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Amygdala Reactivity and Negative Emotionality: Divergent Correlates of Antisocial Personality and Psychopathy Traits in a Community Sample

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Abstract

Previous studies have emphasized that antisocial personality disorder (APD) and psychopathy overlap highly but differ critically in several features, notably negative emotionality (NEM) and possibly amygdala reactivity to social signals of threat and distress. Here we examined whether dimensions of psychopathy and APD correlate differentially with NEM and amygdala reactivity to emotional faces. Testing these relationships among healthy individuals, dimensions of psychopathy and APD were generated by the profile matching technique of Lynam and Widiger (2001), using facet scales of the NEO Personality Inventory-Revised, and amygdala reactivity was measured using a well-established emotional faces task, in a community sample of 103 men and women. Higher psychopathy scores were associated with lower NEM and lower amygdala reactivity, whereas higher APD scores were related to greater NEM and greater amygdala reactivity, but only after overlapping variance in APD and psychopathy was adjusted for in the statistical model. Amygdala reactivity did not mediate the relationship of APD and psychopathy scores to NEM. Supplemental analyses also compared other measures of factors within psychopathy in predicting NEM and amygdala reactivity and found that Factor 2 psychopathy was positively related to NEM and amygdala reactivity across measures of psychopathy. The overall findings replicate seminal observations on NEM in psychopathy by Hicks and Patrick (2006) and extend this work to neuroimaging in a normative population. They also suggest that one critical way in which APD and psychopathy dimensions may differ in their etiology is through their opposing levels of NEM and amygdala reactivity to threat.

Keywords

neural reactivity; statistical suppression; psychopathic personality inventory; fearlessness; crime

Aggression and antisocial conduct comprise attributes of two related psychopathologies, antisocial personality disorder (APD) and psychopathy. APD is recognized by early involvement in antisocial acts, reckless disregard for the rights of others, and a persistent pattern of impulsive and irresponsible behavior. Psychopathy subsumes many of the same traits as APD, but in addition, includes characteristic interpersonal and affective features, such as superficial charm, deceitful and manipulative behavior, callousness, and lack of empathy or remorse. Factor analytic reduction of one prominent psychopathy index, the Psychopathy Checklist – Revised (PCL-R; Hare, 1991), commonly yields two factors, termed Factor 1 (F1) and Factor 2 (F2) (though see Cooke & Michie, 2001). F1 reflects psychopathy's distinctive interpersonal and unemotional qualities, whereas F2 taps the expressions of antisocial disposition and impulsivity common to both psychopathy and APD (though note that the APD diagnosis does include some F1 symptoms including deceitfulness and lack of remorse). Hence, most psychopaths meet criteria for APD (at least in prison settings, see Babiak, Neumann, & Hare, 2010), but most people with APD are not judged psychopathic (Patrick, 2007). While these factors (i.e., F1, F2) and constructs (i.e., APD, psychopathy) are highly correlated, they often show differential associations with outcome variables (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Hicks, Markon, Patrick, Krueger, & Newman, 2004) and even predict outcomes such as suicide in *opposite* directions (Hicks & Patrick, 2006; Verona, Hicks, & Patrick, 2005).

The asymmetric relationship of psychopathy and APD has prompted much speculation regarding etiologic factors that might differentiate the two disorders (though the differences between APD and psychopathy are quite understudied in comparison to the bulk of work comparing psychopathy F1 and F2). Differences in NEM are often cited as one distinguishing aspect, and it is well-established that psychopathic individuals respond minimally to situations thought to induce negative emotions such as experimental exposure to threat and stressors (Arnett, 1997; Lorber, 2004; Patrick, Bradley, & Lang, 1993). These findings have often been interpreted as reflecting *low levels* of anxiety or fear (Fowles, 2007; Lykken, 1957) specific to psychopathy (especially F1), whereas those with APD often show *greater* anxiety and fear (Lorber, 2004; Patrick et al., 1993).

Suppressor Effects

Although low anxiety is described as a core component of psychopathy (Cleckley, 1976), studies linking NEM to globally defined psychopathy yield mixed findings (Hare, 1991; Harpur, Hare, & Hakstian, 1989; Schmitt & Newman, 1999). As Hicks and Patrick (2006) note, the psychometric characteristics of the PCL-R may obscure the role of anxiety, which is a core component of NEM, in psychopathy. In particular, it has been is argued that NEM may covary negatively with the callous detachment described by the PCL-R's first factor, but positively with the impulsive and antisocial traits of the second factor, thereby tending to suppress a statistical association of NEM with globally defined psychopathy (Patrick et al., 1993). Providing support for this claim, Patrick (1994) found trait negative affect to covary

negatively with F1 and positively with F2 when adjusting by partial correlation for the overlap of F1 and F2.

More recently, Hicks and Patrick (2006) described this relationship between PCL-R factors and NEM as an instance of "cooperative suppression," which occurs when entering two correlated predictor variables in a regression model increases the beta-coefficient of one or both predictors above their bivariate associations with the predicted variable (Paulhus, Robins, Trzesniewski, & Tracy, 2004). The two predictors may be highly correlated and the inclusion of both variables (and thus partitioning of shared variance) in the same model may uncover opposing relations with the dependent measure (Mackinnon, Krull, & Lockwood, 2000). In a study assessing multiple facets of negative affect among incarcerated adult males, Hicks and Patrick (2006) confirmed opposite relations of NEM with PCL-R F1 and F2, and these divergent associations strengthened appreciably when F1 and F2 were entered simultaneously as predictors. These findings, along with support from a similar study in children (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999) suggest that global measures of psychopathy, such as the PCL-R, may obscure an underlying heterogeneity of antisocial pathology that encompasses both the callous, unemotional characteristics (i.e., low NEM) of the iconic psychopath as well as the affective lability (i.e., high NEM) and impaired impulse control pathognomonic of APD. *Personality Dimensions*

Much of the work on NEM and antisocial behavior has been done primarily in psychopathy, focusing on the PCL-R, even though APD is far more common as a disorder (Patrick, 2007). Moreover, much of this research focuses only on the extreme of the distribution represented by criminal psychopathy ignoring the dimensionality of psychopathy and APD-related traits (e.g., Edens, Marcus, Lilienfeld, & Poythress, 2006; Widiger, 1993). Consistent with a recent emphasis on dimensional representations of psychopathy and personality disorders generally (e.g., Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; Trull & Durrett, 2005), it may also be asked whether analogous associations extend across the normative range of personality differences. In this regard, the first aim of this study was to determine if the findings of Hicks and Patrick (2006) extend to analogous dimensional measures of APD versus psychopathy-related traits in a non-patient community sample.

One method to address the dimensional nature of personality from normal to abnormal is to describe personality disorder traits as constellations of basic dimensions of personality, particularly using the five factor model (FFM) of personality (Widiger & Costa, 1994), which can be measured by personality assessment instruments such as the NEO Personality Inventory Revised (NEO-PI-R; Costa & McCrae, 1995). To measure dimensions of personality disorders using the NEO-PI-R, researchers have used an expert consensus approach in which experts rated facets of the FFM to create prototypes of personality disorders (Lynam & Widiger, 2001; Miller, Lynam, Widiger, & Leukefeld, 2001). This prototype matching system effectively creates dimensional measures of personality disorders ascertained through a standard measure of personality that fit with FFM conceptions of each disorder, as well as assess these traits from the normal to abnormal range. In line with this method, we used FFM derived scores from the NEO-PI-R within a community sample to test associations between NEM and personality dimensions of APD and psychopathy.

Applicability to neural models of psychopathy and APD

Given the well-established link between various aspects of NEM and amygdala reactivity to social threat and distress (e.g., Carré, Fisher, Manuck, & Hariri, 2012; Etkin et al., 2004; Fakra et al., 2009), it is possible that the divergent relationships between psychopathy, APD, and NEM also exist at the neural level, particularly as evidenced by amygdala reactivity to social signals of threat such as angry facial expressions and social signals of distress such as fearful facial expressions. In fact, among psychopaths, response deficiencies in experimental paradigms tapping aspects of NEM such as fear and emotional learning (especially deficits in fear potentiated startle), are hypothesized to be the product of reduced reactivity of subcortical threat systems, especially the amygdala (Blair, 2006). Moreover, empirical studies show that psychopathy is related to reduced amygdala reactivity during paradigms tapping threat and affective processing (for a review, see Hyde, Shaw, & Hariri, 2013). Less work has examined amygdala reactivity in individuals with APD, but there is evidence for *greater* amygdala reactivity under similar stimulus conditions when comparing non-psychopathic antisocial samples to controls such as adults with intermittent explosive disorder (Coccaro, McCloskey, Fitzgerald, & Phan, 2007), youth with conduct problems but not callous traits (Herpertz et al., 2008; Viding, Sebastian, et al., 2012), male community volunteers high on trait aggression and high on trait anxiety (Carré et al., 2012), and college students high on social deviance dimensions of the Psychopathic Personality Inventory (PPI) (Gordon, Baird, & End, 2004).

Furthermore, recent studies that examined antisocial versus psychopathy traits demonstrated divergent correlations between antisocial and psychopathy traits with amygdala reactivity to masked fear faces in youth (e.g., Viding, Sebastian, et al., 2012) and in a college sample (Gordon et al., 2004). In this second study, Gordon and colleagues (2004) found that those scoring high on F1 of the PPI demonstrated *attenuated* amygdala reactivity to a range of emotional faces, whereas those scoring high on F2 displayed *increased* amygdala reactivity. Moreover, a recent study of adolescents used a theory of mind task to demonstrate divergent relationships with amygdala reactivity *within a suppression framework* between callous-unemotional traits (i.e., psychopathy) and conduct problems (i.e., antisocial behavior) (Sebastian et al., 2012). These studies suggest that, like NEM, amygdala reactivity to emotional faces, may be related in opposite directions: negatively to the callous detachment of the characteristic of psychopathy and positively the antisocial and impulsive behavior of APD (for evidence that this association may be specific to threat and distress i.e., fear and anger faces, see: Carré et al., 2012; Coccaro et al., 2007; Marsh & Blair, 2008; Viding, Sebastian, et al., 2012). Therefore, the second major aim of this study was to extend suppression work on NEM to neural function and test the hypothesis that amygdala reactivity to threat and distress (i.e., anger and fear faces) is related in divergent directions to psychopathy and APD traits, and that this relationship with amygdala reactivity explains (i.e. mediates) the divergent relationship between NEM and psychopathy and APD traits. That is, psychopathy is hypothesized to be related to lower NEM through relatively lesser amygdala reactivity, and APD is hypothesized to be related to higher NEM through relatively greater amygdala reactivity. Additionally, as previous studies have emphasized either fear or anger

faces as stimuli, we tested whether these divergent relationships were specific to amygdala reactivity to anger versus fear faces.

Finally, though the primary goal of this study was to extend suppression findings to FFM conceptualizations of *APD* and psychopathy, previous work in this area (e.g., Hicks & Patrick, 2006) has examined factors *within* psychopathy (i.e., F1, F2). Thus a final aim of the study was to examine the extent to which findings using FFM generated measures of APD and psychopathy would be paralleled by FFM generated measures of F1 and F2 psychopathy. However, as conceptualizations of these factors vary across instrument and measurement (i.e., the PCL-R conceptualization of F1 is quite different then the PPI conceptualization of F1: Hare & Neumann, 2008), we included two different measures of F1 and F2 – one based on the structure of the PCL-R and one based on the structure of the PPI. As PCL-R F1 and F2 have substantial overlap with the unique aspects to psychopathy and APD respectively, we expected results using PCL-R guided measures to be similar to those found with APD and psychopathy measures, particularly when overlap between F1 and F2 is partialled. However, as PPI F1 and F2 are generally found to be uncorrelated with each other (Marcus, Fulton, & Edens, 2013), we did not expect suppression effects with PPI F1 and F2. Moreover, as PPI F1 (fearless dominance) is thought to tap fear deficits more directly (Lilienfeld et al., 2012), we expected PPI F1 to be the more highly related to low NEM and low amygdala reactivity to threat and distress than PCL-R F1.

The Current Study

A central goal of the current study was to examine the differential association of APD and psychopathy traits with the outcomes of threat- and distress-related amygdala reactivity and NEM, with a secondary goal to examine how these findings might vary by different personality based measures of factors within psychopathy. To attain continuous measures of personality disorder traits, a previously validated profile matching count technique was used to create highly overlapping but distinct measures of psychopathy and APD from NEO-PI-R profiles (Lynam & Widiger, 2001; Miller, Bagby, Pilkonis, Reynolds, & Lynam, 2005; Miller et al., 2001), as well as FFM generated measures of F1 and F2 based on the PCL-R using the NEO-PI-R (Derefinko & Lynam, 2006; Widiger & Lynam, 1998) and the PPI using the Multidimensional Personality Questionnaire (Benning et al., 2005). Amygdala reactivity was measured using an fMRI task designed to robustly engage the amygdala through the presentation of socially salient signals of threat and distress (Fakra et al., 2009; Manuck, Brown, Forbes, & Hariri, 2007). Psychopathy (and F1) was hypothesized to be negatively related to amygdala reactivity and NEM, whereas APD (and F2) was expected to be positively related to both constructs. Moreover, we expected results to be stronger when controlling for correlated variance in APD and psychopathy. Furthermore, we hypothesized that differences in NEM between APD and psychopathy would be accounted for by differences in amygdala reactivity. Finally, we expected results to be similar when using F1 and F2 based on the PCL-R structure, and in a similar direction, but without suppression, using the uncorrelated F1 and F2 based on the PPI. The present study was conducted using data from a study of men and women in the community where a greater range of personality was possible and in which relationships between these traits and neural reactivity could be

examined dimensionally and in a sample much larger than related neuroimaging studies (e.g., Marsh et al., 2008; Veit et al., 2002).

Method

Participants

A total of 103 participants were recruited from the University of Pittsburgh Adult Health and Behavior (AHAB) Project, an archival database encompassing detailed measures of behavioral and biological traits among a community sample of 1379 non-patient, healthy adult volunteers (Fakra et al., 2009; Manuck et al., 2007). Participants were middle-aged adults (mean age=44.5 years; SD=6.8; range: 31-54 years) and 45% were male (for more details see Supplemental Methods and Fakra et al., 2009; Hyde, Manuck, & Hariri, 2011; Manuck et al., 2007). Participants reported their race as follows: 88% European-American, 7% African-American, and 4% other races (e.g., Asian-American, multi-racial).

Procedures

Amygdala reactivity paradigm—The experimental fMRI paradigm consisted of four blocks of a face-processing task interleaved with five blocks of a sensorimotor control task (Carré et al., 2012; Fakra et al., 2009; Manuck et al., 2007). During the face-processing task, subjects viewed a trio of faces (expressing either anger or fear) and selected one of two faces (bottom) identical to a target face (top). Each face processing block consisted of six images, balanced for gender and target affect (angry or fearful), all derived from a standard set of pictures of facial affect (Ekman & Friesen, 1976). During the sensorimotor control blocks, subjects viewed a trio of simple geometric shapes (circles, vertical and horizontal ellipses) and selected one of two shapes (bottom) identical to a target shape (top). Each sensorimotor control block consisted of six different shape trios. A variable interstimulus interval during face processing was used to allow for estimation of expression-specific neural activation.

Bold fMRI acquisition parameters—Each participant underwent scanning with a Siemens 3-T MAGNETOM Allegra (Siemens AG, Erlangen, Germany), which is characterized by increased T2* sensitivity and fast gradients (slew rate, 400 T/m/s), which minimize echospacing, thereby reducing echoplanar imaging geometric distortions and improving image quality. Blood oxygen level–dependent (BOLD) functional images were acquired with a gradient-echo echoplanar imaging sequence (TR/echo time=2000/25 milliseconds, FOV=20 cm, matrix=64_64), which covered 34 interleaved axial slices (3-mm slice thickness) aligned with the AC-PC plane and encompassing the entire cerebrum and most of the cerebellum.

Image processing and analysis—Whole-brain image analysis was completed using the general linear model of SPM2 (Wellcome Department of Imaging Neuroscience, London, England). Images for each participant were realigned to the first volume in the time series, spatially normalized into a standard stereotactic space (Montreal Neurological Institute template) using a 12-parameter affine model, and smoothed to minimize noise and residual difference in gyral anatomy with a gaussian filter set at 6 mm FWHM. Voxelwise signal intensities were ratio-normalized to the whole-brain global mean. These preprocessed data

sets were analyzed using second level random-effects models that accounted for both scan-to-scan and participant-to-participant variability to determine task specific regional responses.

Following preprocessing, linear contrasts employing canonical hemodynamic response functions were used to generate BOLD contrast estimates for general reactivity to facial expressions (i.e., all faces>shapes, angry face>shapes, fearful faces>shapes) for each individual. Individual contrast images (i.e., weighted sum of the beta images) from these three contrasts were then used in second-level random effects models that account for both scan-to-scan and participant-to-participant variability to determine mean condition-specific regional responses using one-sample t-tests. All analyses were thresholded at a voxel level of $p<0.05$, FDR corrected for multiple comparisons across the entire brain volume.

Regions of Interest—BOLD contrast estimates (i.e., mean signal) were extracted from clusters exhibiting a main effect of task using the signal above threshold within anatomically defined regions of interest (ROIs). Because of the structural and functional heterogeneity of the amygdala (Davis & Whalen, 2001; Whalen et al., 2001), we examined the ventral and dorsal amygdala independently to determine whether individual differences in personality map on to the amygdala's principal input and output regions, respectively using previously used ROI masks for the ventral, dorsal and whole amygdala (see Supplamental Materials and Hyde et al., 2011). This approach was motivated by previous imaging research demonstrating that individual difference factors map on to specific subregions of the amygdala (Etkin et al., 2004; Manuck et al., 2010).

Measures

NEO-PI-R—The NEO–PI–R (Costa & McCrae, 1992) is a self-report questionnaire developed to assess normal personality dimensions based on the FFM. It consists of 240 items, which are rated on a 5-point scale ranging from 0 (*strongly disagree*) to 4 (*strongly agree*) and provides a score for the five domains of adult personality and six "facet" scales that tap associated features or attributes of the corresponding dimension. Many studies using the NEO– PI–R have consistently shown good validity, reliability and psychometric properties (e.g., high internal consistency and long-term stability over three to six years across the five domains) (Costa & McCrae, 1992), including the present study (for the five broader domains α=.73-.87).

APD & Psychopathy Traits—FFM continuous APD and psychopathy scores were calculated using the expert consensus prototype matching approach of Lynam and Widiger (2001). Experts rated prototype scores of all 10 DSM-IV personality disorders (and psychopathy: Miller et al., 2001) across the 30 facets of the FFM on the NEO-PI-R. Thus for APD and psychopathy, experts generated a profile of facet-level scores for a prototypic individual with APD or psychopathy. These profiles can be then used to generate "scores" for relative APD and psychopathy traits in individuals by using a sum of scores on facets maximally descriptive of the disorder from the prototype profiles (Decuyper, De Clercq, De Bolle, & De Fruyt, 2009; Miller et al., 2005). These methods have been shown in multiple samples as valid continuous measures of personality disorder traits (Lynam & Widiger,

2001; Miller, 2012; Miller et al., 2005). See Table 1 for means and standard deviations of study measures and Supplemental Table 1 for a description of how these dimensions were formed.

Negative Emotionality—NEM scores were generated using the NEM scale from the abbreviated version of the Multidimensional Personality Questionnaire–Brief Form (MPQ-BF) (Manuck, Craig, Flory, Halder, & Ferrell, 2011; Patrick, Curtin, & Tellegen, 2002).

Measures of Psychopathy Factors—As noted above, we also tested alternate ways of assessing our dependent variables by creating F1 and F2 psychopathy scores. The first way was to create F1 and F2 scores based on the structure of the PCL-R using Widiger and Lynam's (1998) rational assignment of NEO-PI-R domains to create PCL-R guided F1 and F2 scores using Derefinko and Lynam's (2006) operationalization of this assignment at the *facet* level. Thus, NEO-PI-R facet scores were weighted and summed as described previously (Derefinko & Lynam, 2006, p. 265) to create F1 and F2 scores. The second approach we used was to create F1 and F2 scores based on a different conceptualization of psychopathy (i.e., the structure derived from the PPI) and measured via items from a different personality questionnaire (i.e., the MPQBF). Thus, we created scores of PPI F1 (fearless dominance) and PPI F2 (impulsive antisociality) by multiplying regression weights described by Benning and colleagues (2005) with z-scores on the appropriate MPQ scales (as measured by the MPQ-BF, see above) to create a measure of fearless dominance and a measure of impulsive antisociality from MPQ scale scores.

Statistical analysis

Primary dependent variables were the BOLD contrast estimates extracted bilaterally from the mean signal in clusters of activation in the whole, ventral, and dorsal amygdala ROIs. Associations of FFM-Psychopathy and FFM-APD scores with amygdala reactivity were examined in regressions using IBM SPSS statistics v.20. Consistent with past studies (e.g., Hyde et al., 2011) gender was controlled for in all regressions given its main effect on amygdala reactivity in this sample (see Table 1)[1]. In all regression analyses extracted amygdala reactivity values were regressed first on APD or psychopathy scores separately, and then onto APD and psychopathy scores simultaneously to assess for overlapping versus unique relationships between psychopathy and APD dimensions and amygdala reactivity. As APD and psychopathy trait scores demonstrated a high correlation (see Table 1), collinearity was a concern and thus tolerance was monitored with scores below 0.10 considered

[1]Note that we controlled for gender in this study because there are main effects of gender on amygdala reactivity in many past studies using this sample (e.g., Hyde et al., 2011), including the current study (see Table 1). We also re-ran all analyses without controlling for gender. The pattern of findings is the same throughout all of the regressions when controlling or not controlling for gender. However, several statistically significant effects became less significant in some regressions (i.e., p>.05), specifically those not controlling for gender and predicting reactivity to faces > shapes in the left amygdala (psychopathy B=−.006, SE=.005, β=−.24, *p*=.21; APD B=.008, SE=.005, β=.33, *p*=.09) and left dorsal amygdala (psychopathy B=−.009, SE=.006, β=−.28, *p*=.14; APD B=.011, SE=.006, β=.37, *p*=.06), as well as those predicting anger > shapes in the right amygdala (psychopathy B=−.002, SE=.001, β=−.23, *p*=.24; APD B=.002, SE=.001, β=.31, *p*=.11). In each of these cases the effect is still in the same direction and of similar magnitude (especially when considering the size of the β) but *p* values changed when not controlling for gender. Additionally, we have also conducted moderated multiple regression analyses with gender as a moderator of all effects. When examining if any possible gender differences are statistically significant, via moderated multiple regression, none of the interaction terms were statistically significant (i.e., all interaction term *p*'s > .10, most interaction term *p*'s > .30). Additionally, as interaction terms can add to collinearity concerns, we monitored measures of collinearity and found that the addition of these interaction terms caused unacceptable levels of collinearity (e.g., tolerance < .10).

problematic (Cohen, Cohen, West, & Aiken, 2003). We used bootstrapped corrected confidence intervals to test for statistically significant mutual suppression where applicable using the "indirect" macro for SPSS (Preacher & Hayes, 2008). We also provide "Sobel" tests to provide a measure of effect size of suppression effect and to be consistent with past studies in this area (Hicks & Patrick, 2006). Similar to mediation in which bootstrapped confidence intervals can be used to test the significance of change in a coefficient from IV to DV after the addition of a third variable, these same tests can be used to determine the statistical significance of the change in the coefficient due to suppression when a third suppression variable is added (MacKinnon, Krull, & Lockwood, 2000). Thus, in these analyses, a confidence interval not containing zero was indicative of statistically significant suppression through a significant change in the regression coefficient when adding a third variable. In the results below, *p* values are derived from bootstrapped bias corrected confidence intervals and z values are provided as effects sizes derived from Sobel tests (though note that Sobel tests are less powerful and only provided for a gross effect size measure).

Results

Descriptive Statistics and Correlations

Table 1 presents descriptive statistics and correlations among study variables. APD scores were positively related to NEM but psychopathy scores were not significantly related to NEM suggesting that, when taken alone, only APD is related in the hypothesized positive direction with NEM. Psychopathy and APD scores demonstrated small, non-significant positive correlations with amygdala reactivity across all contrasts, suggesting that, when taken alone, neither construct predicted amygdala reactivity.

Relationship with Negative Emotionality

To investigate the hypothesis that a major difference between APD and psychopathy is level of negative emotionality, two hierarchical regressions predicting NEM were conducted (all regressions controlled for gender): In the first regression, psychopathy scores were entered first, followed by APD scores, in the second regression APD scores were entered first followed by psychopathy scores. In independent regressions, psychopathy traits were not significantly related to NEM (*B*= −.008 *SE*=.048, *ns)*, whereas APD traits were positively associated with NEM (*B*=.124, *SE*=.042, p<.01). However, when both constructs were entered in the same regression, they each demonstrated significant relationships with NEM and in opposite directions, indicative of mutual suppression (psychopathy: *B*= −.524, *SE*=. 078, p<.001; Sobel z=5.4, 95% CI does not contain 0, and APD: *B*=.542, SE=.072, p<.001, Sobel z= −5.7, 95% CI does not contain 0). Thus, in extending past findings to APD and psychopathy measures (Hicks & Patrick, 2006), high APD traits were related to greater NEM, whereas high psychopathy traits were related to lesser NEM, but only when the overlap of these two constructs was partialled out.

Mutual Suppression of Amygdala Reactivity

Table 2 presents the results of hierarchical regressions predicting amygdala reactivity while controlling for gender. Regressions were run in the same manner as above, with amygdala

reactivity as the outcome instead of NEM. Across analyses, a similar trend emerged: APD traits alone were positively, though non-significantly related to amygdala reactivity. When accounting for psychopathy traits, however, this relationship increased and became significant in several ROIs, suggesting that partialling out overlap with psychopathy strengthened the positive association with amygdala reactivity. Psychopathy traits demonstrated a similar effect: Generally, psychopathy traits showed non-significant positive associations with amygdala reactivity. However, when APD scores were accounted for, the association between psychopathy traits and amygdala reactivity was *negative* and, in many ROIs, significant. Moreover, when examining the significance of the suppression effect, in almost all models in which psychopathy or APD became significant predictors of amygdala reactivity, the suppression effect was statistically significant (see Table 2).

While this trend was relatively consistent across ROIs and contrasts, psychopathy and APD traits demonstrated statistically significant associations in a suppression framework within the following specific amygdala regions of interest and contrasts: left amygdala (all faces), left dorsal amygdala (all faces), right amygdala (anger faces), left amygdala (anger faces), left dorsal amygdala (anger faces), and right ventral amygdala (anger faces). Results were strongest in the left amygdala, in dorsal regions, and to the Anger>Shapes contrast. None of the amygdala ROIs for the Fear>Shapes contrast were significantly predicted by APD and psychopathy traits (see Supplemental Table 2). Thus, the suppression results appeared to be primarily driven by reactivity to anger and to be particularly strong in the dorsal amygdala[2].

Amygdala Reactivity as Explanation for NEM Suppression Results

We examined whether suppression effects on NEM might be accounted for by parallel effects on amygdala reactivity (i.e., to determine whether divergent relationships with amygdala reactivity account for the divergent relationships between NEM, psychopathy, and APD). To test this question, we explored two mediation models in which APD and psychopathy traits predicted NEM and examined whether this path was mediated by amygdala reactivity. To test this mediating relationship we selected the two ROIs (left amygdala and left dorsal amygdala reactivity to All Faces > Shapes) that showed significant suppression relationships with APD and psychopathy traits *and* were related to NEM (β=.20, p<.05 and β=.22, p<.02, respectively). In both models, the addition of amygdala reactivity (left whole and left dorsal amygdala reactivity) to the regression, did not affect the significance of the relationship of psychopathy with NEM, nor the relationship of APD with NEM, nor was there any statistical evidence of mediation effects (all z statistics < .65, all bootstrapped 95% confidence intervals contained 0). Moreover, results were similarly non-significant if NEM and amygdala reactivity were conceptually reversed in the model (e.g., NEM as mediating the relationship between APD and psychopathy and amygdala reactivity). Thus the extent to which NEM and amygdala reactivity have similarly divergent relationships with psychopathy and APD traits does not appear to be due to overlapping

[2]Given the high correlation between APD and psychopathy scores, collinearity was monitored through tolerance values in each regression and the lowest value of tolerance was still above “problematic” levels of 0.10 (tolerance within all regressions in this study > 0.24). Moreover, in all regressions, the standard errors of the predictor variables did not change dramatically as would be expected in the case of problematic collinearity.

variance that the two constructs share (see Venables, Hall, & Patrick, 2013 for evidence that boldness may be a better construct for separating APD and psychopathy).

Exploratory analyses with additional conceptualizations of antisocial constructs

NEO derived PCL-R factors—Though our primary focus was on extending mutual suppression work to NEO-based measures of APD and psychopathy, there are other compelling ways to derive similar constructs that could help clarify the extent to which our findings are generalizable to measures of factors *within* psychopathy that are less highly correlated. The most similar approach is to use Derefinko and Lynam's (2006) approach to use NEO-PI-R facets to create PCL-R guided F1 and F2 scores. Within this sample, these F1 and F2 dimensions were correlated at lower levels (r=.42, $p<.001$; Supplemental Table 3). Using the same approach as above to test for suppression relationships with NEM, F1 did *not* predict NEM either alone or in a suppression regression (p's>.23), whereas F2 positively predicted NEM in a regression alone ($B=.22$, $SE=.05$, $p<.001$) and when controlling for F1 ($B=.23$, $SE=.07$, $p<.001$), with no suppression effects.

When examining relationships with amygdala reactivity using these F1 and F2 scores, we found few instances of significant suppression (see Supplemental Table 4; only 2 marginally significant suppression relationships with F2 scores predicting right and left dorsal amygdala reactivity to anger faces). Generally F1 and F2 were unrelated to amygdala reactivity alone or when controlling for each other. Unexpectedly, right dorsal amygdala reactivity to fear was *positively* and significantly related to F1. In sum, F2 predicted NEM without suppression and, in contrast to results with APD and psychopathy dimensions, neither PCL-R based factor predicted amygdala reactivity, even when partialling their overlap.

MPQ derived PPI factors—We also used a second and more divergent approach to examine F1 and F2 scores by using dimensions derived from another instrument and another conceptualization of psychopathy to determine the extent to which our findings vary by instrument and conceptualization of psychopathy factors. In this case, we used MPQ-derived factors based on the structure of the PPI (Benning et al., 2005). As the PPI contains factors that are generally orthogonal (Marcus et al., 2013) and in this sample the factors were correlated only very modestly (r=.17, $p<.10$), we did not expect mutual suppression. However, as F1 (fearless dominance) is thought to assess fear deficits more directly (see Lilienfeld et al., 2012) and overlaps less with PCL F1 scores, we tested these factors to determine if PPI F1 and F2 would yield a similar or different pattern of results in predicting NEM and amygdala reactivity (Marcus et al., 2013; Patrick, 2007).

In relationship to NEM, we found that PPI F1 (marginally) *negatively* predicted NEM alone (B=−3.14, SE=1.8, $p<.10$) and significantly when controlling for F2 (B=−5.3, SE=1.3, $p<.001$), and surprisingly (because these two factors are not highly correlated), demonstrated a significant suppression effect (z=1.78, p<.05). PPI F2 did *positively* predict NEM alone (B=11.9, SE=1.5, $p<.001$) and when controlling for F1 (B=12.8, SE=1.4, $p<.001$) demonstrating weak suppression effects (z=1.59, p<.05). When examining amygdala reactivity as the outcome, we found that PPI F2 was robustly and *positively* correlated with amygdala reactivity to most of the contrasts and across most of the amygdala subregions,

with no evidence of mutual suppression (see Supplemental Table 5). Interestingly, F1 was not correlated with amygdala reactivity alone or when overlapping variance was partialled out. Overall, PPI factors replicated suppression effects found in regards to NEM and showed a robust positive relationship between F2 and amygdala reactivity to both angry and fearful faces, but no evidence of F1 and amygdala associations or suppression effects.

Discussion

In summary, we replicated and extended the findings of Hicks and Patrick (2006) by showing that dimensional variability in trait measures of APD and psychopathy in a community sample demonstrated suppression effects in their relationship with NEM: APD was positively and psychopathy was negatively correlated with NEM, but only when the shared variance of the two constructs was partialled (which was similarly replicated using PPI psychopathy factors). Moreover, consistent with a study in youth using a different neuroimaging task (Sebastian et al., 2012), we found that this same relationship also existed with amygdala reactivity: APD dimensions were positively and psychopathy dimensions were negatively correlated with threat-related amygdala reactivity, particularly left dorsal amygdala reactivity to anger, when controlling for the overlap of APD and psychopathy. Though NEM and amygdala reactivity had similar relationships with psychopathy and APD traits, and have been correlated in many studies including our current work, amygdala reactivity did not mediate the relationship between NEM and APD or psychopathy. Finally, in exploring different measurements of similar constructs, we found that NEO derived PCL-R F2 and MPQ derived PPI F2 both positively predicted NEM and PPI F2 positively predicted amygdala reactivity. However, results across PCL-R F1 and F2, as well as PPI F1, and NEO based psychopathy scores were more inconsistent. PPI measures of F1 and F2 did replicate divergent relationships with NEM.

Overall, these findings support the notion that, even to the extent that APD and psychopathy may be highly overlapping constructs, two components that may distinguish these dimensions are NEM and amygdala reactivity. Thus, NEM and amygdala reactivity may be key constructs of interest in research that seeks to understand etiologic differences between APD and psychopathy (see also Venables et al., 2013 which suggests that boldness, which is both broader and narrower than NEM may be a more ideal construct that differentiates APD from psychopathy). Although some research has examined differential associations between factors of psychopathy (F1/F2) and NEM or amygdala reactivity, evaluations of these relations with APD as an outcome are rare and thus important given the higher prevalence of APD. Our findings add to growing research in this area and highlight the importance of examining these constructs (APD, psychopathy) separately but concurrently in the same model. Specifically, the current findings suggest that NEM and amygdala reactivity may not show zero-order relations with a unidimensional measure of psychopathy or APD when considered by themselves within the normal range of functioning (where these dimensions may be highly overlapping), but only when their overlap is considered (or in the case of psychopathy when F1 and F2 are separated).

Comparisons to past work on amygdala reactivity in antisocial populations

Though the NEM findings are in line with previous research and extend those findings to a community sample, the novel relationship between amygdala reactivity, APD and psychopathy dimensions bears further consideration. Certainly the negative association between psychopathy and amygdala reactivity is consistent with several other studies in youth and adults (Hyde et al., 2013). However, these studies generally focused on total psychopathy scores or groups of youth high on callous-unemotional traits and conduct problems and did not examine suppression relationships. Thus it is unclear why no significant zero-order (non-suppression) relationships between psychopathy dimensions and amygdala reactivity emerged in this study (though this simliar effect was seen in Sebastian et al., 2012 and PPI F2 in this study showed robust zero-order correlations with amygdala reactivity). It could be that this relationship is much weaker dimensionally in a healthy sample. Without partialling out APD dimensions, the current psychopathy measure in healthy adults may be capturing mild antisociality (as it contains both F1 and F2) rather than anything specific to psychopathy (as evidenced by high correlations between the APD and psychopathy measures). Therefore, it was not until we partialled out the overlapping variance of APD from the psychopathy dimension that we saw more of a “classic” relationship between the unique F1 components of psychopathy and amygdala reactivity. Certainly there is extreme divergence across many traits between healthy adults and criminal psychopaths, which underscores the utility of testing these relationships across the continuum of functioning, particularly when exploring biological correlates which are likely to have dimensional relationships with behavior (Ofrat & Krueger, 2012; Plomin, Haworth, & Davis, 2009).

The current results also appear to be consistent with two recent neuroimaging studies emphasizing that APD and psychopathy dimensions have divergent relationships with amygdala reactivity. In the first study of a different community sample, we found that, while controlling for the overlap of four psychopathy facets, amygdala reactivity to fear was *negatively* associated with the interpersonal facet of psychopathy (part of F1), whereas amygdala reactivity to anger was *positively* associated with the lifestyle facet of psychopathy (part of F2) (Carre, Hyde, Neumann, Viding, & Hariri, 2012). In a second recent study of youth, similar suppressor effects in amygdala reactivity emerged using dimensions of callous-unemotional traits and conduct problems (Sebastian et al., 2012).

Locating results within subregions of the amygdala

Consistent with past studies examining neural correlates of both anxiety and anger traits in the current sample (Carré et al., 2012; Hyde et al., 2011), the dorsal amygdala region that encompasses the central nucleus, sublenticular extended amygdala, and nucleus baysalis of Meynert, was most strongly related to APD and psychopathy in a suppression framework. These components of our dorsal amygdala ROI collectively represent major output pathways through which the amygdala can drive behavioral and physiologic arousal (Davis, Johnstone, Mazzulla, Oler, & Whalen, 2010). Previous work on psychopathy has not examined subregions of the amygdala, though one recent review has emphasized the importance of this approach (Moul, Killcross, & Dadds, 2012). The current findings

highlight the ability to examine subregions of the amygdala and the need for more attention to the heterogeneity of the amygdala in future neuroimaging studies of psychopathy.

Specificity of amygdala reactivity to individual face types

In the current study we found stronger relationships between variables when examining amygdala reactivity to *angry* rather than fearful facial expressions, whereas many past studies have specifically linked psychopathy to amygdala hypo-reactivity to *fearful* faces (e.g., Jones, Laurens, Herba, Gareth, & Viding, 2009; Viding, Fontaine, & McCrory, 2012), with one study showing this effect was specific to fear and not anger (Marsh et al., 2008). However, these studies were done in youth, focused only on callousness plus antisocial behavior, and used neutral faces as a contrast (rather than the simple somatosensory control stimuli used in the present study); thus, it is hard to compare these results directly (though see Marsh & Blair, 2008). In contrast, previous studies on adults with impulsive aggression (Coccaro et al., 2007), have highlighted the importance of the response of the amygdala to anger rather than fear (see also Carré et al., 2012). More research is needed to examine theories (e.g., Blair, 2007) that emphasize the unique importance of fearful facial expressions (versus anger) in eliciting individual differences in amygdala reactivity that are uniquely associated with psychopathy and if the link between amygdala reactivity and various facial expressions will differ in relative strength depending on sample characteristics (e.g., high on APD versus psychopathy traits). Moreover, it should be noted that there were no main effects of a contrast comparing amygdala reactivity to angry versus fearful facial expressions, thus we cannot directly test the proposition that our results were highly specific to angry facial expressions.

Additionally, it is unclear why amygdala reactivity failed to mediate the mutual suppression relationship of psychopathy and APD to NEM. Though amygdala reactivity and NEM were correlated in this study, it is likely that amygdala reactivity is also correlated with other personality dimensions that may also differ between APD and psychopathy (e.g., components of extraversion; see Supplemental Table 1). Moreover, NEM, though related to amygdala reactivity, is likely influenced by a complex network of neural regions, which is not wholly captured by amygdala reactivity. Similarly, it should be noted that NEM is a complex factor containing multiple negative emotions (e.g., anger, sadness) and psychopathy itself is not associated with reductions in all negative emotions (e.g., anger) (see Carré et al., 2012 for findings seperating some of these emotions apart in this sample). Thus our lack of mediation results may be due to conflating multiple negative emotions into one measure of NEM.

Comparison among measures of psychopathy factors

Supplemental analyses examining specific psychopathy subfactors from different measures, yielded interesting and complex results. The most consistent and highly correlated factor across all measures was of general antisociality (e.g., APD, PCL-R-based F2, PPI-based F2). All three of these measures tapping antisociality demonstrated significant positive relationships with NEM and both the APD and PPI F2 measures yielded positive relationships with amygdala reactivity to threat (and to some extent distress). However, findings with psychopathy dimensions and PCL-R and PPI dimensions of F1 were more

divergent, likely based on the differing measurement and conceptualization of F1 and the "unique" components of psychopathy (e.g., PCL-R F1 is conceptually different than PPI fearless dominance and these factors were virtually uncorrelated in this study). Thus it may be safest to conclude that our current findings are most robust in linking amygdala reactivity and NEM *positively* to antisociality across multiple measures whereas any links between F1/psychopathy are more dependent on the measurement instrument employed. Finally, these results highlight that current NEO derived measures of psychopathy and APD share much of their variance (i.e., these two measures were very highly correlated) and tap a similar construct (and thus may not be an ideal measure of what they each purport to assess). Furthermore, their overlap must be partialled to allow for unique relationships with criterion variables to emerge. These caveats should be considered when using the NEO-PI-R to assess psychopathy and APD.

Limitations

Though this study had many strengths including the use of a well-established task for eliciting amygdala reactivity, the ability to separate differential contributions of specific threat-versus distress-related facial expressions and amygdala subregions, a relatively large sample size for a neuroimaging study, and the ability to test dimensional relationships in traits expressed in a community sample, there are some limitations worth noting. First, this study examined associations in a healthy community sample that is unlikely to contain any individuals meeting criteria for clinically diagnosed APD or psychopathy. Thus, this investigation examines how these dimensions operate within healthy individuals and should not be generalized to psychiatric or criminal populations. Moreover, it should be noted that there were near-zero correlations between gender and measures of APD and F2, which run counter to a large body of literature demonstrating higher levels of these dimensions in males. These near-zero correlations may reflect unique aspects of this sample or the ways in which these measures were generated (though this relationship was consistent across all 3 measures). Second, as noted above, we did not use neutral faces within our neuroimaging paradigm and thus cannot directly compare our results to studies directly contrasting angry and fearful expressions with neutral expressions. Third, we used dimensional measures of APD and psychopathy that were derived from the NEO-PI-R and we did not have any "traditional" measures of psychopathy. Our findings did vary when based on other personality derived measures and our results likely would differ had we used diagnostic criteria, self-report, or interview-based measures (e.g., the SCID-II, PPI, PCLR). Moreover, this FFM method of generating APD and psychopathy traits resulted in highly correlated variables. Though we monitored closely for problems of collinearity, and have presented results from other, less correlated, measures, it is important to note that suppression effects are most likely to occur with highly correlated predictors. Moreover, as noted above, given their high overlap and lack of zero-order correlations with NEM and amygdala reactivity, the APD and psychopathy measures appear to have difficulty separating the two constructs they purport to measure (i.e., APD vs. psychopathy which should not be as highly correlated). Additionally these measures contain some problematic features including the inclusion of anxiety into the APD score (which is not a criterion for clinically assessed APD), and the fact that psychopathy includes much of the diagnostic criteria for APD (mostly through psychopathy's F2 with some F1 aspects). Fourth, it is important to note that

this sample was of middle aged adults and thus differs developmentally from many studies that have focused on adolescents or young adults/college students. Overall, these results should be interpreted within the larger literature on APD, psychopathy, NEM and amygdala reactivity across development in both clinical and normative populations.

Conclusions and Implications

The present findings suggest that APD and psychopathy traits in a community sample differ critically in their levels of NEM and amygdala reactivity, particularly when the overlap between APD and psychopathy are taken into account. Thus, accounting for the shared variance between the constructs helps to separate etiologic factors that are unique to these two overlapping constructs (e.g., levels of NEM, amygdala reactivity). The present findings help to replicate seminal work by Hicks and Patrick (2006) and to extend the findings to APD versus psychopathy, using dimensional measures of these constructs in a healthy sample. Moreover, the present findings expand this framework to the neural correlates of these antisocial dimensions and emphasize the differences between these two constructs at the personality and neural level. Finally, by comparing various measures of psychopathy factors, our study highlights concurrence across measures of antisociality in predicting higher NEM and higher amygdala reactivity. In the long term, in helping to better understand the differences in etiology between these disorders, we hope that this basic research will eventually usefully inform larger efforts for early prevention strategies and personalized interventions based on differences in early emerging temperaments, such as NEM (e.g., Dadds & Rhodes, 2008).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Bivariate correlations

	Mean (*SD*)	1	2	3	4	5	6
1. Gender	.56						
2. Age	44.6 (6)	−.09					
3. APD	225 (28)	.03	−.15				
4. Psychopathy	285 (26)	−.14	−.05	.86***			
5. Right amygdala reactivity	.88 (.6)	−.20*	−.12	.12	.07		
6. Left amygdala reactivity	.88 (.7)	−.21*	−.13	.12	.04	.76***	
7. Negative Emotionality	24.6 (12.5)	.11	−.25*	.28**	−.03	.07	.17

Note: SD=standard deviation. Gender 0=male, 1=female. APD=antisocial personality disorder traits. Right and Left amygdala reactivity refer to extracted values based on the main effects of the All Faces>Shapes contrast

* $p < .05$.

** $p < .01$.

*** $p < .001$

Table 2

Hierarchical regressions of APD and Psychopathy dimensions predicting amygdala reactivity

		All Faces>Shapes Contrast					
		Entire amygdala		Dorsal		Ventral	
		Right	Left	Right	Left	Right	Left
Psychopathy	*B* alone (*SE*) Standardized β	.001(.002) .05	.000(.003) .01	.003(.003) .09	.000(.003) .007	−.001(.002) −.03	.001(.002) 04
	B with APD (*SE*) Standardized β	−.006(.005) −.26	−.011(005) −.41*	−.004(.006) −.12	−.014(.006) −.44*	−.006(.005) −.27	−.001(.004) −.03
	Suppression effect (z)	1.97	2.35*	1.34	2.46*	1.41	.53
APD	*B* alone (*SE*) Standardized β	.003(.002) .12	.003(.002) .13	.004(.003) .14	.004(.003) .13	.001(.002) .04	.001(.002) .06
	B with psychopathy (*SE*) Standardized β	.007(.004) .35+	.011(.005) .48*	.007(.006) .24	.015(.006) .51*	.006(.004) .28	.002(.004) .09
	Suppression effect (z)	−1.37	−2.05*	−.62	−2.24*	−1.4	−.19
		Anger Faces>Shapes Contrast					
Psychopathy	*B* alone (*SE*) Standardized β	.000(.001) .01	.000(.001) −.01	.000(.001) .04	.000(.001) −.04	.000(.001) −.003	.001(.001) .09
	B with APD (*SE*) Standardized β	−.003(.001) −.38+	−.002(.001) −.38+	−.002(.002) −.25	−.004(.002) −.53**	−.003(.001) −.41*	.000(.001) −.02
	Suppression effect (z)	2.80**	2.50**	1.80+	2.93**	2.00*	.70
APD	*B* alone (*SE*) Standardized β	.001(.001) .12	.001(.001) .10	.001(.001) .12	.001(.001) .10	.001(.001) .11	.001(.001) 10
	B with psychopathy (*SE*) Standardized β	.003(.001) .45*	.003(.001) .43*	.003(.002) .34+	.004(.002) .56**	.003(.001) .45*	.001(.001) .12
	Suppression effect (z)	−2.00*	−2.00*	−1.36	−2.69**	−1.77+	−.11

N=103. Unstandardized regression coefficients reported (*B*) with standardized coefficients (β) reported in the line below. Gender was controlled for the in the first step of all analyses. "Sobel" tests were used to examine the effect size of mutual suppression effects and significance (p<.05) is denoted from bootstrap bias corrected confidence intervals.

+ $p < .10$.

* $p < .05$.

** $p < .01$