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Personality Disorders and the Persistence of Anxiety Disorders: Evidence of a Time-of-Measurement Effect in NESARC

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Abstract

Recent studies using data from the National Epidemiological Survey of Alcohol and Related Conditions (NESARC) have found that some personality disorders (PDs) increase the persistence of several Axis I disorders. However, these effects are potentially confounded with the data collection wave in which PDs were assessed. Our aim was to extend published analyses to the case of anxiety disorders and to determine the robustness of the associations to analyses examining time-of-measurement effects. Persistence of anxiety disorders was defined either as follow-up diagnosis among participants diagnosed at baseline ("prediction") or baseline diagnosis among participants diagnosed at follow-up ("post-diction"). Results revealed a robust pattern of higher odds ratios for post-diction among PDs assessed at baseline, and lower odds ratios for post-diction among PDs assessed at follow-up, suggesting a time of measurement artifact. Although only 4% of associations were robust to both predictive and post-dictive analyses, these were consistent with previous research.

Keywords

personality disorders; anxiety disorders; NESARC

1. Introduction

Data from the National Epidemiological Survey of Alcohol and Related Conditions (NESARC) have recently been used to investigate the association of personality disorder (PD) diagnoses with the persistence of Axis I disorders. The NESARC is a study of a representative US sample assessed at baseline during 2001–2002 (Grant, Moore, & Kaplan, 2003) and followed-up during 2004–2005 (Grant & Kaplan, 2005). The follow-up allows for

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the empirical examination of a number of critical issues, including factors associated with the persistence of psychiatric disorders. One of the questions that can be uniquely addressed through NESARC is how the presence of a PD can affect persistence of Axis I disorders. Although PDs are shown to be associated with Axis I disorders (Bienvenu & Stein, 2003; Brooks, Baltazar, & Munjack, 1989; Grant et al., 2008; Lenzenweger, Lane, Loranger, & Kessler, 2007; Skodol, Oldham, & Gallaher, 1999; Zanarini et al., 1998), the extent to which PDs predict persistence of Axis I is less clear. Three recent articles using NESARC data show that Borderline PD is consistently associated with the persistence of alcohol dependence (AD), nicotine dependence (ND), and cannabis use disorder (CUD; Hasin et al, 2011); drug use disorder (DUD; Fenton et al., 2011); and major depressive disorder (MD; Skodol et al., 2011), across models that adjust for a number of covariates, including all other PDs.

Although not yet investigated in the NESARC dataset, there are several reasons to expect a similar association of Borderline PD with the persistence of anxiety disorders. Analyses from NESARC and other studies show that Borderline PD is strongly associated with anxiety disorders, with effect sizes comparable to those of associations with MD and DUD (Grant et al., 2008; Comtois, Cowley, Dunner, & Roy-Byrne, 1999; Zimmerman & Mattia, 1999). Confirmatory factor analyses indicate that Borderline PD is associated with the distress sub-factor of the internalizing dimension –including post-traumatic stress disorder (PTSD) and generalized anxiety disorder (GAD) as indicators– but not with the fear sub-factor –including panic disorder with agoraphobia (PDw/A), social phobia (SOP), and specific phobia (SP) as indicators- (Eaton et al., 2011). However, a 10-year follow-up study comparing participants diagnosed with Borderline PD with a group diagnosed with PDs other than Borderline found that the Borderline group showed significantly higher rates of persistence of panic disorder (Silverman, Frankenburg, Reich, Fitzmaurice, & Zanarini, 2012).

In addition, the persistence of anxiety disorders might be associated with PDs other than Borderline PD. In particular, PDs that are a part of cluster C (i.e., Avoidant, Dependent, and Obsessive-Compulsive PDs) might exhibit stronger associations with anxiety-disorder persistence than Borderline PD, given their similar phenomenology (Friborg, Martinussen, Kaiser, Øvergård, & Rosenvinge, 2013). Evidence for an effect of cluster C PDs on the course of anxiety disorders has been found in the few prospective, naturalistic studies that have examined this issue. Results from the Harvard/Brown Anxiety Research Program indicated that both Avoidant and Dependent PD predicted lower rates of remission from GAD during a 5-year follow-up (Massion et al., 2002). In addition, the presence of Avoidant PD predicted lower rates of remission from SOP. Unfortunately, the low rates of some PDs in the sample (i.e., Schizoid, Schizotypal, Narcissistic, and Borderline) precluded analyses of their association with the course of anxiety disorders (Massion et al., 2002). A more recent study analyzing data from the Collaborative Longitudinal Personality Disorders Study (CLPS) replicated the finding of an association between Avoidant PD and lower likelihood of remission from SOP at 7-year follow-up, an expected finding given the overlap in the diagnostic criteria for these disorders (Ansell et al., 2011). Also, Schizotypal PD predicted lower rates of remission from PTSD. Additionally, this study examined the association of PDs with relapse of anxiety disorders. Schizotypal PD was associated with higher rates of relapse from SOP, Obsessive-Compulsive PD was associated with higher rates of relapse from GAD, and Borderline PD was associated with higher rates of relapse from obsessivecompulsive disorder (OCD). In addition, Avoidant PD and Obsessive-Compulsive PD were associated with lower rates of relapse from PDw/A and PTSD, respectively (Ansell et al., 2011). Thus, although in this study the course of anxiety disorders was primarily associated with cluster C PDs, associations with PDs from other clusters were also found. However, assessment of PDs in this study was only limited to Schizotypal, Borderline, Obsessive-

Compulsive, and Avoidant PDs. Moreover, findings from these two studies should be interpreted taking into account the fact that they correspond to treatment-seeking samples, which are known to have ascertainment biases associated with comorbidity (Berkson, 1946). To our knowledge, there is no published study examining the association of all ten PDs with the course of anxiety disorders in the general population.

Although NESARC data afford the opportunity to investigate the hitherto unexplored association of all PDs and anxiety-disorder persistence, there is a design limitation that can potentially compromise the investigation. Specifically, the ten DSM-IV (American Psychiatric Association, 1994) PDs were not all assessed at the same time, and only Adult Antisocial Behavior (AAB; part of the criteria for diagnosing Antisocial PD) was assessed at both waves. To reduce participant burden, Borderline, Narcissistic, and Schizotypal PDs were assessed only at Wave 2, whereas the remaining PDs were assessed only at Wave 1. As a result, any established associations between PDs and the persistence (i.e., disorder present at Wave 2 among those diagnosed at Wave 1) of Axis I disorders, could potentially be compromised by the artifact introduced by wave of assessment. In particular, PDs assessed at Wave 2 might be more likely to show significant associations with persistence of Axis I disorders for the simple reason that both are based on Wave 2 measures and therefore could share common measurement error (e.g., reporting bias) specific to that measurement occasion. Consistent with this, in the prior published papers the strongest predictors of Axis I persistence were PDs measured at Wave 2 (including Antisocial PD which was based on measurement at both waves), raising the possibility that a method artifact associated with time of measurement could be contributing to these findings.

Empirically rigorous methods can aid us in establishing the degree to which findings regarding the association of PDs and disorder persistence are due to a time-of-assessment effect. One way of evaluating this is to use a latent variable approach to model method factors that specifically assesses variance associated with a given time of measurement (as in multi-trait, multi-method factor models; Eid, Lischetzke, & Nussbeck, 2006; see also, Trull, Vergés, Wood, & Sher, in press). However, the design of NESARC confounds timeof assessment with content factors to such a degree that his approach yields solutions that fail to clearly disti nguish constructand method (see Trull, Vergés, Wood, Jahng, & Sher, 2012, for a discussion of this issue). An alternative strategy is to compare the traditional approach used to define persistence prospectively (i.e., the likelihood of a later diagnosis given a diagnosis at baseline) to one where persistence is defined retrospectively. Specifically, persistence may be defined as he likelihood of diagnosis at baseline given a diagnosis at a later time of measurement. The logic here is that if there is no "time-of-measurement effect", findings using predictive (i.e., prospective) or post-dictive (i.e., retrospective) models should be similar or at least not dramatically different¹. However, if, for instance, B orderline PD (measured at Wave2) is more strongly associated with the persistence of anxiety disorders measured prospectively than retrospectively (i.e., essentially reflecting a cross-sectional relationship at Wave 2), but Dependent PD (measured at Wave 1) is more associated with the persistence of anxiety disorders measured retrospectively than prospectively (i.e., essentially reflecting a cross-sectional relationship at Wave 1), these results would strongly suggest a time-of-measurement effect. Moreover, if various fixed covariates that are unlikely to be associated with much measurement error (e.g., sex) show similar patterns of predictive and post-dictive effects, this would indicate that differential

¹We note that to the extent that earlier onset disorders are likely to be more severe, then persistence defined prospectively might be more associated with comorbidity than persistence defined retrospectively. However, this is only a possibility and, even if true, would not suggest a systematic relation between when a covariate was assessed and the relative strength and/or direction of the predictive versus post-dictive relation.

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associations between covariates and persistence operationalized either predictively or postdictively are not determined by the choice of operationalization.

Again, the logic of our approach does not assume that estimates of persistence derived prospectively or retrospectively should be identical. For example, if there tends to be a disorder with later onset, many forms of that disorder may not be diagnosed until a later wave, but such conditions could nevertheless still be the beginning of a chronic condition that would be considered non-persistent using the post-dictive approach. However, this should not differentially affect associations with PDs based on their time of measurement. The issue is the extent to which the magnitude and/or direction of the effect appears to be a joint function of when the PD was assessed and whether the analysis is predictive or post-dictive.

It is possible that some of the PDs assessed only at Wave 2 show a valid effect on persistence measured prospectively and that it is only by chance that the PD was assessed at Wave 2 rather than Wave 1. However, associations between a Wave 2 PD and predictively defined persistence represent, at best, a cross-sectional finding, not a true lagged one, with all the attendant difficulties of interpretation. Further problems of interpretation arise if there is independent evidence to suggest strong time-of-measurement effects, as, for example, with observations of higher correlations among PDs within than between waves, in which case common method variance is indicated. Additionally, as just discussed, the influence of PD type compared to time of measurement on anxiety-disorder persistence can be inferred by the pattern of effects for different PDs measured at each wave. If type of PD exerts more influence than time of measurement on Axis I disorder persistence, then there should be something resembling a random distribution of PD effects between those PDs measured at each Wave. If, on the other hand, time of measurement of the PD exerts more influence than type of PD on Axis I disorder persistence, then a consistent pattern of PD effects on Axis I disorder persistence should be evident for each measurement point.

In sum, the goal of the current paper was to determine the relationship between specific PDs and the persistence of anxiety disorders. Moreover, we directly investigated to what extent the associations found were attributable to the wave in which PDs were assessed. We performed a post-diction of Wave 1 anxiety disorders among participants diagnosed at Wave 2 as well as a more traditional forward prediction in order to compare the two approaches.

2. Method

2.1. Sample

The NESARC is representative of civilians 18 years and older of the US population. The survey oversampled Blacks and Hispanics and young adults between 18 and 24 years. An initial wave of interviews was conducted during 2001–2002 and includes 43,093 respondents (Grant et al., 2003). A follow-up second wave of interviews was performed during 2004–2005 and contains 34,653 of those previously interviewed (Grant & Kaplan, 2005).

2.2. Measures

2.2.1. Anxiety Disorders—The NESARC used the Alcohol Use Disorders and Associated Disabilities Interview Schedule-DSM-IV version (AUDADIS-IV; Grant, Dawson, & Hasin, 2001) to assess DSM-IV Panic Disorder with and without Agoraphobia (PDw/A and PDw/oA), SP, SOP, and GAD. Agoraphobia without a history of Panic Disorder was also assessed, but there were no cases of persistence with this diagnosis. Assessment for SOP involved fear of at least 1 of 14 social or performance situations (Grant, Hasin, Blanco, et al., 2005), whereas assessment for SP involved fear to at least 1 of 12

specific objects or situations (Stinson et al., 2007). Further details about the assessment of these disorders can be found elsewhere (Grant, Hasin, Blanco et al., 2005; Grant, Hasin, Stinson et al., 2005; Grant et al., 2006; Stinson et al., 2007). A composite variable reflecting the presence of any anxiety disorder was also formed. Current analyses used past-12-month diagnoses at Waves 1 and 2.

2.2.2. Personality Disorders—The NESARC assessed Avoidant, Dependent, Paranoid, Obsessive-Compulsive, Schizoid, and Histrionic PDs at Wave 1, and Borderline, Narcissistic, and Schizotypal PDs at Wave 2. Antisocial PD, including assessment of conduct disorder before age 15 and AAB (at or after age 15), was assessed at Wave 1, with AAB (since last interview) reassessed at Wave 2. Although variables for all PDs are available in NESARC, the code for AAB is not available, so we developed an algorithm (available from the first author) to create the two variables for AAB assessed at Wave 1 and 2 because this is the only PD-related construct assessed at both waves, so it provides a direct evaluation of a potential time-of-measurement effect.

2.3. Statistical Analysis

Logistic regression analyses among participants diagnosed at Wave 1 were conducted for each anxiety disorder to determine the association of PDs and Wave 2 diagnostic status. The initial set of regressions included no covariates to determine the unadjusted bivariate association of PDs and anxiety-disorder persistence. A second set of regressions was conducted adjusting for demographics (i.e., sex, age, and race), Axis I disorders (i.e., substance use, unipolar, bipolar, and other anxiety disorders), and all other Axis II disorders (with separate analyses for Antisocial PD, Wave 1 AAB, and Wave 2 AAB). Those findings were compared to results from post-diction of Wave 1 disorders among participants diagnosed at Wave 2. Under the null hypothesis of absence of time-of-measurement effects, we expect that approximately 50% of the associations will be higher for prediction and postdiction regardless of the time of assessment of PDs. We tested the statistical significance of a pattern of findings suggesting a time-of-measurement effect using the probability under the binomial distribution of finding greater than or equal to the number of associations consistent with this effect (i.e., higher associations for post-diction among PDs assessed at Wave 1, and lower associations for post-diction among PDs assessed at Wave 2). Because parameter estimates could be correlated as a result of coming from the same analysis, using the same predictors across analyses, and/or being of the same analysis type (prediction or post-diction), a simple binomial probability will likely yield estimates that are too liberal. Consequently, in these analyses we estimated robust standard errors within a Generalized Estimating Equations framework (Liang & Zeger, 1986) to account for the nonindependence between the contrasts. We also compared results from prediction and postdiction analyses to identify instances in which formerly significant findings become nonsignificant when re-analyzed using post-diction. Analyses used SUDAAN (Research Triangle Institute, 2004) to adjust for the sampling weights in the calculation of standard errors of parameter estimates.

3. Results

3.1. Personality Disorder Comorbidity as a Function of Wave of Assessment

Table 1 shows the correlations among the NESARC PDs. As can be seen, within wave PD correlations (*Median* r = .50 at Wave 1, excluding the correlation between Antisocial PD and AAB-1, and .58 at Wave 2) are substantially higher than cross-wave correlations (*Median* r = .38, excluding the correlation of Antisocial PD and AAB-1 with AAB-2). This pattern of findings is strongly suggestive of time-of-measurement effects.

3.2. Persistence of Anxiety Disorders

Table 2 shows the rates of persistence of anxiety disorders using prediction and post-diction, all of which tend to be fairly low (i.e., less than 26%), although the persistence estimate for any anxiety disorder is over 30%. Rates of persistence are higher for SOP, SP, and GAD, than for PDw/A and PDw/oA. For PDw/oA, SOP, and SP, persistence rates were highly similar across prediction and post-diction. However, due to differences between numbers of participants diagnosed at each wave, rates of persistence differed for PDw/A and GAD when estimated through prediction or post-diction. This difference in prevalence between waves is particularly accentuated in GAD, with a Wave 2 prevalence that almost doubles the estimated prevalence at Wave 1. Thus, results regarding GAD must be taken with caution because there are a sizeable number of participants who are potentially misclassified regarding GAD (i.e., false negatives at Wave 1 and/or false positives at Wave 2).

3.3. Association between PDs and Persistence of Anxiety Disorders

The association of PDs with the persistence of anxiety disorders is shown in Tables 3 (unadjusted for covariates) and 4 (adjusted for covariates)². As can be seen in Table 3, differences between prediction and post-diction reflect a robust pattern of higher odds ratios (ORs) for post-diction among PDs assessed at Wave 1, and lower ORs for post-diction among PDs assessed at Wave 2 (consistent with a time-of-measurement effect). Of the 48 pairs of analyses comparing predictive and post-dictive ORs for PDs (and AAB) assessed at Wave 1 on our six anxiety-disorder measures, 47 (98%) were larger in the post-dictive than in the predictive models. In contrast, of the 24 pairs of analyses comparing predictive and post-dictive ORs for PDs (and AAB) assessed at Wave 2, 23 (96%) were larger in the predictive models. Taken together, 70 out of 72 pairs of analyses were consistent with a time-of-measurement effect, a pattern that is highly statistically significant, even when adjusting for the non-independence of estimates ($p < 10^{-7}$). Notably, with the only exception of Wave 2 AAB predicting PDw/oA persistence, the same pattern is found for AAB measured at Waves 1 and 2, indicating that the observed pattern consistent with a time-ofmeasurement effect is evident even when the same construct is measured at different times. (Note that although the same construct is being measured at both waves, Wave 1 covers adulthood up until Wave 1 assessment while Wave 2 covers the Wave 1 to Wave 2 interval.)

Further, a number of changes in statistical significance are found between prediction and post-diction. For example, although Dependent PD (Wave 1) is not significantly associated with the persistence of PDw/A, SOP, and SP in the forward prediction, these associations become significant in the post-diction. Similarly, although Borderline PD (Wave 2) is significantly associated with the persistence of the same three anxiety disorders in the forward prediction, the associations become much smaller and non-significant in the post-dictive analyses. These changes in statistical significance occur in 35% of the associations between PDs and anxiety disorders. Note that in contrast to this clear pattern for PD diagnoses, no such pattern is noted for associations with demographic variables where OR estimates are strikingly similar regardless of whether persistence was defined on the basis of prediction or post-diction. These patterns of findings suggest that the changes in the association between PDs and anxiety disorders observed when estimating ORs prospectively vs. retrospectively are due to time-specific, shared method variance and not to anything inherent in estimates based upon follow-up versus follow-back data.

Importantly, in spite of the pattern of findings suggesting a time-of-measurement effect, there are some associations (36%) that remain significant in both predictive and post-dictive

 $^{^{2}}$ Additional analyses including two variables coding for diagnosis with any PD measured at Wave 1 and any PD measured at Wave 2 yielded results that were largely consistent with those reported here. Those results are available from the first author.

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models (i.e., these associations are robust against the time-of-measurement effect). Although robust associations occur across all anxiety-disorder and PD measures (with the exception of AAB measured at Wave 2 and Narcissistic PD), the majority are related to GAD and Any anxiety disorder. The PDs that exhibit more robust associations are Avoidant PD (significantly associated with SP, SOP, GAD, and any anxiety disorder), Obsessive-Compulsive PD (significantly associated with PDw/A, SP, GAD, and any anxiety disorder), and Paranoid PD (significantly associated with SP, SOP, GAD, and any anxiety disorder).

Results of logistic regression analyses adjusting for covariates are also consistent with a time-of-measurement effect, although in this case the pattern is less robust (as would be expected given that covariates are drawn from both waves). Consistent with a time-of-measurement artifact, as shown in Table 4, the majority of PDs assessed at Wave 1 show higher ORs in the post-diction, whereas most PDs assessed at Wave 2 show higher ORs in the predictive models. Although 26% of the associations do not follow this general pattern, 53 out of 72 pairs of analyses were consistent with a time-of-measurement effect, a pattern that is statistically significant even after adjusting for the non-independence of estimates (p < .001). Moreover, none of the non-consistent findings involved a change in statistical significance. In contrast, 19 of the associations that suggest a time-of-measurement effect involve changes in statistical significance. Notably, this is the case for all associations involving Borderline PD, including its association with SP which is significant in the prediction and is still significant but *in the opposite direction* (i.e., predicting less persistence) in the post-diction.

In contrast to the numerous associations that were found to be significant in both predictive and post-dictive models of bivariate associations (Table 3), analyses adjusting for covariates yield very few robust associations (Table 4). In particular, Avoidant PD is significantly associated with SOP and Any Anxiety Disorder in both predictive and post-dictive models. Also, Obsessive-Compulsive PD is significantly associated with GAD, Antisocial is negatively associated with PDw/oA, and Schizotypal is negatively associated with PDw/A in both predictive and post-dictive models. However, the significant associations involving Antisocial PD and Schizotypal PD are not present in the bivariate analyses (see Table 3), indicating a suppression effect that makes it difficult to interpret clinically.

3.4. Statistical Significance of Difference between Associations

One limitation of the approach used in the preceding analyses is that a direct statistical test cannot be easily derived from the odds ratios obtained from prediction and post-diction, given that these are overlapping samples. A solution to this problem can be achieved by using multinomial logistic regression with a dependent variable that includes four groups: diagnosed at both waves (used as reference group), diagnosed only at Wave 1, diagnosed only at Wave 2, and not diagnosed at either Wave. This allows for a direct test of the equality of odds ratios from Wave 1 and Wave 2 only diagnoses. These analyses were conducted in Mplus (Muthén & Muthén, 1998-2012), and involved a chi-square difference test based on loglikelihood values and scaling correction factors (Satorra & Bentler, 2001) to determine if model fit was significantly worse when equality constraints were applied. As can be seen in Table 5, results are consistent with previous analyses in that associations reflect a time-of-measurement effect (although the direction of associations is reversed compared with previous tables because the reference group is now comprised of participants with persistent diagnosis). Moreover, odds ratios for groups diagnosed only at Wave 1 and only at Wave 2 are significantly (p < .05) different in 75% of cases, and *all* significant differences are in the predicted direction according to a time-of-measurement effect³.

4. Discussion

The importance of investigating the effect of PDs on the prospective course of anxiety disorders has been repeatedly highlighted in the literature (Ansell et al., 2011; Bienvenu & Stein, 2003; Brandes & Bienvenu, 2006; Massion et al., 2002). However, the only studies examining this issue have used treatment-seeking samples (Ansell et al., 2011; Massion et al., 2002). The availability of a nationally representative sample with a 3-year follow-up, including data on all ten PDs represents an exceptional opportunity to identify the effects of specific PDs adjusting for the presence of any other PD. Unfortunately, NESARC included assessment of 7 PDs at Wave 1 and 3 PDs at Wave 2, so that the estimation of unique effects for each PD becomes confounded with time of measurement. In the current analyses, we addressed this issue by comparing predictive models of persistence (i.e., Wave 2 anxiety disorders among participants diagnosed at Wave 1) with post-dictive models (i.e., Wave 1 anxiety disorders among participants diagnosed at Wave 2).

Results of the *bivariate* predictive models suggested that several associations are significant. In fact, all PDs showed statistically significant associations with the persistence of any anxiety disorder in these models. Moreover, Borderline PD emerged as the only PD showing significant associations with all anxiety-disorder measures in the bivariate predictive models. However, in post-dictive models 28% of those associations became non-significant, suggesting that they were not robust to a time-of-measurement effect. Consistent with this interpretation, all the associations that became non-significant in post-dictive analyses involved PDs measured at Wave 2. The clearest presentation of this finding is provided by AAB -measured at both waves (since age 15 at W1 and during the past three years at W2)-, showing a pattern of predictive and post-dictive associations that is reversed depending on the wave of assessment (with the only exception of Wave 2 AAB predicting PDw/oA persistence), suggesting that method effects are sufficiently larger than construct effects to determine the overall direction of association. Additional analyses using multinomial logistic regression revealed that this time-of-measurement effect yielded statistically significant differences in odds ratios.

Because the PDs were highly comorbid with one another, a multivariate analysis was necessary to detect unique effects of PDs adjusting for the presence of other PDs. As expected, the significant associations in multivariate analyses were highly reduced. However, predictive models still yielded some significant findings. For instance, four PDs (Avoidant, Borderline, Narcissistic, and Schizotypal) were associated with the persistence of any anxiety disorder. In addition, similar to bivariate analyses, Borderline PD had significant associations with all anxiety-disorder measures. However, post-dictive analyses revealed that only three associations were robust to a time-of-measurement effect, namely, Avoidant PD predicting persistence of SOP and any anxiety disorder, and Obsessive-Compulsive PD predicting GAD (although associations between Antisocial and PDw/oA, and between Schizotypal and PDw/A were robust to a time-of-measurement effect in multivariate analyses, they were not significant in bivariate analyses). This suggests that all other nonrobust associations are functionally cross-sectional and should not be considered validly predictive of persistence.

Indeed, if focus had been on predictive analyses as has been commonly utilized with this data set, the conclusion would have been that Borderline PD is consistently associated with

³There are five cases in Table 5 in which two non-significant odds ratios that are in the same direction are found to be significantly different from each other. Although this may be counterintuitive, it is possible that two non-significant estimates that are in the same direction might be significantly different from each other, provided that the covariance between the estimates is large and positive. We used the following equation to calculate the difference between each pair of estimates: $z = (b_2 - b_1) / (SE_1^2 + SE_2^2 - 2*cov(b_1, b_2))^{1/2}$ (Muthen, & Muthen, 1998–2012).

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higher rates of persistence in all anxiety disorders, even after adjustment for a number of covariates. However, taking into consideration the possible time-of-measurement effect via post-diction analyses, the associations involving Borderline PD become either non-significant or, even, *significant in the opposite direction*. This highlights the importance of this issue in the interpretation of results involving PDs in NESARC.

The design used in NESARC precludes a direct comparison among PDs measured at different waves; indeed, it is this methodological weakness that motivated our analytic approach. However, the findings of robust associations involving Avoidant and Obsessive-Compulsive PD are consistent with the broader literature. The two studies that examined the link of PDs with the persistence of anxiety disorders reported a significant association between Avoidant PD and SOP (Ansell et al., 2011; Massion et al., 2002). The similarity between these disorders has been extensively discussed in the literature (Rettew, 2000; Widiger, 1992). Although Avoidant PD has been found to reflect a higher severity of social inhibition and impairment in some studies (Hummelen, Wilberg, Pedersen, & Karterud, 2007; Turner, Beidel, Dancu, & Keys, 1986), specifically regarding the Avoidant PD criterion involving emotional guardedness (Marques et al., 2012), other researchers have reported only minimal differences (Ralevski et al., 2005). In addition, both disorders have been found to share the same genetic liability (Reichborn-Kjennerud, 2007). In sum, the finding that Avoidant PD is uniquely associated with SOP persistence might reflect the fact that diagnosis of both disorders at a given wave is indicative of a more reliable assessment of the same construct, rather than of two distinct, comorbid disorders (Widiger, 1992).

With regard to the association between Obsessive-Compulsive PD and GAD, there are fewer previous reports. Obsessive-Compulsive PD was found to be associated with higher rates of relapse from GAD in the CLPS (Ansell et al., 2011). However, this study did not find an association with GAD persistence. Although the common aspects of GAD and OCD have been studied (Gentes & Ruscio, 2011), in particular regarding similarities between worries and obsessions (Abramowitz & Foa, 1998; Comer, Kendall, Franklin, Hudson, & Pimentel, 2004), the association of GAD and Obsessive-Compulsive PD has not been examined in detail. Data from the CLPS showed that GAD was significantly associated with two Obsessive-Compulsive PD criteria: preoccupation with details and reluctance to delegate tasks (Eisen et al., 2006). The authors suggested that these criteria may be associated with intolerance of uncertainty, considered a core feature of both GAD and OCD (Gentes & Ruscio, 2011). In NESARC, Obsessive-Compulsive PD has also been found to be associated with the incidence of GAD, although this association becomes non-significant when adjusting for other psychiatric disorders (Grant et al., 2009). In spite of this background knowledge that supports the potential association of Obsessive-Compulsive PD and GAD, it is important to remember that results regarding GAD must be taken with caution in the current study, given the differences in GAD prevalence between waves (see Table 2). Moreover, although this finding was robust to replication across predictive and post-dictive models, it needs to be emphasized that only three of 66 analyses were robust in this way, consistent with chance expectation. Thus, although the priors for expecting an association between cluster C PDs and various anxiety disorders would be relatively high (arguing against a "false discovery"), these findings should be replicated in an independent sample.

The current results raise serious concerns about the validity of the conclusions of published findings suggesting a strong effect of PDs on Axis I disorder persistence using NESARC data, since they did not rigorously address the time-of-measurement effect. One article, for example, simply conjectures that an artifactual finding due to time-of-measurement "is unlikely because i) other personality disorders were significant predictors of other outcomes in the same dataset ..., and ii) Axis II disorders were diagnosed based on symptoms that were defined in the interview as being stable and enduring" (Fenton et al., 2011, p. 606).

However, the evidence cited to support the first point is based on papers where the most consistent predictors of Axis I disorder persistence were PDs assessed at Wave 2. Moreover, even if PDs are defined as stable and enduring, such definitions do not directly translate into empirical reality as we clearly demonstrate. Further, as shown compellingly in Table 3, PDs measured by the AUDADIS do not behave as other stable variables like demographic predictors. Another published report addressed this critical time-of-measurement issue more systematically (Skodol et al., 2011). This study compared participants with and without a PD with regard to mental health functioning and life events indicative of impaired functioning at both waves. They found that participants diagnosed with a PD were more impaired at both waves, regardless of when the PD was diagnosed. Although this provides evidence that the PD diagnoses imply durable impairment, it does not rule out an artifact due to time of assessment when comparing PDs in their association with those outcomes. In fact, to support the idea of a time artifact, Tables 1 and 2 in their data supplement showed a pattern of higher associations of PDs with outcomes when both were assessed at the same time. For instance, seven of the PDs (in Table 1) are associated with lower scores in the mental component summary measured at the same wave than the other wave. The two exceptions (Schizotypal and Narcissistic PD) show negligible differences between the two waves. In addition, of the seven changes in statistical significance between waves reported in Table 2 of their data supplement, six correspond to significant associations with outcomes measured at the same wave and non-significant associations with outcomes measured at the alternative wave.

Although it would be ideal to quantify the time-of-measurement effect in a way that allows for a correction so that accurate persistence estimates can be made in NESARC, to the best of our knowledge this is not possible given that no PD was measured at both waves (in the case of Antisocial PD, only AAB was measured again at Wave 2). Thus, drawing strong conclusions about the role of PDs in the persistence of anxiety disorders from NESARC is compromised by the design, which results in clear time-of-measurement effects. However, "absence of evidence is not evidence of absence" and the compromised robustness of findings that are conditional upon time of measurement should not be taken to demonstrate that some conditions (e.g., Borderline PD) are irrelevant to persistence of anxiety disorders. Rather, the NESARC simply does not provide strong evidence in this regard. In the future, when response burden is an issue for longitudinal studies as it was for NESARC, we recommend a missing-by-design strategy (i.e., subgroups of respondents receiving different packets of assessment; Graham, Taylor, Olchowski, & Cumsille, 2006) rather than the approach used in NESARC, where different constructs were assessed at different times.

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Highlights

- We examined the association of PDs with the persistence of anxiety disorders.
- The pattern of associations indicates a time-of-measurement effect.
- Previous findings involving PDs in NESARC should be taken with caution.

Table 1

Correlations among personality disorders measured at Wave 1, Wave 2, and at both waves

				Measured a	t Wave 1					Meas	sured at Wave	2
	Antisocial	AAB-1	Avoidant	Dependent	OCPD	Paranoid	Schizoid	Histrionic	AAB-2	BPD	Schizotypal	Narcissistic
/ave 1												
ntisocial	1.00											
.AB-1	0.77	1.00										
voidant	0.38	0.35	1.00									
ependent	0.44	0.27	0.81	1.00								
CPD	0.39	0.37	0.54	0.50	1.00							
aranoid	0.47	0.44	0.69	0.65	0.61	1.00						
chizoid	0.42	0.36	0.62	0.55	0.58	0.69	1.00					
listrionic	0.48	0.46	0.50	0.65	0.55	0.67	0.46	1.00				
Vave 2												
AB-2	0.45	0.44	0.27	0.40	0.16	0.31	0.28	0.38	1.00			
PD	0.36	0.37	0.51	0.49	0.35	0.51	0.40	0.48	0.51	1.00		
chizotypal	0.37	0.31	0.47	0.47	0.39	0.48	0.44	0.41	0.45	0.77	1.00	
arcissistic	0.31	0.27	0.29	0.32	0.32	0.38	0.28	0.46	0.46	0.66	0.65	1.00

and Wave 2 PDs. AAB = Mana Adult Antisocial Behavior; BPD = Borderline Personality Disorder; OCPD = Obsessive-Compulsive Personality Disorder.

Table 2

Persistence (Percentage and Standard Errors) of Different Anxiety Disorders Defined by Prediction and Post-diction

Estimation	Panic Disorder w/ Agoraphobia	Panic Disorder w/o Agoraphobia	Social Phobia	Specific Phobia	GAD	Any Anxiety
Prediction ^{<i>a</i>}	17.18 (2.99)	11.97 (1.72)	21.98 (1.66)	25.58 (1.05)	24.24 (1.99)	35.11 (0.90)
Post-diction ^b	12.52 (2.27)	10.47 (1.49)	24.54 (1.69)	24.43 (1.12)	13.11 (1.16)	31.29 (0.99)
W1 Prevalence ^c	0.56 (0.05)	1.55 (0.07)	2.75 (0.13)	7.13 (0.26)	2.06 (0.10)	11.08 (0.33)
W2 Prevalence ^c	0.79 (0.06)	1.79 (0.09)	2.54 (0.12)	7.50 (0.21)	3.78 (0.15)	12.52 (0.28)
<i>Note</i> . GAD = Gene	ralized Anxiety Diso	rder.				

 $^{\prime\prime}$ Prevalence of disorder at Wave 2 among those diagnosed at Wave 1.

 $b_{\mbox{Prevalence}}$ of disorder at Wave 1 among those diagnosed at Wave 2.

 c Past-12-month diagnoses.

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Forward and backward bivariate associations (odds ratios and 95% confidence intervals) of personality disorders and the persistence of anxiety disorders

	Panic Disorder v	v/ Agoraphobia	Panic Disorder v	w/o Agoraphobia	Social .	Phobia	Specific	: Phobia	GA	D	Any Au	ıxiety
Predictor	$\begin{array}{l} Prediction \\ W1 \rightarrow W2 \\ (N=218) \end{array}$	Post-diction W2→W1 (N = 304)	Prediction W1 \rightarrow W2 (N = 557)	Post-diction W2→W1 (N = 646)	Prediction W1→W2 (N = 989)	Post-diction W2→W1 (N = 945)	Prediction W1 \rightarrow W2 (N = 2579)	Post-diction W2→W1 (N = 2758)	Prediction W1 \rightarrow W2 (N = 746)	Post-diction W2 \rightarrow W1 (N = 1363)	$\begin{array}{l} Prediction \\ W1 \rightarrow W2 \\ (N = 4010) \end{array}$	Post-diction W2→W1 (N = 4561)
Measured at V	Vave 1											
Sex ^a	0.33 (0.15–0.74)	0.50 (0.22–1.11)	0.60 (0.31–1.18)	0.71 (0.38–1.36)	1.01 (0.67–1.53)	1.15 (0.74–1.77)	1.32 (1.04–1.69)	1.28 (0.98–1.66)	1.22 (0.75–1.99)	1.24 (0.76–2.02)	1.32 (1.11–1.57)	1.30 (1.08–1.56)
Age^{b}	2.25 (0.75–6.75)	2.59 (0.91–7.38)	1.52 (0.68–.37)	1.91 (0.85–4.29)	0.88 (0.60–1.30)	1.11 (0.72–1.72)	1.12(0.86 - 1.45)	1.13 (0.85–1.50)	1.30 (0.70–2.42)	1.21 (0.69–2.14)	0.95 (0.79–1.15)	1.04 (0.86–1.26)
Race ^c	$0.50\ (0.18{-}1.41)$	0.28 (0.10-0.77)	0.38 (0.20-0.72)	0.39 (0.20–0.77)	0.87 (0.56–1.33)	0.70 (0.47–1.06)	0.74 (0.59–0.94)	0.60 (0.47–0.78)	0.82 (0.52–1.29)	0.75 (0.50–1.14)	0.81 (0.68–0.97)	0.71 (0.60–0.84)
AAB-1	1.33 (0.60–2.92)	1.92 (0.93–3.98)	1.22 (0.65–2.28)	2.03 (1.09–3.78)	1.16(0.79 - 1.69)	1.66 (1.13–2.45)	1.30 (1.01–1.67)	1.66 (1.26–2.19)	0.79 (0.48–1.30)	1.48 (0.94–2.35)	1.45 (1.22–1.73)	2.06 (1.71–2.49)
Antisocial	$1.00\ (0.32 - 3.15)$	1.78 (0.54–5.86)	$0.61 \ (0.19 - 1.99)$	0.71 (0.22–2.31)	1.47 (0.85–2.53)	2.29 (1.26-4.17)	1.28 (0.83–1.96)	2.08 (1.40–3.08)	1.23 (0.63–2.39)	2.84 (1.53–5.25)	1.71 (1.23–2.37)	2.88 (2.09–3.96)
Avoidant	1.88 (0.73–4.82)	4.32 (1.69–11.02)	0.99 (0.42–2.32)	1.57 (0.60-4.08)	3.04 (2.07–4.47)	$6.86 \ (4.41 - 10.67)$	1.72 (1.20–2.45)	3.50 (2.39–5.13)	2.39 (1.49–3.81)	6.35 (3.91–10.33)	2.68 (2.06–3.48)	7.40 (5.44–10.06)
Dependent	1.85 (0.55–6.25)	7.07 (1.83–27.26)	1.05 (0.12-8.87)	1.20 (0.13–10.70)	2.08 (0.91-4.74)	2.88 (1.12–7.45)	2.06 (0.92-4.61)	4.98 (2.32–10.69)	3.58 (1.63–7.85)	6.93 (2.85–16.83)	3.48 (2.04–5.95)	10.74 (5.48–21.07)
OCPD	2.34 (1.08–5.11)	5.29 (2.40–11.65)	1.52 (0.74–3.14)	1.93 (0.92-4.06)	1.33 (0.92–1.92)	3.07 (2.03-4.64)	1.38 (1.05–1.80)	2.93 (2.18–3.93)	2.50 (1.62–3.86)	5.47 (3.49–8.57)	1.80 (1.51–2.14)	3.99 (3.28–4.86)
Paranoid	$1.34\ (0.55 - 3.26)$	2.92 (1.25–6.83)	1.33 (0.63–2.81)	1.89 (0.84-4.27)	1.98 (1.32–2.97)	4.91 (3.11–7.75)	1.79 (1.31–2.43)	3.74 (2.70–5.16)	1.97 (1.29–3.00)	5.60 (3.65-8.61)	2.40 (1.92–2.99)	6.06(4.87 - 7.54)
Schizoid	1.21 (0.54–2.75)	2.69 (1.11–6.54)	1.05 (0.31–3.61)	2.02 (0.56–7.33)	1.57 (1.03–2.40)	3.63 (2.31–5.71)	1.30 (0.91–1.86)	2.45 (1.69–3.54)	2.29 (1.46–3.58)	5.39 (3.47–8.37)	1.58 (1.26–1.99)	3.83 (2.91–5.04)
Histrionic	0.83 (0.22–3.16)	1.89 (0.49–7.32)	2.09 (0.77–5.68)	1.83 (0.70-4.79)	1.42 (0.80–2.52)	1.79 (0.92–3.47)	1.50 (0.96–2.34)	4.06 (2.48–6.65)	2.05 (0.97-4.37)	3.72 (1.88–7.35)	2.05 (1.46–2.88)	4.52 (3.10–6.58)
Measured at V	<u>Vave 2</u>											
AAB-2	2.01 (0.65–6.19)	1.13 (0.35–3.67)	1.12 (0.31–4.14)	1.23 (0.33–4.56)	2.26 (0.98–5.20)	0.65 (0.33–1.30)	1.59 (0.81–3.13)	1.19 (0.67–2.11)	2.40 (0.87–6.62)	1.17 (0.51–2.70)	2.04 (1.32–3.17)	1.05 (0.70–1.59)
Borderline	3.16 (1.27–7.89)	1.10 (0.42–2.88)	2.70 (1.43–5.11)	2.02 (1.07–3.81)	3.52 (2.35–5.25)	1.14 (0.77–1.67)	2.42 (1.78–3.30)	1.15 (0.89–1.49)	3.21 (2.03–5.06)	1.63 (1.08–2.46)	4.97 (4.04–6.11)	1.72 (1.45–2.04)
Narcissistic	2.17 (0.89–5.28)	1.11 (0.44–2.75)	1.97 (0.93–4.14)	1.68 (0.81–3.50)	2.54 (1.53-4.20)	0.94 (0.60–1.47)	1.53 (1.06–2.21)	0.78 (0.56–1.09)	3.09 (1.77–5.40)	1.48 (0.90–2.44)	2.97 (2.26–3.90)	1.20 (0.95–1.52)
Schizotypal	$0.80\ (0.34{-}1.88)$	$0.45\ (0.19{-}1.08)$	1.93 (0.77–4.82)	1.11 (0.42–2.94)	3.19 (2.10–4.86)	1.33 (0.88–2.00)	2.77 (1.93–3.97)	1.19 (0.89–1.59)	3.09 (1.85–5.15)	1.79 (1.17–2.75)	4.26 (3.28–5.52)	1.62 (1.30–2.02)
Vote. $GAD = G\epsilon$	neralized Anxiety D	isorder; AAB = Adu	ılt Antisocial Behavi	or; $OCPD = Obsessiv$	re-Compulsive Perso	mality Disorder.						

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^aMale is reference group.

 b_{18} to 29 is reference group.

^cWhite, Non-Hispanic is reference group.

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Forward and backward associations (adjusted odds ratios and 95% confidence intervals) of personality disorders and the persistence of anxiety disorders

	Panic Disorder	· w/ Agoraphobia	Panic Disorder w	v/o Agoraphobia	Social 1	Phobia	Specific	Phobia	GA	Q	Any A	ixiety
Predictor	Prediction W1 \rightarrow W2 (N = 218)	$\begin{array}{l} \text{Post-diction} \\ \text{W2} \rightarrow \text{W1} \\ (\text{N} = 304) \end{array}$	$\begin{array}{l} Prediction \\ W1 \rightarrow W2 \\ (N = 557) \end{array}$	Post-diction W2→W1 (N = 646)	Prediction W1 \rightarrow W2 (N = 989)	Post-diction W2 \rightarrow W1 (N = 945)	Prediction W1 \rightarrow W2 (N = 2579)	Post-diction W2→W1 (N = 2758)	Prediction W1 \rightarrow W2 (N = 746)	Post-diction W2 \rightarrow W1 (N = 1363)	$\begin{array}{l} \mbox{Prediction} \\ \mbox{W1} {\rightarrow} \mbox{W2} \\ \mbox{(N = 4010)} \end{array}$	Post-diction W2→W1 (N = 4561)
Measured at '	Wave 1											
AAB-1 ^a	0.96 (0.34–2.74)	0.45 (0.15–1.37)	0.68 (0.35–1.32)	1.39 (0.71–2.74)	$0.84\ (0.55{-}1.28)$	1.25 (0.74–2.11)	$1.08\ (0.81 - 1.44)$	1.08 (0.78–1.49)	0.57 (0.31–1.03)	0.63 (0.33–1.21)	1.05 (0.85–1.29)	1.18(0.94 - 1.49)
Antisocial ^a	1.50 (0.43–5.19)	0.57 (0.08–4.15)	0.25 (0.08–0.83)	0.26 (0.08–0.91)	0.98 (0.52–1.85)	1.29 (0.59–2.81)	0.93 (0.55–1.56)	1.47 (0.92–2.37)	0.92 (0.43–1.95)	1.03 (0.44–2.39)	1.07 (0.75–1.53)	1.37 (0.97–1.96)
Avoidant	1.04 (0.39–2.81)	$0.74\ (0.14-3.88)$	0.72 (0.20–2.54)	0.45 (0.08–2.43)	2.27 (1.37–3.75)	4.36 (2.50–7.59)	1.04 (0.67–1.63)	1.24 (0.76–2.02)	1.12 (0.61–2.06)	1.70 (0.86–3.38)	1.53 (1.12–2.09)	3.15 (2.13–4.66)
Dependent	0.97 (0.27–3.44)	19.34 (2.85–131.09)	1.35 (0.12–15.17)	0.60 (0.02–17.32)	0.83 (0.29–2.37)	0.33 (0.09–1.19)	0.92 (0.39–2.15)	0.71 (0.20–2.59)	2.44 (0.93–6.41)	1.60 (0.58-4.38)	1.20 (0.63–2.27)	1.17 (0.51–2.70)
OCPD	2.58 (1.00–6.70)	1.61 (0.59–4.37)	1.34 (0.61–2.97)	1.19 (0.53–2.65)	0.96 (0.61–1.51)	1.15(0.68 - 1.93)	1.04 (0.76–1.41)	1.71 (1.21–2.42)	1.85 (1.15–2.99)	2.86 (1.74-4.71)	1.19 (0.96–1.48)	2.20 (1.73–2.80)
Paranoid	1.63 (0.50–5.34)	0.31 (0.07–1.31)	1.21 (0.43–3.43)	1.47 (0.41–5.28)	0.92 (0.54–1.59)	2.41 (1.23-4.70)	1.22 (0.82–1.81)	2.11 (1.34–3.33)	0.90 (0.56–1.45)	1.33 (0.75–2.36)	1.27 (0.96–1.68)	2.50 (1.87–3.34)
Schizoid	0.52 (0.18–1.47)	1.45 (0.43-4.92)	0.71 (0.25–2.03)	1.12 (0.29-4.30)	$0.83\ (0.50{-}1.40)$	1.31 (0.68–2.52)	0.80 (0.52–1.22)	$0.90\ (0.54{-}1.50)$	1.51 (0.94–2.42)	1.63 (0.94–2.83)	0.77 (0.59–1.01)	1.20 (0.85–1.68)
Histrionic	0.47 (0.07–2.97)	3.23 (0.33–31.51)	1.94 (0.59–6.34)	0.84 (0.27–2.60)	$0.94\ (0.46 - 1.93)$	0.86 (0.34–2.19)	1.08 (0.66–1.78)	2.63 (1.43–4.83)	1.09(0.48-2.49)	$0.96\ (0.42-2.19)$	0.97 (0.67–1.40)	1.56 (0.98–2.49)
Measured at 1	Wave 2											
AAB-2 ^a	3.77 (0.83–17.11)	2.31 (0.36–14.76)	0.80 (0.20–3.20)	1.33 (0.35–5.02)	1.41 (0.49–4.04)	0.57 (0.26–1.23)	$1.14\ (0.54-2.40)$	1.37 (0.69–2.73)	1.75 (0.61–5.07)	0.79 (0.34–1.87)	1.03 (0.60–1.77)	$0.78\ (0.48{-}1.26)$
Borderline	4.55 (1.30–15.91)	1.19(0.37 - 3.80)	2.62 (1.11–6.20)	1.85 (0.84-4.07)	1.91 (1.14–3.19)	0.72 (0.40–1.27)	1.60 (1.06–2.41)	0.63 (0.42–0.93)	1.91(1.08 - 3.40)	0.68 (0.38–1.22)	2.70 (2.11–3.47)	0.94 (0.74–1.19)
Narcissistic	3.76 (1.11–12.79)	$1.89\ (0.48-7.51)$	2.01 (0.79–5.09)	2.04 (0.91-4.58)	1.33 (0.72–2.49)	0.82 (0.44–1.52)	1.08 (0.74–1.60)	0.71 (0.48–1.04)	2.22 (1.19–4.13)	1.25 (0.69–2.28)	1.71 (1.26–2.32)	0.93 (0.72–1.20)
Schizotypal	0.14 (0.04–0.56)	0.22 (0.05–0.97)	0.82 (0.28–2.36)	0.53 (0.19–1.51)	1.77 (1.03–3.02)	0.92 (0.50–1.69)	1.74 (1.15–2.64)	0.80 (0.52–1.22)	1.53 (0.84–2.77)	1.01 (0.57–1.79)	1.79 (1.28–2.51)	0.94 (0.69–1.27)
Vote. Adjusting	for demographics (se	x, age and race), Axis I	disorders (substance	use, unipolar, bipolar,	and other anxiety d	isorders), and all ot	her Axis II disorders	. Models with Any 4	Anxiety Disorder as	dependent variable o	did not include anxi	ty disorders as covaria

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ttes. GAD = aury Dis Anxiety eq ē

^aSeparate analyses were conducted for Wave 1 AAB, Wave 2 AAB, and Antisocial PD. Estimates for all other PDs are from regression including Antisocial PD.

Table 5

Odds Ratios from Multinomial Logistic Regression predicting Diagnosis only at Wave 1 or only at Wave 2 (with Diagnosis at both Waves as Reference Group), and Significance Tests of Equality of the Odds Ratios

	Non-Persistent P	anic Disorder w/	Agoraphobia	Non-Persistent P.	anic Disorder w/o	Agoraphobia	Non-Persis	tent Social	Phobia 1	Von-Persiste	ent Specific	Phobia	Non-P	ersistent G	AD	Non-Pers	istent Any /	unxiety
Predictor	W1 only	W2 only	χ ² (1)	W1 only	W2 only	χ ² (1)	W1 only	W2 only	χ ² (1)	W1 only	W2 only	χ ² (1)	W1 only	W2 only	$\chi^{2(1)}$	W1 only	W2 only	χ ² (1)
Measured at V	<u>Vave 1</u>																	
AAB-1	0.75	0.52	1.82	0.82	0.49*	10.25^{*}	0.86	0.60*	6.50^{*}	0.77*	0.60*	5.82*	1.27	0.67*	19.90*	0.69*	0.49*	17.87*
Antisocial	1.00	0.56	3.85	1.64	1.40	0.37	0.68	0.44*	3.17	0.78	0.48^{*}	7.19*	0.82	0.35^{*}	15.52*	0.59*	0.35^{*}	12.92*
Avoidant	0.53	0.23*	9.22*	1.02	0.63	2.53	0.33*	0.15^{*}	30.52*	0.58*	0.29*	20.09*	0.42*	0.16^{*}	23.01*	0.37*	0.14^{*}	55.23*
Dependent	0.54	0.14^{*}	10.58*	0.95	0.83	0.06	0.48	0.35^{*}	0.94	0.49	0.20^{*}	7.38*	0.28^{*}	0.14^{*}	3.39	0.29*	*60.0	11.33*
OCPD	0.43*	0.19^{*}	9.18*	0.66	0.52	1.66	0.75	0.33*	31.86^{*}	0.73*	0.34^{*}	48.22*	0.40^{*}	0.18^{*}	30.03*	0.56^{*}	0.25*	89.55*
Paranoid	0.74	0.34^{*}	8.41*	0.75	0.53	2.48	0.51^{*}	0.20*	22.46*	0.56^{*}	0.27*	37.63*	0.51^{*}	0.18^{*}	43.46*	0.42*	0.17*	66.42*
Schizoid	0.82	0.37*	8.14*	0.95	0.50	7.13*, ^a	0.64^{*}	0.28^{*}	25.27*	0.77	0.41^{*}	20.35*	0.44^{*}	0.19*	23.69*	0.63^{*}	0.26^{*}	54.15*
Histrionic	1.20	0.53	3.63	0.48	0.55	0.23	0.71	0.56	0.78	0.67	0.25^{*}	24.68*	0.49	0.27*	5.80*	0.49*	0.22*	27.39*
Measured at V	Vave 2																	
AAB-2	0.50	0.89	1.48	0.89	0.82	0.07	0.44	1.54	19.57*	0.63	0.84	1.41	0.42	0.85	7.16*, ^a	0.49*	0.95	17.27*
Borderline	0.32^{*}	0.91	18.20^{*}	0.37*	0.49*	2.90	0.28*	0.88	68.56*	0.41^{*}	0.87	34.34*	0.31^{*}	0.61^{*}	21.50*	0.20^{*}	0.58*	123.83*
Narcissistic	0.46	0.91	4.06*, ^a	0.51	0.60	0.46	0.39*	1.06	26.68*	0.65^{*}	1.28	23.11*	0.32^{*}	0.67	17.16^{*}	0.34^{*}	0.83	65.21*
Schizotypal	1.25	2.23	5.38*, ^a	0.52	0.90	7.30*, ^a	0.31^{*}	0.76	25.37*	0.36^{*}	0.84	31.16*	0.32^{*}	0.56^{*}	9.06*	0.24^{*}	0.62^{*}	71.56*
<i>Note</i> . $GAD = G\epsilon$	meralized Anxiety	Disorder; AAB =	Adult Antisocial	Behavior; OCPD =	- Obsessive-Compu	lsive Personality	Disorder.											

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^aAlthough the effect estimates were not significantly different from an odds ratio of 1.00 they were significantly different from one another (see footnote 2).