accomplished by comparing the similarity of MZ twins (who share all of their genetic material) on a phenotype relative to the similarity of DZ twins (who shared on average 50% of their segregating genes) on the phenotype. More specifically, genetic influences on a trait are inferred if the correlation between scores for MZ twins is greater than the correlation for DZ twins ($r_{MZ} > r_{DZ}$). Heritability is then computed as the ratio of the genetic variance to the total phenotypic (genetic plus environmental) variance. In the case where $r_{MZ} > 2r_{DZ}$, it can be inferred that all of the similarity between members of a twin pair is due to genetic influences. Shared environmental influences refer to environmental influences that contribute to similarity among family members on a trait, and are inferred if $2r_{DZ} > r_{MZ}$. Nonshared environmental influences refer to environmental influences that contribute to differences among family members, and are inferred if $r_{MZ} < 1$, that is, if MZ twins are not identical in terms of scores on a trait. This component of variance includes measurement error along with systematic but nonshared sources of environmental influence.

These models can be readily extended to the multivariate case using a Choleksy decomposition to distinguish genetic environmental influences that are unique to a given phenotype and those that are shared with other phenotypes. Estimates of the genetic covariance can then be standardized to quantify the genetic correlation between two phenotypes, which provides an index of the amount of heritable variance that is shared between two phenotypes. Similar correlations can also be calculated to index the amount of overlapping shared and nonshared environmental influences across traits. Notably, genetic and environmental correlations are independent of the heritability of a trait. For example, the heritability estimates for two traits could be high, but the genetic correlation between the two could be low, and vice versa. Finally, the Choleksy decomposition can also be used to estimate the extent to which the phenotypic association between two traits is attributable to genetic and environmental influences.

All models were fit with the computer program Mx (Neale, Booker, Xie, & Maes, 2002) using full information maximum likelihood information, which can accommodate missing data. Model fit was evaluated by comparing -2 times the log-likelihood (-2LL). The difference in the -2LL between nested models approximates a χ^2 distribution, which permits a likelihood ratio test to compare the relative fit of competing models. We also used Akaike's Information Criterion (AIC) to evaluate model fit. The AIC fit statistic balances overall fit with model parsimony and penalizes fit for unnecessary parameters (χ^2 – 2df), with lower values indicative of better fit.

Results

Associations among externalizing disorders, trait disinhibition, and P300 amplitude

Zero-order correlations for externalizing symptom variables and trait disinhibition scores with P3 brain response amplitude are presented in Table 1. Symptoms of antisocial behavior and substance use disorders showed modest but significant inverse associations with P300 amplitude (*r*s ranged -.14 to -.10), as did the composite index of externalizing problems (i.e., average of all symptom counts; r = -.14). Trait disinhibition scores also showed a significant inverse relationship with P300 amplitude. Fig. 1 depicts average ERP waveforms across all target stimulus trials for individuals scoring high versus low on trait disinhibition (upper and lower quartiles, respectively, of the sample distribution) to illustrate the nature of effects modeled statistically as continuous scores. As can be seen in the Figure, those scoring high on Disinhibition exhibited clearly reduced amplitude of the P300 brain potential response relative to those scoring low.

Next, to test for unique versus shared contributions to prediction, a series of regression analyses was performed in which Disinhibition scores were entered along with symptoms of individual antisocial and substance use variables as predictor of P300 amplitude. Results from these analyses are also displayed in Table 1. In each analysis, the overall model emerged as significant, with Disinhibition alone contributing distinctively to prediction of P300 amplitude (βs ranged from -.14 to -.16).

Table 1
Associations of disorder symptom variables and trait disinhibition scores in the prediction of target P300 amplitude: Zero-order correlations and regression model results

	Zero-Order	Regression Model (symptom counts + Disinhibition scores)		
	Pearson's *r*	Multiple *R*	Symptoms β	Disinhibition β
Adult Antisocial Behavior	**-.140****	**.184****	-.062	**-.142***
Conduct Disorder	**-.099***	**.178****	-.031	**-.163****
Substance Abuse	**-.125****	**.181****	-.047	**-.152****
Symptom Composite	**-.142****	**.183****	-.062	**-.142***
Trait Disinhibition	**-.176****	–	–	–

Note: Coefficients are for the prediction of P300 brain response amplitude. *$p < .05$; **$p < .01$.

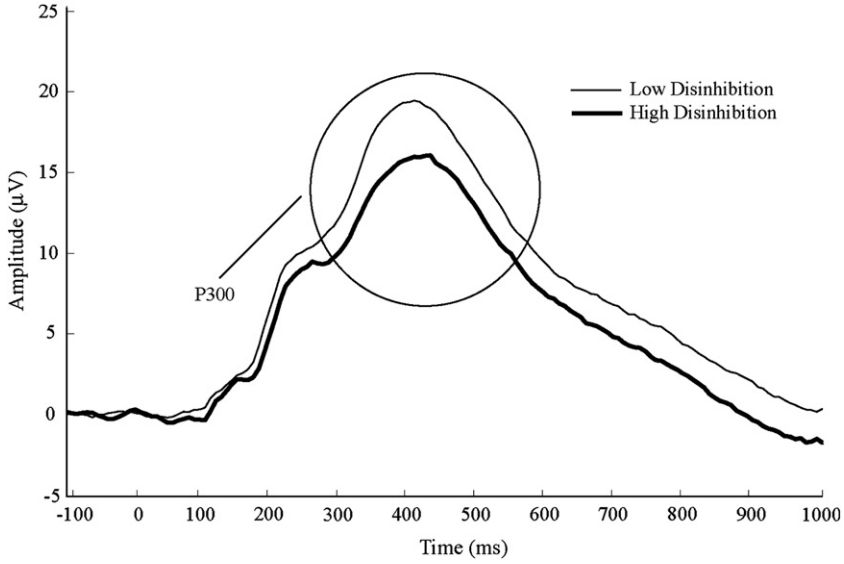

Fig. 1. Average P300 brain response waveform for target stimuli at electrode site Pz for participants scoring in lowest and highest quartiles of the distribution of Disinhibition scores for the study sample.

The mediating role of disinhibition

According to the heuristic outlined by Baron and Kenny (1986), results from the foregoing regression analyses suggest that the associations between externalizing symptom variables and P300 amplitude were mediated by trait disinhibition. Fig. 2 depicts the case of mediation in which adult antisocial behavior, conduct disorder, and substance use disorder symptoms affect P300 through disinhibition. More formal statistical analyses (normal theory tests, indirect effect estimates) confirmed that disinhibition significantly mediated the relationship between target P300 and each symptom variable: adult antisocial behavior, $z = -2.43$, $p < .05$, a X $b = -.08$, 95% CI = [-.13, -0.03]); conduct disorder, $z = -2.93$, $p < .01$, a X $b = -.07$, 95% CI = [-.10, -.03]; substance use disorder, $z = -2.65$, $p < .01$, a X $b = -.08$, 95% CI = [-.12, -.03]). Trait disinhibition also mediated the relationship between reduced P300 and the composite index of externalizing problems, $z = -2.37$, $p < .05$, a X $b = -.08$, 95% CI = [-.13, -.03]. These findings indicate that observed relationships between antisocial/addictive disorders and reduced P300 brain response reflect the influence of an underlying trait of disinhibition proneness.

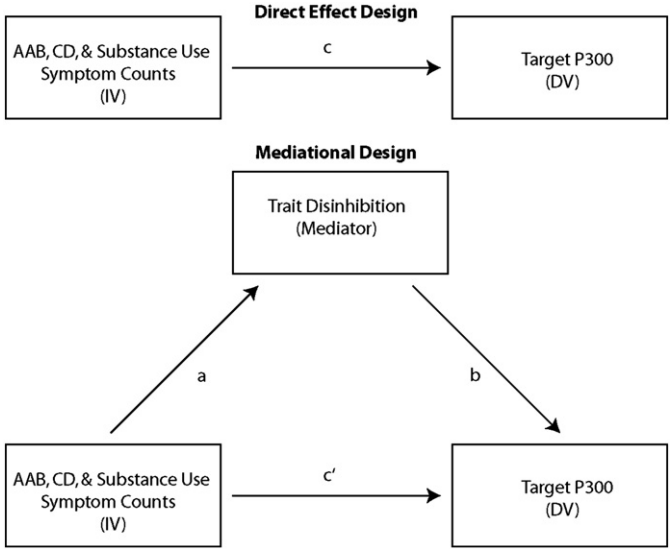

Fig. 2. Schematic depiction of direct-effect and mediational designs for relationship between individual disorder symptom variables (AAB = adult antisocial behavior, CD = conduct disorder, and Substance Use = unit-weighted average of abuse and dependence symptoms for alcohol and all illicit drugs) and amplitude of P300 brain response. In the direct effect design, *c* represents the total effect of the IV (symptom variable) on the DV (P3 amplitude), including the effect of the mediator (trait disinhibition); in the mediational design, *a* reflects the effect of the IV on the mediator, *b* represents the effect of the mediator on the DV, and *c'* represents the effect of the IV on the DV after controlling for associations of each with the mediator.

Trait disinhibition, P300, and externalizing psychopathology: The role of genetic contributions

Next, we used the twin data to estimate the relative contributions of genetic and environmental influences to the variance and covariance among trait disinhibition, the composite of externalizing symptoms, and P300 amplitude. The Mx-generated MZ and DZ correlations for the three variables are reported in Table 2. For each variable, the MZ correlation was greater than the DZ correlation, indicating additive genetic influence on each phenotype. For trait disinhibition and P300 amplitude, $r_{MZ} > 2r_{DZ}$, indicating twin similarity was due solely to additive genetic influences. For the composite of externalizing symptoms, $2r_{DZ} > r_{MZ}$, indicating some influence of shared environmental factors. Given the observation of $r_{MZ} < 1$ for all variables, a contribution of nonshared environmental influences was also inferred for each phenotype.

We then fit the Cholesky model to derive more precise estimates of the additive genetic, shared environmental, and nonshared

Table 2
Twin correlations and estimates of additive genetic (A), shared environmental (C), and nonshared environmental variance components (95% condence intervals) for disinhibition, externalizing, and P300 amplitude

Variable	MZ*r*	DZ*r*	A	C	E
Disinhibition	.60	.24	.59 (.46, .69)	.00	.41 (.31, .54)
Externalizing	.78	.56	.52 (.40, .64)	.26 (.16, .35)	.22 (.17, .30)
P300 amplitude	.69	.09	.70 (.57, .78)	.00	.30 (.22, .43)

Note. In the best fitting model, C is fixed to zero for Disinhibition and P300 amplitude; thus, confidence intervals are not provided. MZ*r* and DZ*r* = correlations for MZ and DZ twin pairs, respectively, generated using Mx (Neale et al., 2002).

environmental influences on each phenotype and the covariance among them (model fit -2LL = 2802.43, df = 1224, AIC = 354.43). In addition to full ACE models, we tested more parsimonious models by dropping paths from the full model and examining the impact on model fit. Because the twin correlations suggested no shared environmental influences on trait disinhibition and P300 amplitude, we dropped the shared environmental effects on those variables from the model (resulting also in omission of shared environmental influences on the covariance among the variables within the model). This change resulted in no significant chi-square change while yielding a reduction in AIC ($\chi^2(5) = 1.74$, $p > .10$, AIC = 346.17), indicating improved model fit. Next, because the genetic correlation between trait disinhibition and the composite of externalizing symptoms was so high within the full Choleksy model ($r_a = 0.97$, 95% CI = [0.60-1.00]), we tested whether the genetic correlation could be fixed to 1.00 without a significant decrease in model fit. We found this to be the case ($\chi^2(2) = 0.10$, $p > .10$, AIC = 342.26), indicating that trait disinhibition scores and the externalizing symptom composite reflected overlapping genetic liabilities. Finally, the nonshared environmental correlation between the composite of externalizing symptoms and P300 amplitude in the full Cholesky model was small and non-significant (r_e = -0.10, 95% CI = [-0.30-0.11]), and thus could be dropped from the model without a decrement in fit ($\chi^2(1) = 1.21$, $p > .10$, AIC = 341.47).

A visual depiction of the final model is provided in Fig. 3. In the figure, there is a latent genetic factor that represents the genetic liability underlying trait disinhibition and the composite of externalizing symptoms. This factor is connected to a second genetic factor that represents the genetic influences on P300 amplitude. The number next to the path connecting the latent genetic factors is the genetic correlation, and squaring the coefficients for the paths connecting the genetic factors to the measured phenotypes provides the estimate of heritability for each variable. The composite of externalizing symptoms also includes a latent shared environmental factor that accounts for a portion of its variance, and each variable is also influenced by distinct (but correlated) latent nonshared environmental factors. Estimates of the genetic and environmental variance components and 95% CI's are provided in Table 2. Trait disinhibition and P300 amplitude exhibited high heritability, no shared environmental influences, and moderate nonshared environmental influences. The composite of externalizing symptoms exhibited moderate heritability and small to moderate shared and nonshared environmental influences.

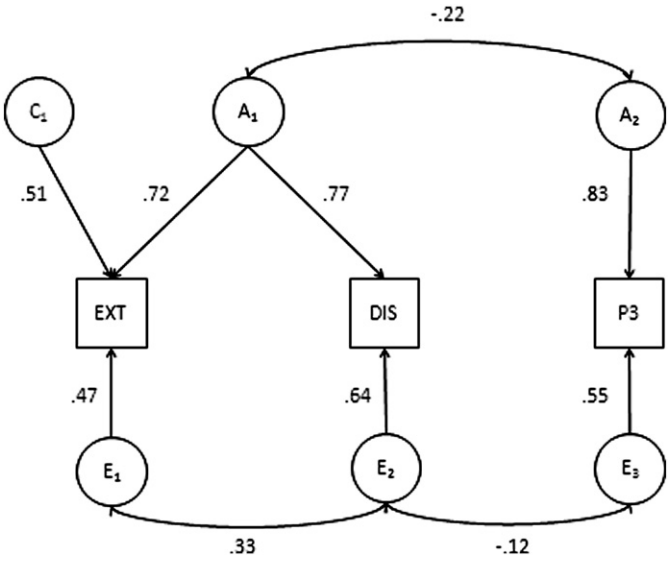

Fig. 3. Associations among factors reflecting additive genetic (A), shared environmental (C), and non-shared environmental (E) contributions to trait disinhibition (DIS), a composite of antisocial behavior and substance use disorder symptoms (EXT), and P300 response amplitude. Squaring the coefficients next to the paths connecting the ACE factors to the manifest variables provides estimates of the ACE variance components for each. Coefficients next to the curved lines connecting the ACE factors to one another other reflect genetic (A_1 with A_s) and nonshared environmental (E_1 with E_x) correlations.

Table 3
Phenotypic, genetic, and nonshared environmental correlations (95% confidence intervals) among disinhibition, externalizing, and P300 amplitude

				Percent of covariance	
Variables	r_p	r_a	r_e	A	E
Disinhibition-Externalizing	.65 (.59, .71)	1.00	.33 (.17, .47)	85%	15%
Disinhibition-P300	-.19 (-.28, -.08)	-.22 (-.37, -.06)	-.12 (-.27, .03)	77%	23%
Externalizing-P300	-.13 (-.23, -.04)	-.22 (-.37, -.06)	.00	100%	0%

Note. In the best fitting model, the genetic correlation between Disinhibition and Externalizing is fixed to 1.00, and the nonshared environmental correlation between Externalizing and P300 is fixed to 0.00; thus, confidence intervals are not provided for these parameter estimates. Additionally, shared environmental influences were limited to the Externalizing variable only and so did not contribute to the covariance among the variables. r_p = phenotypic correlation; r_a = additive genetic correlation; r_e = nonshared environmental correlation.

Table 3 provides the phenotypic, genetic, nonshared environmental correlations and 95% CIs for these correlations. As already discussed, the genetic correlation between trait disinhibition and the composite of externalizing symptoms could be fixed to 1.00; thus, within the model, trait disinhibition and externalizing exhibited the same genetic correlation with P300 amplitude (r_a = -0.22). Trait disinhibition also evinced medium and small nonshared environmental correlations, respectively, with the composite of externalizing symptoms and with P300 amplitude. Finally, a large phenotypic correlation was evident between trait disinhibition and the composite of externalizing symptoms, with almost all (85%) of the association due to genetic influences. P300 amplitude exhibited small but robust phenotypic correlations with trait disinhibition and with the composite of externalizing symptoms. Genetic influences alone accounted for the relationship between P300 amplitude and the composite of externalizing symptoms, and for most (77%) of the association between P300 amplitude and trait disinhibition.

Discussion

Based on theoretical conceptions and empirical findings from the criminological and psychiatric literatures, we hypothesized that deficiencies in self-control, operationalized through a self-report index of trait disinhibition reflecting general externalizing proneness (cf. Krueger et al., 2007), would account for associations between behavioral pathology in the form of DSM-IV disorder symptoms and reduced P300 amplitude. Results from regression analyses and normal theory and indirect effects tests provided compelling evidence that trait disinhibition mediated relations between externalizing disorders and reduced P300 amplitude in the current sample. Additionally, we predicted that the phenotypic association between trait disinhibition and antisocial/addictive problems would reflect common heritable variance, and that this common heritable variance would account for observed associations of P300 with externalizing problems and trait disinhibition. Consistent with this, results from biometric analyses confirmed that the variance in common between symptoms of antisocial/addictive disorders and trait disinhibition was largely attributable to common genetic influences and that these influences accounted almost entirely for observed relations with P300.

Thus, results from the current study demonstrate that genetic contributions to symptoms of antisocial behavior and substance use disorders overlap strongly with those for trait disinhibition. Additionally, the

common liability indexed by trait disinhibition scores and aggregate symptoms of antisocial and substance-related disorders accounts for the phenotypic relations of each with reduced P300. Although the magnitude of genetic overlap between P300 and trait disinhibition was modest in magnitude (r_A = -.22), the level of association was consistent with that reported by Hicks et al. (2007) in a separate sample of adolescent participants from the community. Relatedly, molecular genetic studies of single gene markers and mental disorders indicate that genetic markers account for relatively modest amounts of variance in psychiatric disorders (Faraone, Doyle, Mick, & Biederman, 2001). Furthermore, whereas studies involving specific gene loci and phenotypic expression of disorders have not consistently replicated (Hirschhorn, Lohmueller, Byrne, & Hirschhorn, 2002; Prathikanti & McMahon, 2001), results indicating a strong genetic basis to externalizing and P300 associations have replicated across multiple samples varying in age and gender.

Findings from the current study replicate and extend previous work indicating that a common vulnerability underlies antisocial/addictive disorders (Krueger et al., 2002; Young et al., 2000) and that reduced P300 response serves as a neurophysiological indicator of this common vulnerability. Vis-à-vis aforementioned theories of crime, our results suggest that reduced P300 response represents a neural indicator of deficient self-control as discussed by Gottfredson and Hirschi (1990). As such, our results have important implications for criminological perspectives on the origins of behavioral deviancy and for research on the role of neurobiological systems in impulse control problems more broadly.

Before discussing broader implications, it is important to acknowledge certain key limitations of the current study. One limitation concerns the potential generalizability of the findings. Because the current study utilized a community sample, the base rates of antisocial and substance use problems were lower in comparison with correctional or other samples in which impulse control problems are common (i.e., 23% of the current sample exhibited no symptoms of antisocial behavior or substance use disorders). Given research demonstrating similar correlates for trait disinhibition in correctional samples (Venables & Patrick, 2012), we would predict a similar role for disinhibition proneness in mediating P300/externalizing associations in samples of this type. However, this possibility should be directly tested using data from correctional and other samples in which more severe externalizing problems are common.

A further limitation of the present study is that it relied exclusively on interview-based assessment of maladaptive behaviors, which may be susceptible to some of the same problems of distortion as self-report (cf. Lilienfeld & Fowler, 2006). Accordingly, it would be desirable in future studies to include additional measures of criminal/antisocial behavior, including objective archival measures (e.g., offenses as listed in official criminal records). Yet another limitation concerns the use of P300 amplitude as a neurobiological indicator of disinhibitory proneness. While reduced amplitude of P300 brain response operates as a reliable neurophysiological indicator of externalizing psychopathology, little is known about specific cognitive mechanisms underlying this association. To address this issue, it will be important in future research to incorporate experimental task procedures specifically designed to test for processes reflected in P300 reductions. Additionally, research using alternative measurement methods with greater effectiveness for localizing sources of brain activation in lab task procedures (e.g., fMRI) would also be valuable.

Conclusions: Implications and future directions for research of self-control

Notwithstanding these limitations, the current findings have important implications. Harkening back to Gottfredson and Hirschi (1990), one major critique of the self-control theory is that it discounts the importance of biological factors contributing to deficient self-control (Beaver et al., 2008). Our findings join the growing body of evidence from disciplines of psychology, criminology, and neuroscience indicating an important contribution of biogenic influences to deficient self-control (Haberstick et al., 2005; Johnson et al., 2005; Larsson et al., 2004). Also consistent with previous findings (Cohen, 1999; Wright & Beaver, 2005), we found no evidence for shared environmental effects on self-control capacity operationalized as trait disinhibition, although we did find some contribution of shared environment to antisocial-addictive symptoms (albeit markedly lower than that of genes). In view of these findings, a revision of self-control theory that places emphasis on the role of genetic influences is clearly warranted. Moreover, the findings of the current study pertaining to P300 as a marker of the heritable variance in trait disinhibition indicate a role for brains mechanisms in deficient self-control. Focusing on biological and neurocognitive processes in relation to self-control theories of criminality can lead to new lines of inquiry in criminological research. For example, does P300 prospectively predict recidivism or career criminality, and would disinhibition mediate such a predictive relationship?

The current findings also have implications for identifying and pursuing targets for neurobiological studies of criminality, antisocial personality disorder, substance use problems, and psychopathology more broadly. Disinhibition, operationalized using a brief (i.e., 30-item) scale measure, proved highly effective in indexing heritable tendencies toward antisocial/addictive problems in the current study. This approach to assessing externalizing tendencies can serve as an alternative to more time-/resource-intensive interview based approaches in large-scale screening studies or multi-measure assessment protocols. Results from the present study are also of relevance to current initiatives (e.g., Sanislow et al., 2010) directed at redefining clinical problems in neurobehavioral as opposed to traditional diagnostic (e.g., DSM or ICD) terms. In the context of such efforts, trait disinhibition scores can serve as valuable self-report based referent for investigation of neurobiological and genetic factors relevant to deviant and criminal behavior. Related to this, trait disposition indexed in this way can serve as a useful point of contact between criminological and psychiatric investigations of biological-genetic factors that contribute to antisocial deviancy.

In sum, the findings of this study indicate that dispositional differences in self-control operationalized as trait disinhibition account for reductions in amplitude of P300 brain response in individuals exhibiting antisocial and substance use problems, and that the variance in trait disinhibition that accounts for the relationship between externalizing problems and P300 response reflects mostly genetic influences. As such, our findings provide compelling evidence that deficits in self-control contributing to behavioral deviancy in the form of antisocial/addictive behavior are substantially heritable, and based at least partly in disturbances of brain function.

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Note

1. The alternative label "P3b" is sometimes applied to the P300 response to target stimuli in an oddball task context, to distinguish it from the somewhat earlier-occurring P3 response to novel stimulus events (termed "P3a"). In line with prior research focusing on brain response to target stimuli in relation to impulse-control problems (e.g., Iacono et al., 2002, 2003; Patrick et al., 2006), we use the term "P300" through the current paper.

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