Neuroticism and its Associations with Higher Cognitive Functions

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Dedication

Dedicated to my mom, who tells me to write.

Abstract

Psychopathology may be understood better as a spectrum, as opposed to a dichotomy, and the traits that underlie this spectrum can shed light on the underlying mechanisms of the pathology. The personality trait Neuroticism, which relates to the experience and expression of negative emotion, is strongly associated with psychopathology; the aspects of Neuroticism—N-Withdrawal and N-Volatility—share variance but are also uniquely associated with different types of psychopathology. Intelligence and creativity are two other traits that are associated with psychopathology; intelligence is negatively correlated, while creativity is sometimes positively correlated. Some of this correlation can be explained through their relationship to Neuroticism and its aspects. The current study examined the relationship between intelligence, creativity, and the aspects of Neuroticism, as well as explored potential neural mechanisms of Neuroticism. N-Volatility was negatively correlated with intelligence, while N-Withdrawal showed a curvilinear relationship, where subjects in the middle of the N-Withdrawal spectrum performed best on cognitive tasks. However, N-Volatility positively predicted creativity—particularly artistic creativity—in some samples, while N-Withdrawal showed no or a slight negative correlation. Finally, N-Withdrawal was negatively associated with functional connectivity in areas of the brain related to emotional regulation and decision making. These findings suggest that personality mediates the relationship between psychopathology, creativity, and intelligence, and imply a neural substrate of Neuroticism.

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1. Introduction

Patterns of human behavior, cognition, or affect—traits—generally fall on a normal curve, with most people falling somewhere in the middle and relatively few falling on the extreme ends. In many cases, pathological traits are the extreme end of a normal spectrum of a trait—as opposed to a dichotomous trait, where a person either has a pathological trait or does not (Craddock & Owen, 2010; Wright et al., 2013). Traits reflect patterns of biological processes in the brain, and persons with pathological traits should have abnormal functioning in the brain. Thus, studying the normal biological processes that underlie a trait can illuminate the malfunction that occurs in the extreme, pathological form of the trait. Further, traits that are commonly associated with psychopathology may result from similar cognitive and affective functions that create the pathology.

Therefore, in order to better understand psychopathology, it is necessary to understand the normal function of traits that are associated with it. Neuroticism, as the personality trait most strongly linked with psychopathology (e.g., Ormel et al., 2004; Widiger, 2011), obviously bears closer examination. Since high Neuroticism is associated so strongly with certain forms of psychopathology (e.g., Kotov et al., 2010; Ormel, Riese, & Rosmalen, 2012), the association that other traits show with these disorders may in fact be causally related to Neuroticism, as opposed to the disorder itself.

Neuroticism correlates with almost all types of psychopathology (Kotov et al., 2010); this correlation is so strong that Neuroticism measures can be unhelpful in distinguishing between disorders (Ormel, Rosmalen, & Farmer, 2004). Further, because

of the close relationship between Neuroticism and psychopathology, certain traits—such as anxiety—correlate so closely with Neuroticism that the terms are sometimes used interchangeably and likely relate to the same underlying cognitive and affective processes. At the neural level, Neuroticism relates to individual variation in responding to negatively-interpreted stimuli (Ormel et al., 2013); on a more cognitive and affective level, variations reflect differences in experience and expression of negative emotions (Caspi, Roberts, & Shiner, 2005; Markon, Krueger, & Watson, 2005; Matthews, 2004). Neuroticism relates to brain structures and systems that modulate sensitivity to punishment as well as negative affect (DeYoung, 2010). There have been myriad studies examining the neural correlates of Neuroticism; they have implicated the amygdala (e.g., Cremers et al., 2010; Holmes et al., 2012; Kim & Whalen, 2009; Omura, Constable, & Canli, 2005) and the cingulate cortex (e.g., Canli et al., 2004; Eisenberger, Lieberman, & Satpute, 2005), as well as other structures (e.g., Allen & DeYoung, in press; Feinstein, Stein, & Paulus, 2006; Haas, Constable, & Canli, 2008). It is clear that Neuroticism as a personality construct directly relates to neural activity.

1.1 Aspects of Neuroticism.

The qualities that cluster in Neuroticism have been empirically grouped into two separate aspects: Volatility (N-Volatility) and Withdrawal (N-Withdrawal). N-Volatility includes facets like irritability and angry hostility, reflecting the outward expression of negative emotion (DeYoung, Quilty, & Peterson 2007). N-Withdrawal includes facets such as anxiety and fearfulness, reflecting inward expression of negative emotion (DeYoung, Quilty, & Peterson, 2007). Both N-Volatility and N-Withdrawal relate to how

individuals cope with threat, and individuals can be high or low on both measures. N-Withdrawal generally consists of avoiding or withdrawing from possible negative situations, and N-Volatility relates to increased attention to negative situations and to corresponding actions (Cunningham et al., 2010). Both aspects correspond to the individual's tendency to interpret ambiguous or neutral stimuli in a negative way (van Doorn & Lang, 2010). Measuring the aspects separately may capture more of the variance and account for contradictory findings for Neuroticism (van Doorn & Lang, 2010).

There is also evidence that the two aspects are associated with differential behavioral responses. For example, in a prisoner's dilemma paradigm, subjects who scored more highly on N-Withdrawal measures were more likely to cooperate, possibly because they had a greater fear of punishment or negative situations; this effect was not present for high N-Volatility scores (Hirsh & Peterson, 2009). Another experiment looked at performance with different levels of subject investment; for difficult tasks with a high level of investment, subjects who scored more highly on N-Volatility performed worse, whereas subjects who scored more highly on N-Withdrawal performed better (van Doorn & Lang, 2010). This could also reflect the association of N-Withdrawal and punishment avoidance. In many studies, the effect of Neuroticism on performance has seemed ambiguous, possibly because its aspects can have opposing effects (Bipp & Kleinbeck, 2011).

1.2 Neuroimaging and the Aspects

Differences in the expression of the aspects are also reflected in brain structure and

functioning. Neuroticism has been associated both positively and negatively with ventral prefrontal cortex functioning in response to negative stimuli (Kennis, Rademaker, & Geuze, 2012), which may parallel the finding of the aspects' opposing effects. DeYoung (2010) has hypothesized that N-Volatility is associated with the flight-fight-freeze system (FFFS), whereas N-Withdrawal is associated with the behavioral inhibition system (BIS); these differences may be reflected in variations in amygdala activation (Cunningham et al., 2010). The BIS is linked to passive avoidance (characteristic of N-Withdrawal) and is distributed between several different neural systems (McNaughton & Corr, 2004). The FFFS controls active responses to aversive stimuli, and it seems to make use of parallel responses in a similar neural hierarchy (Corr, DeYoung, & McNaughton, 2013; McNaughton & Corr, 2004). These systems are connected to emotional processing and subjective experience of negative affect (Ochsner et al., 2002). Like Neuroticism itself, N-Volatility and N-Withdrawal directly relate to neural activity; however, as Allen and DeYoung (in press) note in their review of the literature, very few neuroimaging studies have separated the aspects. As the aspects influence behavior in different ways, it stands to reason that these differences would be reflected in brain structure and functioning.

1.3 Internalizing and Externalizing

The aspects are also associated with different types of psychopathology. It is important to note that comorbidity in mental illness—or multiple disorders occurring at rates greater than chance—is very high; as many as 60% of patients diagnosed with one disorder will be diagnosed with another in their lifetime (Kessler, Berglund, Demler, Jin, & Walters, 2005). This suggests that some disorders may share similar etiological bases,

as well as similar patterns of cognition, affect, and behavior. Two broad factors that correspond with different types of psychopathology are Internalizing and Externalizing. Internalizing and Externalizing both consist of a spectrum with multiple related disorders that share an etiological basis (Kramer, Krueger, & Hicks, 2008). Mood disorders and anxiety disorders fit on the Internalizing spectrum, while substance abuse, antisocial behavior, and impulsive disorders fit on the Externalizing spectrum (Griffith et al., 2010; Hicks et al., 2009; Kramer, Krueger, & Hicks, 2008).

Internalizing is involved in the inward expression of negative emotion and heightened emotional response to negative stimuli. It is characterized by rumination, or repetitively thinking negative thoughts without taking action to alleviate them (e.g., McLaughlin & Nolen-Hoeksema, 2012). Rumination is a core symptom of depression and anxiety disorders (McLaughlin & Nolen-Hoeksema, 2012; Oppeneheimer et al., 2012) but may be more related to vulnerability to anxiety disorders (Lopez et al., 2012). However, a general negative cognitive style—the tendency to interpret ambiguous stimuli as negative stimuli—more closely relates to depression (Rood et al., 2012). Both anxiety disorders and depression are highly comorbid, and share underlying genetic vulnerability (e.g., Kendler et al., 1995; Middledorp et al., 2005). In fact, depression and anxiety disorders may share substantial comorbidity precisely because of their shared underlying vulnerability and traits; they are highly correlated with anxious-fearful facets of Neuroticism (Kramer, Krueger, & Hicks, 2008; Tyrer, Seivewright, & Johnson, 2003). The comorbidity may be the result of shared psychological processes that manifest slightly differently across individuals (Krueger, 1999).

Like Internalizing, Externalizing consists of a spectrum where shared processes manifest in slightly different ways (Markon & Krueger, 2006). Externalizing accounts for shared variance between substance use disorders and antisocial behavior (Krueger et al., 2002), as well as childhood antisocial disorders (ODD, CD) and ADHD (King, Iacono, & McGue, 2004). A number of studies have found that these disorders share an etiological basis (e.g., Krueger et al., 2002; Krueger & South, 2009) and that their genetic predisposition may become stronger in adverse environments (Hicks et al., 2009). High Neuroticism is associated with Externalizing, as are high Extraversion, low Agreeableness, and low Conscientious (DeYoung et al., 2008; Settles et al., 2012). Externalizing is also negatively associated with cognitive ability (DeYoung et al., 2008; Finn et al., 2009).

Neuroticism is consistently connected with various Internalizing disorders (e.g., Griffith et al., 2010; Hettema et al., 2006; Krueger 1999), as well as Externalizing disorders (e.g., DeYoung et al., 2008; Settles et al., 2012). Although few studies have examined the aspects of Neuroticism in relation to Internalizing and Externalizing, the Internalizing disorders share substantial variance and are closely related to N-Withdrawal. Externalizing is broader than N-Volatility; it correlates with other Big Five traits, such as low Agreeableness and low Conscientiousness. However, it also encompasses angry hostility and outward expression of negative affect, which characterize N-Volatility. In relation to psychopathology, its most relevant traits may be aggression and impulsivity, both of which have links to N-Volatility (DeYoung et al., 2007; Krueger et al., 2007).

1.4 Neuroticism and Higher Cognitive Functions

The aspects of Neuroticism have not often been separated in existing literature, and they may affect cognitive and affective processes in different ways. This may explain why Neuroticism has contradictory associations with some traits. The present research focuses on clarifying the relation of Neuroticism to complex cognitive functions, including intelligence and creativity. There are some conflicting findings with regard to the associations between Neuroticism and the latter traits; separating the aspects could explain some of these findings. In addition, studying the links between cognitive traits and psychopathology could point toward mental processes that they share. For example, although the neuroscience underlying creativity is not fully understood, it seems that the cognitive processes that facilitate creativity may also facilitate pathological traits (Dietrich, 2004; Dietrich, 2007; Rybakowski et al., 2008). Since distilling disorders to their defining characteristics can help uncover the underlying neural subsystems, it stands to reason that other traits that correlate with psychopathology may also help illuminate these disorders. As intelligence and creativity are two traits that are linked to Neuroticism and have a significant impact on healthy life outcomes, they bear examination in closer detail.

2.1 Study 1: Cognitive Ability and Neuroticism

There is a robust negative correlation between intelligence and psychopathology (Heinrichs & Zakzanis, 1998; Raine et al., 1992; Rund, 1998; Snitz, MacDonald, & Carter, 2006), and, again, because Neuroticism is a personality trait that correlates with many disorders, its relation to intelligence could illuminate the specific processes that are

risk factors or protective factors. Studying intelligence might point to ways to maximize its potential—leading to better life outcomes—and studying its link to psychopathology may help in developing interventions for those who are at risk. The personality trait most closely associated with intelligence—Openness/Intellect—has a complicated relationship with psychopathology; the aspects of Openness/Intellect relate to Neuroticism in opposite directions (DeYoung, Grazioplene, & Peterson, 2012). This may account in part for the popular perception of the relationship between "genius" and "madness" (DeYoung et al., 2012); although intelligence negatively correlates with psychopathology, this relationship may not hold true for the aspects of O/I. For example, psychoticism, a pathological trait that associates with psychotic disorders, is positively correlated with O-Openness, but is negatively correlated with O-Intellect; because of this, its association with the higher-level trait of Openness/Intellect was suppressed (DeYoung, 2014). Separating the aspects of Neuroticism may also shed more light on its connections with other traits.

The first study examined the relationship between Neuroticism and performance on various cognitive tasks, including the Wechsler Adult Intelligence Scale (WAIS) and Raven Progressive Matrices. The goal of study one was to determine the relationship between *trait* Neuroticism—as opposed to *state* neuroticism, or having a current mood that is more or less anxious—on cognitive performance. State neuroticism is well established to have a non-linear relationship with cognitive performance (the relationship resembles an inverted u-shaped curve; e.g., Beckman, 2013), but the relationship between trait Neuroticism and cognitive performance—as well as intelligence more generally—is not as clearly understood.

Neuroticism has a weak negative correlation with intelligence (DeYoung et al., 2014), but few studies have tested for a non-linear relationship. Non-linear relationships require relatively larger samples in order to find effects, particularly if the effects are small (Major, Johnson, & Deary, 2014). In one study, the authors expected to find a quadratic effect of g on a measure of emotional stability (or reverse Neuroticism), but found only a linear association in males, and no association (linear or non-linear) in females (Major et al., 2014). Another study tested the non-linear effects of Neuroticism on intelligence, but found the linear model was a better fit (Moutafi, Furnham, & Paltiel, 2005). However, neither of these studies separated Neuroticism into its aspects; if the aspects affected intelligence or cognitive performance in different ways, each would effectively mask the associations of the other. Therefore, the current study examined the aspects separately.

In defining intelligence, many—including an APA task force—have emphasized a psychometric approach; although individuals often perform differently on different measures, the common factor g can be extracted, which may be the best fundamental measure of intelligence (Neisser et al., 2006). Tasks directly related to intelligence include various cognitive abilities, such as problem-solving, pattern recognition, inductive and deductive reasoning, and ability to learn from experience (Neisser et al., 1996). The general intelligence factor (g) accounts for more than 50% of intelligence-test variance, an effect that is stronger for below-average ability subjects (Deary et al., 1996). This general factor is probably the best operationalized measure of intelligence.

In terms of personality, intelligence most closely relates to Openness/Intellect

(DeYoung et al., 2007; DeYoung, 2011; DeYoung et al., 2014). Data from a meta-analysis are consistent with intelligence as a facet of O/I (DeYoung, 2011). O-Intellect correlates with both general intelligence and specifically verbal and nonverbal intelligence (DeYoung et al., 2014). Performance on both verbal and nonverbal intelligence tasks significantly correlate with O/I, and no other personality factor (DeYoung, Peterson, & Higgins, 2005). Although intelligence is protective against almost all forms of psychopathology, the relationship between the latter and O/I is not as straightforward. Neuroticism correlates negatively with the Intellect aspect of O/I, and correlates positively with the Openness aspect (DeYoung, Grazioplene, & Peterson, 2012). Although intelligence negatively correlates with psychopathology, that may not hold true for O/I. The trait psychoticism, as measured by the Personality Inventory for the DSM-V (PID, a measure of diagnostic traits relating to personality disorders), shows a modest positive relationship with O/I (Watson et al., 2013). However, studies have consistently found a negative relationship between psychotic disorders (such as schizophrenia and schizotypal personality disorder) and cognitive ability (Heinrichs & Zakzanis, 1998; Raine et al., 1992; Rund, 1998; Snitz, MacDonald, & Carter, 2006). These findings may be explained by variance specific to the aspects of Openness/Intellect.

Bipolar disorder positively correlates with O-Intellect and shows no correlation with intelligence, even when it includes psychotic features (Quilty et al., 2013; Zammit et al., 2004). Subjects with bipolar disorder do suffer from certain cognitive deficits (e.g., Daban et al., 2012; Robinson et al., 2006). One large cohort study found that men with

low intelligence were at the highest risk for developing bipolar disorder, but men with high intelligence also had an elevated risk; however, the risk was only for the disorder without any other comorbid disorders (Gale et al., 2013). Bipolar disorder, depression, and schizophrenia, share significant genotypic and phenotypic variance, and it is difficult to study them in isolation, considering that their frequent comorbidity most likely reflects their shared etiology.

Externalizing disorders are characterized by decreased cognitive ability, as well as by high Extraversion, low Agreeableness, and low Conscientiousness. Externalizing behavior relates specifically to reward-seeking and novelty-seeking—which typify Extraversion and O/I (Golimbet et al., 2007; Roberti, 2004)—while also relating to poor impulse control and lack of behavioral inhibition, which correlates negatively with intelligence (Alderson et al., 2010; Jurado & Rosselli, 2007; Kerns, McInerney, & Wilde, 2001). Subjects with Externalizing disorders such as ADHD and anti-social personality disorder (ASPD) consistently score more poorly on intelligence measures (e.g., Bridgett & Walker, 2006; Kuntsi et al., 2004; Vitacco, Neumann, & Jackson, 2005).

The relationship between Internalizing and intelligence is more ambiguous. Lower intelligence is a risk factor for the development of depression and anxiety, but the mechanism is poorly understood (Gale et al., 2009). Rumination, or the propensity to dwell on a thought, idea, or emotion, is frequently observed in Internalizing disorders such as major depressive disorder or various anxiety disorders (Mellings & Alden, 2000; Michel et al., 2013; Muris et al., 2005; Nolen-Hoeksema, 2000). It would be logical to assume that ruminating, as a central symptom in Internalizing disorders, would have an

effect on cognitive ability, if only because ruminating is a specialized form of cogitating. Studies have also been mixed in this regard: while some have posited that rumination (and therefore Internalizing) enhances problem solving abilities, others have suggested that rumination will restrict working memory by increasing cognitive load, and therefore inhibit the ability to focus on other cognitive problems.

A number of studies have demonstrated that depression, anxiety, and rumination harm performance on cognitive tasks across multiple domains, including social problem-solving (e.g., Watkins & Baracaia, 2002), executive functions (e.g., Austin, Mitchell, & Goodwin, 2001), and memory (e.g., Burt, Zembar, & Niederehe, 1995), as well as learning and decision making (e.g., McAllister, 1981; Murphy et al., 2001). However, other studies have indicated that under certain conditions, subjects with depression can perform as well or better as unimpaired subjects (e.g., Ambady & Gray, 2002; Au et al., 2003; Yost & Weary, 1996). Like most personality traits, rumination is best viewed as a spectrum, and those with Internalizing disorders would be at the far end of the ruminating spectrum. For those in the middle of the spectrum, rumination may improve complex problem solving abilities, whereas, at the extreme end, it would interfere and be associated with psychopathology. Thus, at a population level, decreased intelligence is a risk factor for Internalizing disorders; however, up to a certain level, rumination may have a positive effect on cognitive performance, which suggests a nonlinear association.

2.2 Hypotheses

Considering the contradictory findings related to intelligence, cognitive performance, and Neuroticism, it follows that separating the aspects of Neuroticism may

shed light on the true relationship. Just as the O-Openness and O-Intellect aspects relate differently to different forms of psychopathology, N-Volatility and N-Withdrawal may relate to cognitive performance differently. N-Withdrawal is more strongly connected to Internalizing disorders, as well as their characteristic symptom, rumination; these have shown conflicting associations with cognitive performance in prior research. N-Volatility is more associated with the Externalizing disorders, which consistently have a negative effect on cognitive performance and intelligence. Therefore, N-Volatility is predicted to have an overall negative association with various cognitive performance measures.

Many general measures of Neuroticism have emphasized N-Withdrawal more than N-Volatility, and N-Withdrawal is also more closely aligned with common definitions of "state" Neuroticism (the tendency to feel emotions like anxiety; rumination; internal expressions of negative affect). One way to explain the contradictory findings regarding trait Neuroticism and cognitive performance would be through a non-linear relationship, similar to what has been observed with state neuroticism. Thus, N-Withdrawal is predicted to have a curvilinear relationship with performance on cognitive measures, resembling the inverted-u-shaped curve seen for state neuroticism (Figure 1).

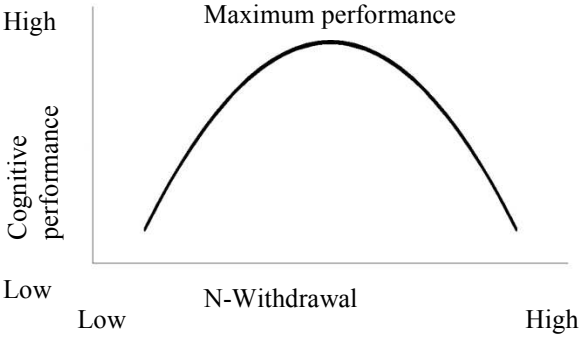

Figure 1. Curvilinear relationship predicted between cognitive performance and N-Withdrawal. Performance is highest for those in the middle of the spectrum, as opposed to either end.

2.3 Methods

2.3.1 Participants.

Data were used from several preexisting data sets. Sample 1 consists of 234 males recruited from Yale and the surrounding New Haven community, including nearby colleges. The age range of Sample 1 was between 18 and 40 (M=23.6, SD=5.0), and approximately half of the sample was students; the rest had a range of occupations and incomes (see DeYoung et al., 2011, sample 2, for more details on the collection and demographics of this sample).

Sample 2 included 166 students (51 males and 115 females) attending a selective high school program in Cambridge, England; their ages ranged from 16-18 (M=16.9, SD=0.7; see Kaufman et al., 2009, for more information on the collection and demographics of this sample).

Sample 3 includes 305 subjects (151 males and 154 females) recruited from the Minneapolis/St. Paul community, primarily through Craigslist and other forms of internet advertising. The sample was primarily Caucasian (72%) and contained a variety of professions, with relatively few students (12%). The ages of the participants ranged from 20-40 (M=26.2, SD=5; see Kaufman et al., 2015 for more information on the collection and demographics of this sample).

2.3.2 Measures.

Personality. Personality was assessed using the Big Five Aspects Scale (BFAS; DeYoung et al., 2007). The BFAS assesses personality at the level of the Big Five as well as the two aspects within each Big Five trait. The Big Five themselves have been

repeatedly empirically confirmed across studies, and are widely accepted in personality research (Digman, 1990). The Big Five traits can also be broken down into facets, or specific components of each trait—for example trust, modesty, and altruism are facets of agreeableness from the NEO-PI-R scales (Costa & McCrae, 1992). Naturally, each trait can be partitioned into any number of narrower components. However, one empirically based factor solution, analyzing 15 facets within each of the Big Five, suggested that each trait consists of two correlated, but distinct, aspects (DeYoung, 2007). When these aspects were correlated with genetic factor scores, the results suggested that each aspect relates to distinct genetic substrates, implying a biological basis for the factor segregation (DeYoung, 2007). The aspect scales on the BFAS were used to assess N-Volatility and N-Withdrawal.

Intelligence. The WAIS is one of the most prominent intelligence measures, and its validity and reliability have been repeatedly demonstrated. Reliability estimates range from 0.7-0.9, and it also correlates highly with the Stanford-Binet test (Wechsler, 2008). It was originally designed as an intelligence test, and it measures cognitive performance in adults across a number of domains (Wechsler, 1958). In samples 1 and 3, the WAIS was used as a measure of cognitive performance. In sample 2, the calculated latent variable "g" was used as one measure of general cognitive performance. Latent "g' was calculated using tasks from three domains of cognitive ability: verbal, mental rotations, and perceptual. The three tasks were the Differential Aptitudes Test (DAT; the verbal subtests were used), the Mental Rotations Test (MRT; rotation; this had a significant correlation of .43 with the DAT), and the Raven's advanced progressive matrices test

(perceptual; this had a significant correlation of .53 with the DAT and .59 with the MRT; Kaufman et al., 2009). The Raven Progressive Matrices is often used as a measure of general cognitive ability, designed as a shorter alternative to the WAIS (Raven, 2003); it was also used as a measure of cognitive performance in sample 2.

2.4 Analysis.

WAIS was used as a measure of cognitive performance in sample 1 (M=121.9, SD=11.7) and sample 3 (M=113.6, SD=15.4). In sample 2, the overall measure of "g" was used (M=0, SD=0.9), along with the Raven advanced progressive matrices test (M=21.7, SD=5.4), as WAIS scores were unavailable. The Raven test is a good measure of general cognitive performance on a specific task, as well as a good marker of g; g is a latent variable representing shared variance in cognitive performance across several different tasks.

The three samples were analyzed separately, using the same model. First, scores for N-Withdrawal and N-Volatility were centered at zero to avoid collinearity with quadratic terms; they were then squared in order to calculate their quadratic term. Scores on the cognitive tasks were then regressed onto N-Volatility and N-Withdrawal, along with age and gender (except in sample 1, the all-male sample). In samples 2 and 3, the interaction between gender and the linear and quadratic terms for N-Withdrawal was also tested for significance. In sample 1, which included students attending Yale as well as members of the surrounding community, Yale-student status was also included in each model.

Each regression had three blocks. The first block included N-Withdrawal, N-Volatility, and the covariates; the second block included those variables along with the

quadratic term for N-Withdrawal; and the third block included all variables in the second block along with the quadratic term for N-Volatility. For each block, the significance of the change in the R^2 value was calculated, which tested the significance of the addition of the quadratic variables to the model.

2.5 Results

Table 1. Regression Model for WAIS Scores, Sample 1.

Block 1	Parameter	B	Standard Error	Standardized β	t	*p*-value
	Age	0.063	0.136	.027	0.467	.641
	Yale Status	**14.395**	**1.669**	**.497**	**8.623**	**<.001**
	N-Volatility	**-3.496**	**1.076**	**-.229**	**-3.250**	**.001**
	N-Withdrawal	2.055	1.125	.129	1.827	.069
Block 2						
	Age	0.064	0.136	.027	0.472	.637
	Yale Status	**14.390**	**1.674**	**.497**	**8.598**	**<.001**
	N-Volatility	**-3.486**	**1.082**	**-.229**	**-3.221**	**.001**
	N-Withdrawal	2.031	1.148	.127	1.770	.078
	N-Withdrawal (Quadratic)	0.107	0.970	.006	.110	.912
Block 3						
	Age	0.064	.136	.027	0.470	.638
	Yale Status	**14.527**	**1.680**	**.502**	**8.646**	**<.001**
	N-Volatility	**-3.608**	**1.090**	**-.237**	**-3.310**	**.001**
	N-Withdrawal	2.089	1.150	.131	1.817	.071
	N-Withdrawal (Quadratic)	-0.243	1.038	-.014	-.234	.815
	N-Volatility (Quadratic)	0.979	1.028	.057	.952	.342

Note. Bolded estimates are significant at a level of $p < 0.05$.

Model Significance: R^2 Change

Block	R	R^2	Standard Error	R^2 Change	F-Change	*p*-value
1	.326	.106	1.85705	.106	6.873	<.001
2	.327	.107	1.86081	.000	0.066	.797
3	.327	.107	1.86477	.000	0.025	.875

Table 2. Regression Model for g, Sample 2.

Block 1	Parameter	B	Standard Error	Standardized β	t	*p*-value
	Age	.110	.108	.078	1.025	.307
	Sex	**-.440**	**.148**	**-.231**	**-2.983**	**.003**
	N-Volatility	**-.242**	**.106**	**-.210**	**-2.285**	**.024**
	N-Withdrawal	**.269**	**.120**	**.212**	**2.249**	**.026**
Block 2						
	Age	.121	.107	.086	1.130	.260
	Sex	**-.479**	**.147**	**-.251**	**-3.252**	**.001**
	N-Volatility	**-.225**	**.105**	**-.195**	**-2.136**	**.034**
	N-Withdrawal	**.274**	**.119**	**.215**	**2.309**	**.022**
	N-Withdrawal (Quadratic)	**-.187**	**.091**	**-.154**	**-2.046**	**.042**
Block 3						
	Age	.122	.107	.086	1.142	.255
	Sex	**-.487**	**.147**	**-.256**	**-3.309**	**.001**
	N-Volatility	**-.243**	**.106**	**-.210**	**-2.284**	**.024**
	N-Withdrawal	**.265**	**.119**	**.209**	**2.238**	**.027**
	N-Withdrawal (Quadratic)	**-.246**	**.104**	**-.203**	**-2.372**	**.019**
	N-Volatility (Quadratic)	.120	.100	.104	1.198	.233

Note. Bolded estimates are significant at a level of $p < 0.05$.

Model Significance: R^2 Change

Block	R	R^2	Standard Error	R^2 Change	F-Change	*p*-value
1	.303	.092	.851	.092	4.071	.004
2	.339	.115	.842	.023	4.187	.042
3	.351	.123	.841	.008	1.434	.233

Table 3. Regression Model for Ravens Progressive Matrices, Sample 2.

Block 1	Parameter	B	Standard Error	Standardized β	t	*p*-value
	Age	0.447	0.661	0.052	0.676	.500
	Sex	-1.630	0.907	-.0141	-1.798	.074
	N-Volatility	**-1.559**	**0.652**	**0-.223**	**-2.393**	**.018**
	N-Withdrawal	**1.944**	**0.736**	**0.252**	**2.641**	**.009**

Block 2	Parameter	B	Standard Error	Standardized β	t	*p*-value
	Age	0.507	0.657	0.059	0.773	.441
	Sex	**-1.858**	**0.906**	**-0.161**	**-2.050**	**.042**
	N-Volatility	**-1.458**	**0.648**	**-0.208**	**-2.250**	**.026**
	N-Withdrawal	**1.970**	**0.730**	**0.255**	**2.699**	**.008**
	N-Withdrawal (Quadratic)	**-1.093**	**0.562**	**-0.149**	**-1.947**	**.053**
Block 3						
	Age	0.511	0.658	0.060	.777	.439
	Sex	**-1.885**	**0.909**	**-0.163**	**-2.073**	**.040**
	N-Volatility	**-1.514**	**0.656**	**-0.217**	**-2.309**	**.022**
	N-Withdrawal	**1.943**	**0.732**	**0.252**	**2.653**	**.009**
	N-Withdrawal (Quadratic)	**-1.283**	**0.641**	**-0.175**	**-2.002**	**.047**
	N-Volatility (Quadratic)	0.382	0.616	0.055	.619	.537

Note. Bolded estimates are significant at a level of $p < 0.05$.

Model Significance: R^2 Change

Block	R	R^2	Standard Error	R^2 Change	F-Change	*p*-value
1	.259	.067	5.227	.067	2.906	.023
2	.298	.089	5.183	.022	3.789	.053
3	.302	.091	5.193	.002	0.384	.537

Table 4. Regression Model for WAIS Scores, Sample 3.

Block 1	Parameter	B	Standard Error	Standardized β	t	*p*-value
	Age	-0.156	.174	-.051	-.893	.373
	Gender	0.951	1.753	.031	.543	.588
	N-Volatility	**-3.613**	**1.310**	**-.192**	**-2.757**	**.006**
	N-Withdrawal	**3.883**	**1.480**	**.182**	**2.623**	**.009**
Block 2						
	Age	-0.182	.173	-.060	-1.054	.293
	Gender	0.965	1.735	.031	.556	.578
	N-Volatility	**-3.696**	**1.298**	**-.196**	**-2.849**	**.005**
	N-Withdrawal	**4.489**	**1.483**	**.211**	**3.027**	**.003**
	N-Withdrawal (Quadratic)	**-3.492**	**1.306**	**-.153**	**-2.674**	**.008**

Block 3	Parameter	B	Standard Error	Standardized β	t	*p*-value
	Age	-.0182	.173	-.060	-1.052	.294
	Gender	0.983	1.741	.032	.565	.573
	N-Volatility	**-3.777**	**1.386**	**-.200**	**-2.726**	**.007**
	N-Withdrawal	**4.524**	**1.500**	**.212**	**3.015**	**.003**
	N-Withdrawal (Quadratic)	**-3.562**	**1.373**	**-.156**	**-2.594**	**.010**
	N-Volatility (Quadratic)	0.204	1.221	.011	.167	.867

Note. Bolded estimates are significant at a level of $p < 0.05$.

Model Significance: R^2 Change

Block	R	R^2	Standard Error	R^2 Change	F-Change	*p*-value
1	.191	.036	15.257	.036	2.829	.025
2	.243	.059	15.103	.022	7.148	.008
3	.243	.059	15.128	.000	0.028	.867

N-Volatility was expected to have an overall negative relationship with cognitive performance, and no non-linear relationship was expected. In all three samples, this was the case (Tables 1-4). The quadratic term for N-Volatility was not significant in any of the models and did not cause a significant change in the R^2 of the models.

N-Withdrawal was expected to have a significant non-linear relationship with cognitive performance, and this was the case in two of the samples. In sample 1, the linear term for N-Withdrawal was not significant ($p = .069$). The addition of the quadratic term did not cause a significant change in the R^2 value of the model ($p = .797$). Results were not different in sample 1 when Yale-student status was excluded from the analysis. In the other two samples, N-Withdrawal had a significant non-linear relationship with cognitive performance in at least one of the models. The non-linear model resembles that seen in models of the relationship between state neuroticism (or anxiety) and cognitive

performance; it is an inverted u-shaped curve, where subjects in the middle perform better than subjects on either extreme (Figures 2 and 3). In samples 2 and 3, gender did not have a significant interaction effect with either the linear or quadratic term for N-Withdrawal.

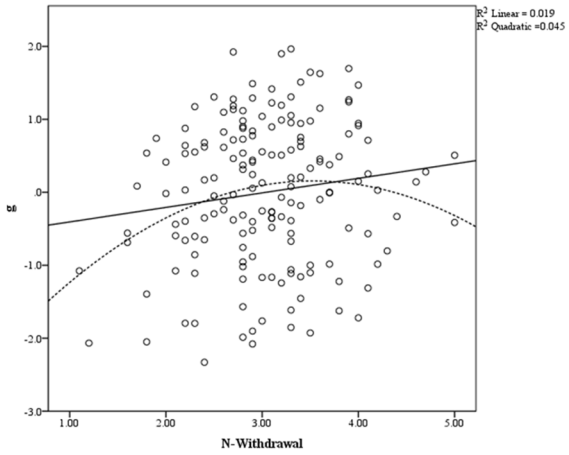

Figure 2. Linear and quadratic relation between g and N-Withdrawal in sample 2, after controlling for age, gender, and N-Volatility.

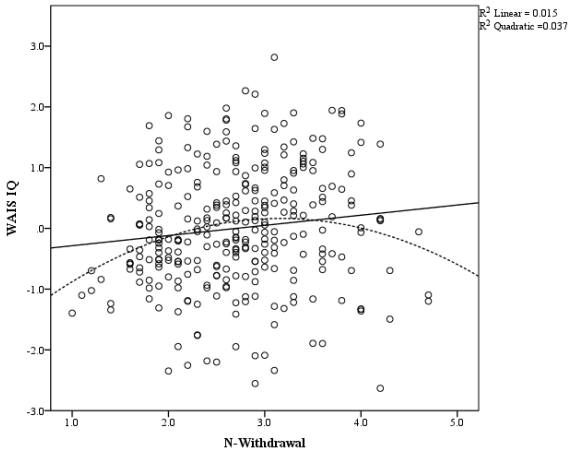

Figure 3. Linear and quadratic relation between WAIS IQ and N-Withdrawal in sample 3, after controlling for age, gender, and N-Volatility.

2.6 Discussion

In this study, the advantages of non-linear modeling are apparent, as these models better explain the relationship between N-Withdrawal and cognitive performance than linear models in two of the samples. The "inverted u-shaped curve" has been well-replicated as a representation of the relationship between state neuroticism—current feelings of anxiety or negativity in the moment—and cognitive performance. State neuroticism, anxiety, or arousal is helpful up until a certain point with cognitive performance—as a certain level of engagement is necessary to maximize performance—

but past a certain threshold it interferes. As Neuroticism represents a pattern of thinking, feeling, and behaving in particular ways, replicating the effects of state neuroticism might be expected. The state of mind that corresponds to higher anxiety more often occurs in a person who is higher in N-Withdrawal as a trait. The current results suggest that N-Withdrawal is the principal driver of this effect; N-Volatility has an overall negative impact on cognitive performance that is not better explained through a non-linear model. It also reinforces the importance of separating the aspects; if Neuroticism was analyzed as a whole, it would mask the effects of the aspects and show little to no relationship. This may help explain some of the previous conflicting findings as to the relationship between Neuroticism and cognitive performance.

The non-linear model of N-Withdrawal also may explain contradictory findings, particularly with regard to Internalizing disorders, as well as a core feature, rumination. Rumination is closely related to N-Withdrawal; subjects who score more highly on measures of N-Withdrawal would have a tendency to ruminate more often. Previous studies have been mixed as to whether rumination aids or interferes with cognitive performance. The non-linear relationship between N-Withdrawal and cognitive performance would help explain these results. Rumination would have a positive association with problem solving, but only up to a point. This would also explain the negative association between intelligence and Internalizing disorders, which would generally include people at the extreme end of the N-Withdrawal spectrum.

Sample 1 unexpectedly showed no relationship between N-Withdrawal and cognitive performance; there was no significant linear or non-linear effect. Further,

gender did not show a significant interaction in the other samples; therefore, the lack of effect is not due to the fact that sample 1 is all male. However, sample 1 did show the same relationship between N-Volatility and cognitive performance. N-Volatility consistently had a negative, non-linear relationship with cognitive performance, which is in line with previous findings. Many studies examining the inverse relationship between intelligence and psychopathology have investigated schizophrenia and psychotic spectrum disorders (e.g., Khandaker et al., 2011) or Externalizing disorders (e.g., Bridgett & Walker, 2006), the latter of which is more associated with N-Volatility. Bipolar disorder is associated with lower cognitive processing speed as well, to the point where it has discriminant validity between patients with bipolar disorder, their unaffected first-degree relatives, and the general population (Daban et al., 2012). Unlike N-Withdrawal, where cognitive performance is maximized in the middle of the spectrum, N-Volatility has a uniformly negative effect.

Both cognitive performance and the aspects of Neuroticism can be viewed as endophenotypes for different forms of psychopathology. The concept of endophenotypes were initially developed in order to bridge the gap between symptoms and genetics and to measure more stable behavioral phenotypes. Cognitive endophenotypes related to working memory and processing speed have been used to relate neural processes to specific genes and specific disorders (e.g., Leiser et al., 2009; Wedenoja et al., 2008). Initially, endophenotypes were intended to link to specific genes, as this had proved difficult with disorders; this effort generally did not succeed. However, endophenotypes proved to have clinical use in their discriminant validity between patients and healthy

controls (e.g., Daban et al., 2012). N-Withdrawal and N-Volatility may also serve as endophenotypes; they are a straightforward phenotype that can be assessed reliably, and they relate to genetic variance (DeYoung et al., 2007).

Separating the aspects is particularly important in identifying the etiology of different types of psychopathology, as N-Withdrawal and N-Volatility are hypothesized to represent different underlying neural processes. Further, their associations with cognitive performance suggest particular neural processes that may be involved in the disorders, and may, therefore, provide the neural basis of the relationship between psychopathology and intelligence. The effect of N-Volatility on cognitive performance suggests that the former may explain some of the basis of the negative association between intelligence and Externalizing disorders. The aspects can also account for some of the overlap and comorbidity in different types of psychopathology; for example, N-Withdrawal as an endophenotype reflects the neural substrate of shared variance in Internalizing disorders.

The effect of N-Withdrawal on cognitive performance helps explain previous findings related to IQ and Internalizing disorders, specifically depression, and suggests a possible non-linear relationship, which was demonstrated in two of the three samples tested. As with psychotic disorders, where traits like apophenia—or the tendency to see patterns and meaningful connections in random data—encourage creativity if they stop short of full disorder, which then interferes with creativity (DeYoung, Grazioplene, & Peterson, 2012; Michalica, 2010), N-Withdrawal may enhance cognitive performance until a certain point, after which the disorder such as depression interferes with cognitive

performance. On the other hand, N-Volatility has a uniformly negative association with cognitive performance; this is similar to cognitive processing endophenotypes associated with disorders such as schizophrenia and bipolar, where there is a linear negative relationship between particular features associated with the disorder and cognitive performance on various tasks.

3.1 Study 2: Creativity and Neuroticism

Creativity is most strongly associated with the Big Five trait Openness/Intellect, as is intelligence (Batey, Furnham, & Safiullina, 2010; King, Walker, & Broyles, 1996; Silvia et al., 2009). Unlike intelligence, creativity has a stronger link to the O-Openness aspect, as opposed to O-Intellect (Nusbaum & Silvia, 2011a); however, this may hold true only for artistic creativity, rather than scientific creativity. Kaufman et al. (2015) found that O-Openness predicts creative achievement in the arts, while O-Intellect predicts creative achievement in the sciences. When the aspects are separated, O-Openness predicts artistic creativity, but not fluid intelligence, and the reverse is true for O-Intellect (Nusbaum & Silvia, 2011a). Unsurprisingly, intelligence is also a good predictor of creativity, although the two also have unshared variance (Batey, Furnham, & Safiullina, 2010; Nusbaum & Silvia, 2011b).

Creativity is commonly measured with divergent thinking tasks, self-reports of creative achievement or behavior, and personality measures (e.g., Batey, Furnham, & Safiullina, 2010; Benedek et al., 2012; Kéri, 2009). The second study will examine the relationship between Neuroticism and creativity, using the Creative Achievement Questionnaire (CAQ) as the primary measure of creativity. Creativity can be difficult to

measure, but the CAQ has a high degree of reliability, predictive validity, and convergent validity with other creativity measures, and is comparatively easier to score (Carson, Peterson, & Higgins, 2005). Although it is a self-report measure, the CAQ seems to accurately measure real world creative achievement (Carson, Peterson, & Higgins, 2005).

Creativity has shown a contradictory empirical relationship with several forms of psychopathology (c.f. de Manzano et al., 2010; Rybakowski et al., 2008 as opposed to Dietrich, 2007). Some forms of psychopathology have shown positive associations with creativity measures (Acar & Runco, 2012; Andraesen, 1987; De Pauw & Mervielde 2010). Further, specific white matter architecture in the frontal cortex is associated, in normal subjects, with variance in divergent thinking (a common measure of creativity) as well as variance in psychotic traits (Jung et al., 2010). However, other measures have shown a negative relationship between creativity and psychopathology (Dietrich, 2007).

These results may be partially explained by personality's role as a mediating variable, with a personality profile that corresponds to higher creativity also corresponding to an increased risk of psychopathology (Barrantes-Vidal, 2004; Chavez-Eakle, Lara, & Cruz-Fuentes, 2005); however, the psychopathology itself interferes with creativity, leading to conflicting findings. The cognitive processes that facilitate creativity also facilitate pathological traits, in that the same processes that make creative thought occur more often may also make pathological ways of thinking more likely (Dietrich, 2004; Dietrich, 2007; Rybakowski et al., 2008). People who have certain facets of psychoticism (such as apophenia or magical thinking) may be more creative, up to a certain threshold—for those with full-blown schizophrenia or other psychotic disorders,

their psychotic traits impair creativity (e.g., Michalica, 2010). This explains why traits related to the psychotic spectrum are positively associated with creativity, while psychotic disorders themselves may not be.

The mediating role of personality is supported by the fact that subjects with bipolar disorder still score highly on creativity measures when they are successfully treated (Andraesen & Glick, 1988; Belmaker, 2004). This suggests that the symptoms of the disorder themselves are not driving increased creativity; rather, a third variable accounts for both creativity and symptomatology or mediates the relationship between the two. Features of certain disorders may be beneficial for creativity, but may impair creativity in subjects who are over the threshold for a disorder; this could also explain why treating the symptoms of a disorder does not affect creativity scores. However, the role of personality is further complicated by the etiological connections between different disorders. Although disorders can share similar genetic and neural substrates, the disorders themselves are associated with creativity in different ways.

Bipolar disorder shares variance with schizophrenia and psychotic disorders, as well as with depression (e.g., Akiskal et al., 2010; Bramon & Sham, 2001; Berrettini, 2001; Huang et al., 2010). Both bipolar and depression can present with psychotic features, and they are so frequently comorbid with schizophrenia that a separate diagnostic category—schizoaffective disorder—was created to describe the disorders presenting together (APA, 2013). The disorders share both phenotypic variance—common outward symptoms (APA, 2013)—and underlying genetic variance (e.g., McGuffin et al., 2003; Purcell et al., 2009). Major depressive disorder shows substantial

overlap with other Internalizing disorders, especially anxiety disorders (e.g., Fergusson, Boden, & Horwood, 2011; Seligman & Ollendick, 1998; Wolitzky-Taylor et al., 2010). Finally, both bipolar disorder and psychotic disorders are often comorbid with anxiety-related Internalizing disorders (Achim et al., 2011; Akiskal et al., 2010; Buckley et al., 2009; Cosoff & Hafner, 1998), the latter of which again generally show no or a slightly negative association with creativity (Silvia & Kimbrel, 2010).

To return to the personality profile underlying creativity, O/I (and specifically O-Openness) is the personality trait with the strongest link; however, Neuroticism and its aspects may also be related. A meta-analysis of creativity research noted that artistically creative subjects tended to be more hostile and impulsive—components of N-Volatility—compared to subjects who were higher on scientific creativity (Feist, 1998). Bipolar disorder is more strongly associated with N-Volatility, while depression is more strongly associated with N-Withdrawal (e.g., Quilty et al., 2012); bipolar disorder has shown a positive association with creativity, while depression has shown no or a slight negative association (Andraeson, 1987; Landen, 2012; Santosa et al., 2007; Silvia & Kimbrel, 2010; Srivastava et al., 2010). The personality profile associated with higher creativity could be associated with higher Neuroticism (particularly N-Volatility), and therefore with a higher risk for certain types of psychopathology (such as bipolar disorder).

3.2 Hypotheses

Setting aside the complex etiological relationship between affective disorders, anxiety disorders, and psychotic disorders, it is likely that N-Volatility will show a positive relationship with creativity, while N-Withdrawal will show a negative or non-

association with creativity, as is the case with Internalizing disorders. The literature suggests that not only do the aspects of Neuroticism correlate differently with creativity, but the different domains of creativity may associate differently as well. One large-scale study—using one of the samples from the current study—found no relationship between either artistic or scientific creative achievement and Neuroticism (Kaufman et al., 2015). However, this study did not examine the aspects of Neuroticism separately as simultaneous predictors, which could lead suppressive effects, as associations with the individual aspects could be suppressed by their shared variance.

Building on previous research that looked at specific types of psychopathology and their association with creativity, it is possible that their underlying features—that is, N-Volatility and N-Withdrawal—could account for some of this association and predict artistic and scientific creativity in similar ways. Therefore, N-Volatility should have a positive association with artistic creativity, while N-Withdrawal should have no association, or a slightly negative association, mirroring the relationship between bipolar and unipolar depression with creativity. However, N-Volatility should have a negative association with *scientific* creativity, which is more associated with O-Intellect and intelligence (Kaufman et al., 2015)—and thus may relate negatively to N-Volatility, as shown in study 1—while N-Withdrawal should have no effect.

3.3 Methods

3.3.1 Participants

Study two used the same samples as study one, plus the addition of sample 4. Sample 4 consisted of 323 males and females recruited from the Toronto area, including

the University of Toronto and the University of Waterloo. Age in this sample ranged between 17 and 61 (M=20.7, SD=3.9), and subjects came from relatively diverse backgrounds (see Kaufman et al., 2015, for more details on the collection and demographics of these data).

3.3.2 Measures

Personality and Intelligence. Sample 4 only included WAIS scores for a small subset of the sample, which were used as a measure of intelligence in supplementary analyses. As before, samples 1 and 3 included the WAIS as an intelligence measure, while sample 2 used the latent variable "g". In all four samples, BFAS was used to measure the aspects of the Neuroticism and the aspects of Openness/Intellect.

Creativity. Creativity was assessed using the Creative Achievement Questionnaire (CAQ; Carson, Peterson, & Higgins, 2005). The CAQ is a self-report measure that examines achievement across 10 different creative domains. Subjects receive more points for higher levels of achievement; for example, in visual arts, subjects receive no points if they indicate that they "have no training or recognized talent in this area," receive one point if they "have taken lessons in this area," all the way up to seven points if their "work has been critiqued in national publications." Carson et al. (2005) demonstrated that the CAQ has predictive validity, convergent validity with other creativity assessments, and discriminant validity from measures of intelligence. The domains can be divided into several subsets; Carson et al. (2005) used a principal components analysis to identify a two factor solution. The factors were labeled as scientific creativity (including invention and science achievements) and artistic creativity (including drama, writing, humor,

music, visual arts, and dance achievements). Analyses for study 2 used separate scores for scientific and artistic creativity, along with scores on the overall CAQ, which also included achievements related to architecture (which was not included in a factor; Carson et al. found that architecture did not fit on either a two-factor or three-factor model of the CAQ), and culinary achievements (which were excluded from the science factor). However, the overall CAQ scores are biased in the direction of artistic creativity, as there are more items relating to artistic domains.

3.4 Analysis

All samples were analyzed separately, due to their different measures of intelligence. A generalized loglinear Poisson regression model was used, in which scores on the CAQ were regressed onto N-Volatility and N-Withdrawal, along with intelligence, O-Openness and O-Intellect, age, and gender (except in the all-male sample). In samples 1 and 3, WAIS IQ scores were used to represent intelligence; in sample 2, g—which was derived from a number of cognitive tests—was used. In sample 4, only a small subset of the sample included WAIS scores (N=125; total N=323), so there was no control for intelligence. The Poisson regression was used because of the skewed distribution of the CAQ (Figure 4). Mean scores and standard deviations for the CAQ and its artistic and scientific factors are shown in Table 5.

	Overall CAQ M (SD)	Artistic CAQ M (SD)	Scientific CAQ M (SD)
Sample 1	22.1 (17.0)	17.0 (15.3)	4.2 (5.7)
Sample 2	16.4 (13.2)	13.7 (12.7)	1.8 (2.8)
Sample 3	9.2 (6.0)	6.6 (4.9)	1.6 (1.5)
Sample 4	11.2 (11.0)	9.0 (9.8)	1.6 (2.7)

Table 5. Means and standard deviations of overall CAQ scores, artistic CAQ scores, and scientific CAQ scores in samples 1-4.

Regressions were performed using the Huber/White/Sandwich linearized estimator, which is robust to violations of the assumptions of standard Poisson regression and allows analysis of logarithmically distributed data that do not contain independent observations (Breslow, 1990). All variables were entered simultaneously in each model. In all samples, three regressions were analyzed: the CAQ was as a whole, and then the artistic and scientific domains.

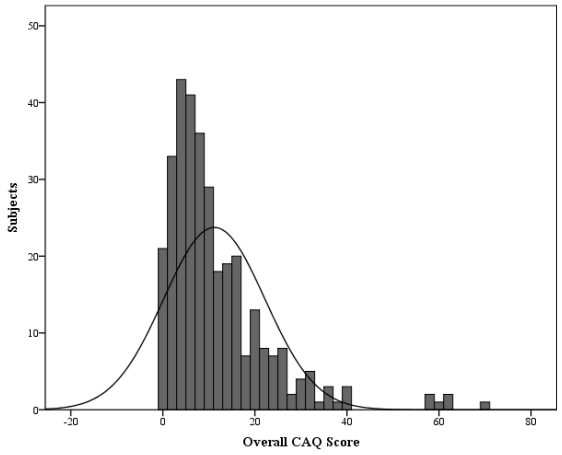

Figure 4. Poisson distribution of overall CAQ scores in sample 4.

3.5 Results

Table 6. Spearman's Rho for CAQ total, arts, and sciences scores correlated with variables in regression models.

Sample 1	CAQ Total	CAQ Arts	CAQ Sciences
CAQ Total	-	-	-
CAQ Arts	.906**	-	-
CAQ Sciences	.368**	.096	-
N-Volatility	.119	.138*	-.043
N-Withdrawal	-.035	-.022	-.087
O-Intellect	.263**	.198**	.214**
O-Openness	.488**	.472**	.169**
IQ	.081	-.026	.293**
Age	-.005	-.087	.043

Sample 2	CAQ Total	CAQ Arts	CAQ Sciences
CAQ Total	-	-	-
CAQ Arts	.946**	-	-
CAQ Sciences	.212**	-.004	-
N-Volatility	-.043	.015	-.150
N-Withdrawal	-.062	-.038	-.009
O-Intellect	.168*	.119	.315**
O-Openness	.313**	.351**	-.014
g	.000	-.042	.271**
Sex	.089	.184*	-.251**
Age	.024	.019	.054
Sample 3	CAQ Total	CAQ Arts	CAQ Sciences
CAQ Total	-	-	-
CAQ Arts	.953**	-	-
CAQ Sciences	.568**	.387**	-
N-Volatility	.056	.069	-.062
N-Withdrawal	.092	.102	-.010
O-Intellect	.203**	.149**	.297**
O-Openness	.429**	.447**	.149**
IQ	.186**	.150**	.216**
Age	-.117*	-.139*	-.128*
Gender	.236**	.185**	.303**
Sample 4	CAQ Total	CAQ Arts	CAQ Sciences
CAQ Total	-	-	-
CAQ Arts	.939**	-	-
CAQ Sciences	.454**	.210**	-
N-Volatility	-.018	.035	-.193**
N-Withdrawal	-.106	-.073	-.147**
O-Intellect	.241**	.174**	.250**
O-Openness	.299**	.302**	.077
Gender	.000	-.005	-.011
Age	-.086	-.110*	-.013

Note. *Correlation is significant at $p < .05$. **Correlation is significant at $p < .01$.

Table 6 shows Spearman's rank correlation coefficients between the CAQ and CAQ factors scores and the other variables in the regression model. N-Volatility was predicted to have a positive effect on scores on the Creative Achievement Questionnaire. This was the case in three of the four samples; Table 7 shows results from samples 1-4. The exception was with sample 2, which was on average younger than the other three samples; however, age did not seem to be associated with the number of creative achievements (Kaufman et al., 2015). Sample 2 consisted of only students, which was also true for sample 4, but not samples 1 and 3.

Table 7. Poisson Regression Model for Overall CAQ Scores.

Sample 1	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	N-Volatility	**.196**	**.0755**	**6.757**	**1**	**.009**
	N-Withdrawal	-.077	.0814	.902	1	.342
	O-Intellect	**.198**	**.1015**	**3.815**	**1**	**.051**
	O-Openness	**.473**	**.0919**	**26.551**	**1**	**<.001**
	WAIS IQ	-.006	.0056	1.122	1	.289
	Age	-.004	.0087	.234	1	.629
Sample 2						
	N-Volatility	-0.031	0.099	0.098	1	.754
	N-Withdrawal	-0.159	0.107	2.217	1	.136
	O-Intellect	0.132	0.119	1.241	1	.265
	O-Openness	**0.348**	**0.096**	**13.221**	**1**	**<.001**
	g	-0.065	0.072	0.813	1	.367
	Gender	-0.057	0.098	0.344	1	.557
	Age	-0.141	0.133	1.128	1	.288
Sample 3						
	N-Volatility	**0.076**	**0.029**	**6.978**	**1**	**.008**
	N-Withdrawal	-0.045	0.035	1.671	1	.196
	O-Intellect	0.035	0.039	0.796	1	.372
	O-Openness	**0.371**	**0.036**	**105.802**	**1**	**<.001**
	WAIS IQ	**0.005**	**0.001**	**11.987**	**1**	**.001**
	Age	0.000	0.003	0.001	1	.978
	Gender	**-0.259**	**0.039**	**45.275**	**1**	**<.001**

Sample 4	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	N-Volatility	**0.219**	**0.077**	**8.045**	**1**	**.005**
	N-Withdrawal	**-0.200**	**0.085**	**5.615**	**1**	**.018**
	O-Intellect	**0.215**	**0.101**	**4.543**	**1**	**.033**
	O-Openness	**0.283**	**0.092**	**9.486**	**1**	**.002**
	Age	**-0.035**	**0.017**	**4.567**	**1**	**.033**
	Gender	0.108	0.099	1.208	1	.272

Note. Bolded estimates are significant at a level of $p < 0.05$.

Figure 5 shows the log transformed overall CAQ scores in sample 1 correlated with N-Volatility, after controlling for age, O-Openness and O-Intellect, and WAIS scores; however, it should be noted that R^2 statistics do not exist in Poisson regression models, and the calculated R^2 value in the linear regression are skewed due to the non-normal distribution of CAQ scores.

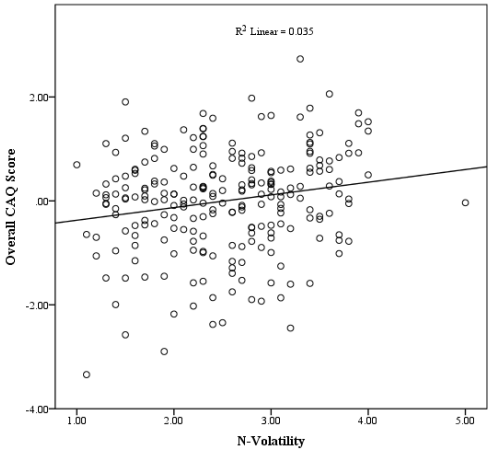

Figure 5. Correlation between log transformed overall CAQ score and N-Volatility in sample 1, after controlling for age, O-Openness and O-Intellect, and WAIS scores.

N-Withdrawal was predicted to have no association, or a slightly negative association, with overall creativity scores. This relationship was not significant in three of the samples, and there was a significant negative relationship between overall creative achievement and N-Withdrawal in sample 4.

Table 8. Poisson Regression Model for CAQ Artistic Scores.

Sample 1	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	N-Volatility	**0.220**	**0.082**	**7.147**	**1**	**.008**
	N-Withdrawal	-0.080	0.093	0.738	1	.390
	O-Intellect	0.149	0.113	1.738	1	.187
	O-Openness	**0.612**	**0.103**	**35.584**	**1**	**<.001**
	WAIS IQ	**-0.016**	**0.006**	**7.099**	**1**	**.008**
	Age	-0.016	0.011	2.271	1	.132
Sample 2						
	N-Volatility	**.333**	**0.087**	**14.783**	**1**	**<.001**
	N-Withdrawal	**-.280**	**0.093**	**9.019**	**1**	**.003**
	O-Intellect	.131	0.108	1.477	1	.224
	O-Openness	**.394**	**0.092**	**18.436**	**1**	**<.001**
	g	**-.044**	**0.020**	**4.744**	**1**	**.029**
	Age	**.333**	**0.087**	**14.783**	**1**	**<.001**
	Gender	.079	0.110	.512	1	.474
Sample 3						
	N-Volatility	0.093	.063	2.187	1	.139
	N-Withdrawal	-0.068	.069	0.965	1	.326
	O-Intellect	-0.056	.083	0.450	1	.502
	O-Openness	**0.513**	**.076**	**46.080**	**1**	**<.001**
	WAIS IQ	0.005	.003	2.770	1	.096
	Age	-0.002	.010	0.055	1	.815
	Gender	**-0.255**	**.078**	**10.769**	**1**	**.001**
Sample 4						
	N-Volatility	**0.333**	**0.087**	**14.783**	**1**	**<.001**
	N-Withdrawal	**-0.280**	**0.093**	**9.019**	**1**	**.003**
	O-Intellect	0.131	0.108	1.477	1	.224
	O-Openness	**0.394**	**0.092**	**18.436**	**1**	**<.001**
	Age	**-0.044**	**0.020**	**4.744**	**1**	**.029**
	Gender	0.079	0.110	.512	1	.474

Note. Bolded estimates are significant at a level of $p < 0.05$.

Table 9. Poisson Regression Model for CAQ Scientific Scores.

Sample 1	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	N-Volatility	0.154	0.136	1.269	1	.260
	N-Withdrawal	-0.202	0.145	1.953	1	.162
	O-Intellect	0.227	0.174	1.704	1	.192
	O-Openness	0.003	0.170	0.000	1	.986
	WAIS IQ	**0.024**	**0.009**	**6.572**	**1**	**.010**
	Age	0.013	0.016	0.682	1	.409

Sample 2	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	N-Volatility	**-0.408**	**0.180**	**5.112**	**1**	**.024**
	N-Withdrawal	0.304	0.173	3.076	1	.079
	O-Intellect	**0.466**	**0.157**	**8.770**	**1**	**.003**
	O-Openness	-0.139	0.186	.559	1	.455
	g	0.113	0.149	.576	1	.448
	Gender	-0.097	0.166	.344	1	.557
	Age	**0.714**	**0.231**	**9.594**	**1**	**.002**
Sample 3						
	N-Volatility	-0.014	0.077	0.035	1	.851
	N-Withdrawal	0.070	0.089	0.613	1	.434
	O-Intellect	**0.362**	**0.096**	**14.171**	**1**	**<.001**
	O-Openness	-0.036	0.090	0.160	1	.689
	WAIS IQ	**0.009**	**0.003**	**7.051**	**1**	**.008**
	Age	-0.011	0.010	1.167	1	.280
	Gender	**-0.355**	**0.105**	**11.336**	**1**	**.001**
Sample 4						
	N-Volatility	-0.294	0.159	3.425	1	.064
	N-Withdrawal	0.125	0.196	0.405	1	.524
	O-Intellect	**0.552**	**0.205**	**7.248**	**1**	**.007**
	O-Openness	-0.157	0.209	0.566	1	.452
	Age	-0.001	0.025	0.002	1	.960
	Gender	0.227	0.191	1.421	1	.233

Note. Bolded estimates are significant at a level of $p < 0.05$.

N-Volatility was expected to have a positive effect on creativity in the Artistic domain, and a negative effect on creativity in the Scientific domain. N-Volatility did have a significant positive association with artistic creativity in samples 1, 2, and 4 (Table 8). It also showed a significant negative effect on scientific creativity in sample 2 (Table 9), and was approaching significance in sample 4 ($p = .064$).

N-Withdrawal was expected again to have no relationship or a slightly negative relationship with both artistic and scientific creativity. It had a significant negative relationship with artistic creative achievement in samples 2 and 4, and no other

significant relationships. In sample 2, it trended toward a positive effect on scientific creativity (p = .079).

Table 10. Poisson Regression Model for Subset of Sample 4 with WAIS Scores.

Overall CAQ	Parameter	B	Standard Error	Wald χ^2	df	p-value
	N-Volatility	**0.237**	**0.094**	**6.319**	**1**	**.012**
	N-Withdrawal	**-0.329**	**0.105**	**9.711**	**1**	**.002**
	O-Intellect	0.113	0.138	0.675	1	.411
	O-Openness	**0.261**	**0.126**	**4.335**	**1**	**.037**
	WAIS Verbal	0.011	0.007	2.755	1	.097
	WAIS Math	-0.011	0.016	0.454	1	.500
	Gender	0.230	0.164	1.974	1	.160
	Age	**-0.065**	**0.024**	**7.478**	**1**	**.006**
Artistic CAQ						
	N-Volatility	**0.276**	**0.109**	**6.402**	**1**	**.011**
	N-Withdrawal	**-0.350**	**0.120**	**8.470**	**1**	**.004**
	O-Intellect	0.055	0.152	0.129	1	.719
	O-Openness	**0.356**	**0.125**	**8.133**	**1**	**.004**
	WAIS Verbal	0.010	0.008	1.401	1	.236
	WAIS Math	-0.011	0.017	0.407	1	.524
	Gender	0.033	0.174	0.036	1	.850
	Age	**-0.078**	**0.027**	**8.527**	**1**	**.003**
Scientific CAQ						
	N-Volatility	0.070	0.269	.068	1	.794
	N-Withdrawal	-0.353	0.305	1.342	1	.247
	O-Intellect	0.203	0.240	.716	1	.397
	O-Openness	-0.054	0.260	.044	1	.834
	WAIS Verbal	0.020	0.011	3.590	1	.058
	WAIS Math	-0.007	0.033	.050	1	.822
	Gender	**1.143**	**0.295**	**15.013**	**1**	**<.001**
	Age	-0.010	0.048	.041	1	.840

Note. Bolded estimates are significant at a level of $p < 0.05$. N=123.

All analyses were repeated with quadratic terms for N-Volatility and N-Withdrawal. No quadratic terms showed significant effects using the robust standard

estimator. Results were similar when the smaller subset of sample 4, using WAIS in addition to O-Intellect, was analyzed, and did not differ from the sample overall (Table 10).

3.6 Discussion

Separating Neuroticism into its aspects—which have different associations with psychopathology, as well as distinct underlying neural subsystems—is important to clarify its exact effects on other traits; this approach is validated in study two, and suggests that analyzing Neuroticism as a whole can mask the effects of the aspects. Not only do the aspects predict creativity in different ways, they predict different *types* of creativity in different ways. In several samples, N-Volatility positively predicts artistic creative achievement, as well as creative achievement overall (which, in the CAQ, is biased toward artistic creative achievement), but has a negative effect on scientific creative achievement. N-Withdrawal had little to no effect on all types of creativity, with some samples showing a slight negative effect.

This pattern is consistent with previous findings that suggested that subjects who were more artistically creative tended to be more hostile and impulsive compared to subjects who were higher on scientific creativity (Feist, 1998). Hostility and impulsivity are closely related to N-Volatility (DeYoung et al., 2007). On the other hand, depression is more strongly associated with N-Withdrawal (Quilty et al., 2012), and depression has shown no association or a slight negative association with creativity (Silvia & Kimbrel, 2010; Srivastava et al., 2010). Similarly, bipolar disorder is more strongly associated with N-Volatility, and bipolar disorder has shown a positive association with creativity

(Andraeson, 1987; Landen, 2012; Santosa et al., 2007; Simeonova et al., 2005). These findings also suggest that the aspects of Neuroticism may play a mediating role in the relationship between psychopathology and creativity; some of the variance is related to the underlying phenotypes that contribute to the development of the disorder. Again, this hypothesis is supported by the fact that treating bipolar disorder does not decrease creativity (Andraesen & Glick, 1988; Belmaker, 2004), suggesting that the association is not due to the symptoms of bipolar disorder.

These findings reinforce the importance of focusing on Neuroticism and its aspects as common underlying factors across different types of psychopathology. The etiological relationship between types of psychopathology (such as Internalizing and Externalizing disorders, or mood disorders and psychotic disorders) is murky and often poorly understood; the DSM categorizations of these disorders do not capture these etiological relationships. Neuroticism and its aspects are more measureable phenotypes that may reflect the complex relationships between disorders, as well representing the spectrum of psychopathology—as opposed to the dichotomies imposed by the DSM. The personality profile associated with psychopathology may represent a mediating factor in the observed associations, with a personality profile that corresponds to higher creativity also corresponding to an increased risk of psychopathology. If the development of psychopathology is associated with the aspects of Neuroticism, and creativity is associated with the aspects of Neuroticism, then psychopathology and creativity would appear to be related—even though the latter two may not be causally linked.

There were several unexpected findings in study 2. First, N-Withdrawal did not

have a consistent relationship with creative outcomes. It had a negative relationship with artistic creativity in two samples, and it actually trended toward a positive effect on scientific creativity in sample 2; however, overall, N-Withdrawal did not seem to influence creative achievement. This again reinforces the importance of separating the aspects when determining the relationship between Neuroticism and other traits; there could be suppressive effects when the aspects are analyzed together, which would prevent significant effects from being observed.

An alternative explanation for the lack of effect of N-Withdrawal on creative achievement is that psychopathology itself is typically associated with negative effects on creative achievement. N-Withdrawal may have a positive effect up to a point, but when psychopathology develops (for example, Internalizing disorders are particularly associated with high N-Withdrawal), creativity is impaired. However, the quadratic terms for N-Volatility and N-Withdrawal did not have significant effects in any of the models. This could be in part due to the non-normal nature of the CAQ data, which would make it more difficult to observe quadratic effects, particularly because the models used the more stringent robust standard estimator in order to control for any overdispersion and the non-independence of the count data. A model with a normal dependent variable would be more likely to find small effects.

Conversely, N-Withdrawal may simply have a limited effect on creative achievement; this would also be consistent with previous studies where Internalizing disorders either had no relationship or a slight negative relationship with creative achievement. N-Volatility has a positive relationship with creative achievement in three

of the four samples, particularly artistic creative achievement (and a negative effect on scientific creative achievement in one sample); although it is again feasible that a non-linear relationship exists (that is, that N-Volatility has positive effects to a certain point, and then has a negative effect), this was not demonstrated in the current study. Future studies may examine this possibility in more detail; however, it does appear that N-Volatility is the driving force in the relationship between creativity and Neuroticism.

4.1 Study 3: Intrinsic Connectivity Networks Associated with Neuroticism

Both structural and functional differences in the brain have been associated with Neuroticism overall, as well as the separate aspects. The final study will examine functional correlates of Neuroticism, using intrinsic connectivity networks as the primary measure. Intrinsic connectivity networks are related to the fundamental architecture of the brain, and reflect patterns of connectivity between regions—regions that are frequently active in synchrony are presumed to have more functional connectivity. The first resting connectivity network identified was the "resting state" or default-mode network (DMN); this functional network is particularly "active" even when the brain is at "rest" (Smith et al., 2009).

Functional neuroimaging studies first sought to identify regions within the brain that were active during specific tasks, and saw increases in particular regions, as expected; however, task-induced *decreases* in activity were also observed (Raichle et al., 2001). This meant activity was greater in particular regions during the control or "baseline" state—the control condition measured brain activity when the subject was at rest, which generally took the form of a simple visual fixation task or lying quietly with

closed eyes. Thus, this baseline activity was dubbed the "resting" or "default" state (Raichle et al., 2001).

Resting state activity includes areas where activity increases during a task (task-positive components) and areas where activity increases during rest (task-negative components; Broyd et al., 2009). The correlation in activity between regions suggested that task-positive activity was not the only measure of interest; functional connectivity between regions was also important, which could be measured through correlated activity in different regions. The patterns of synchronization—regions that are often active or inactive at the same time—reveal functional connectivity, which reflects the underlying architecture of the brain. In particular, correlations between regions that are active at rest appear to signify intrinsic connections in the brain, especially as they show synchronized activity across different resting states (Fox & Raichle, 2007). Synchronized activity in different regions was thus taken to reflect the underlying functional connectivity in the brain, where regions that are more often synchronized are able to communicate more easily, reflecting their intrinsic connectivity.

Intrinsic connectivity networks include the Default Mode network, along with other patterns of synchronization within the structures of the brain; they are a measure separate from either functional activation or structural components. In the third study, the networks were identified by an independent components analysis (ICA) and correlated with Neuroticism. The specific intrinsic connectivity networks chosen for further analysis were determined *a priori*, based on previous research investigating the underlying neural basis for Neuroticism.

Individual variation in personality traits correlates with functional activity in neuroimaging studies, implying that the personality traits are markers of these neural systems. FMRI and similar studies have catalogued differences in brain structure and function that relate to high levels of Neuroticism. One issue with many existing MRI and fMRI studies of personality is that they are underpowered, or may not control sufficiently for non-independence of tests (Button et al., 2013; Vul et al., 2009; Yarkoni, 2009). However, studies that use *a priori* hypotheses and establish regions of interest can increase their power; larger sample sizes also increase power. Although false positive findings are always a risk, having strong *a priori* reasoning behind a study may be more beneficial than concentrating on eliminating false negatives, as the former can be self-correcting (Fielder, Kutzner, & Krueger, 2012). Further, focusing on studies with large samples to find reliable effects can suggest *a priori* hypotheses that are more likely to be borne out.

Finding behavioral correlates of personality traits can be a useful first step in *a priori* reasoning—if a trait is associated with a particular behavior and that behavior is associated with activity or connectivity in a particular area of the brain, then that area might also relate to the trait. It also suggests a possible neural mechanism for the trait. One consistent finding is that subjects with high Neuroticism are more sensitive to punishment, and some studies have suggested that depression in particular is linked to decreased sensitivity to reward. This blunted response to reward actually predicts the development of depression (Bress et al., 2013). Adolescent subjects at high risk for depression show significantly less reward sensitivity (Foti et al., 2011). Other studies

have found that subjects high in Neuroticism avoid "risky" decisions and have increased activation in the insula region following a punishing response (Paulus et al., 2003); they also show increased response to uncertainty (Hirsh & Inzlicht, 2008). Subjects high in Neuroticism showed more activity in the right anterior insula when anticipating losses (Wu et al., 2014). Higher Neuroticism also correlates with higher reactivity to punishment (e.g., Thake & Zelenski, 2013). Hirsh and Inzlicht (2008) further suggest that individuals who are high in Neuroticism may be characterized by a stronger aversion to uncertain stimuli than to negative stimuli.

Subjects high in Neuroticism had more activation in the amygdala and the anterior cingulate cortex during situations with emotional conflict (Haas et al., 2007). The cingulate cortex-amygdala circuit is associated with emotional regulation and response to negative social encounters, and results from multiple studies suggest that this circuit is dysfunctional in subjects with higher levels of Neuroticism (Cremers et al., 2010; Haas et al., 2007; Ormel et al., 2013; Pezawas et al., 2005). This could signify that subjects with high Neuroticism have less control over their emotional responses. One large study found that individuals on the extreme end of negative affect showed an inverse relationship between the size of the amygdala and the rostral anterior cingulate cortex/medial PFC, suggesting that imbalance between the two corresponds with increased negative emotions (Holmes et al., 2012).

Further, other studies suggest that the impaired connection between the amygdala and the cingulate cortex in subjects with high Neuroticism decreases their ability to ignore anxiety-provoking stimuli (Ormel et al., 2013). Neuroticism was associated with

greater activity in the insula and anterior cingulate during experience of cognitive and emotional pain (Coen, 2011). Neuroticism also correlates with increased amygdala response to negative stimuli (Kennis, Rademaker, & Geuze, 2013; Canli et al., 2001; Wright et al., 2001), especially faces (Cremer et al., 2010; Haas, Constable, & Canli, 2008). However, at least one very small study (N=18) suggests that the two facets of Neuroticism may predict amygdala activation differently. Cunningham et al. (2010) presented positive, negative, and neutral images in an fMRI; subjects could choose to press a button and "approach" the stimulus—which made the image larger—or "avoid" the stimulus, which made the image smaller. They showed that subjects with high N-Withdrawal had increased amygdala activation to all approached stimuli, while subjects with high N-Volatility only had increased activation to negative stimuli, whether approached or avoided.

Subjects who were highly anxious (which closely correlates to Neuroticism and N-Withdrawal) showed increased transient activity in the PFC compared to normal subjects in neutral conditions; in negative conditions, the activity in low-anxiety subjects matched that of high-anxiety subjects (Fales et al., 2008). This could imply that the anxious subjects are in fact using more of their cognitive resources; Fales et al. (2008) suggests that the reduced cognitive control of negative emotions is the result of greater working memory load in neutral conditions. Subjects with social anxiety disorder showed differences in resting state networks that included the anterior cingulate, the orbitofrontal cortex, and the angular gyrus, which may relate to the failure of emotional regulation that is present in anxiety disorders (Liao et al., 2010). Interestingly, both the PFC and the

cingulate cortex were hyperactive in patients with depressive traits (Schöning et al., 2009). This could imply a mechanism for the cognitive deficits. However, most studies examined Neuroticism or negative affect more generally, and the separate aspects of Neuroticism could have unique effects on cognitive functioning.

4.2 Hypotheses

The first regions of interest identified *a priori* as having a possible association with Neuroticism were the insula, the amygdala, and the rostral anterior cingulate—the insula because of its association with punishment and negative arousal (Wu et al., 2014; Coen, 2011), the amygdala because of its role in negative emotion and approach and avoidance behavior (Cunningham et al., 2010); and the anterior cingulate because of its role in emotional regulation (Etkin, Egner, & Kalisch, 2011). These three regions have more evidence for their role in Neuroticism than any others (Allen & DeYoung, in press). An intrinsic connectivity network that included these areas will be the first analyzed. Psychopathology is not always associated with decreased connectivity scores in related ICNs—for example, a recent study found that addiction was associated with increased functional connectivity in the insula, which may relate to increased punishment sensitivity; on the other hand, it found reduced connectivity in areas related to impulse control (Wisner et al., 2013). However, for this study Neuroticism was predicted to have a negative association with connectivity in the first network (the anterior cingulate, insula, and amygdala) because of Neuroticism's negative effect on emotional regulation. A second ICN that includes the anterior and posterior cingulate was analyzed, with the same effect predicted.

In order to further investigate the relationship between Neuroticism and cognition, two networks that have been linked to anxiety and are involved in cognition were analyzed. The third ICN chosen for analysis included the angular gyrus, which plays a role in arithmetic and visuospatial tasks (e.g., Göbel, Walsh, and Rushworth, 2001; Grabner et al., 2008). However, in addition to its role in cognitive function, it showed differential connectivity in social anxiety disorders (Liao et al., 2010) and was more active during a "worry" condition compared to a "neutral" condition (Servaas et al., 2014). It also works with other regions to give context to memories, and is particularly important in social cognition; it plays a role in decision making and judgement, and especially in determining intentions of others through mental representation (Seghier, 2013).

Similarly, the orbitofrontal cortex (OFC) is associated with decision making, particularly in response to rewards and punishment, as well as impulse regulation (Berlin, Rolls, & Kischka, 2004). Previous studies have found decreased resting state connectivity between the amygdala and OFC in subjects with anxiety disorders, which may related to their ability to process potential threats (Hahn et al., 2011). The same study found that the anterior and posterior cingulate may play a role in modulating this effect. Therefore, the final ICN chosen for analysis included the orbitofrontal cortex and the anterior cingulate. As before, it was predicted that Neuroticism will have a negative effect on resting state connectivity in these networks.

Much of the previous research examining resting state networks and neuroticism has focused more on Internalizing disorders, which are more closely related to N-

Withdrawal. Further, it would make sense that punishment sensitivity and harm avoidance are the underlying constructs driving the effects, which again are more closely related to N-Withdrawal. Therefore, for all networks, N-Withdrawal was predicted to have a stronger negative association than N-Volatility.

4.3 Methods

4.3.1 Participants.

The participants consisted of a smaller subset of Sample 3, described in study one. This sample included 244 subjects who had scans that contributed to the analyses. A smaller subset of the sample (N=218) which used a more stringent movement correction was also analyzed; as results were largely the same in both samples, the results for the larger sample will be presented.

4.3.2 Measures

Personality and Intelligence. As in the previous studies, the BFAS was used to measure the aspects of Neuroticism, while the WAIS was used to measure intelligence.

Neuroimaging. The intrinsic connectivity networks (ICNs) were generated using a meta-ICA pipeline to optimize network consistency (Poppe et al., 2013). This used the MELODIC (Multivariate Exploratory Linear Optimized Decomposition into Independent Components) function in FSL to compute group level probabilistic independent components. For each of the 25 ICAs, randomized subject order was used that included a smaller subset of the sample, due to both computational limits and the desire to reduce the likelihood of overfitting. A dimensionality restraint of 60 was chosen based on prior optimization findings (Poppe et al, 2013). The 60 components from each ICA were then

concatenated into a single file that was used as input for MELODIC. Procedures suggested by Kelly et al. (2010) were used to identify possible artifacts, such as those involved in autonomic functions, as well as possible artifacts created by movement within the scan. For the final analysis, 27 non-artifactual components were retained, using techniques outlined by Poppe et al. (2013).

During the preprocessing stage, motion correction and motion conduction were conducted in order to reduce within-subject movement. A movement correction was computed; it is a summary statistic that reflects the average displacement across the six movement parameters (translational displacements along the X, Y, and Z axes, and rotational displacements of pitch, yaw, and row). This statistic, the root mean square head position change (RMS movement), is included as a covariate in all analyses.

In order to compute the network coherence scores—or mean connectivity scores—for each network, a subject-level spatial map was derived for each subject using dual regression (Poppe et al., 2013 and Wisner et al., 2013). For each subject, the mean of all voxels within each group level map was computed separately for each component. The values for each subject in each component reflect network coherence; larger values indicate more synchronized dynamics across voxels, and therefore, greater coherence. All components had a positive skew, which was corrected through a log-transformation. Finally, all network coherence values were standardized to z-scores. Connectivity scores between networks were calculated as well, and then standardized to z-scores. Again, higher values indicated greater coherence in the connectivity between the networks.

4.4 Analysis

The intrinsic connectivity networks of interest were, in the original set of components, ICN 8 (the angular gyrus; Figure 6.1), ICN 12 (the anterior cingulate, insula, amygdala, and surrounding limbic areas; Figure 6.2), ICN 15 (the aCC and orbitofrontal cortex; Figure 6.3), and ICN 22 (aCC and pCC; Figure 6.4).

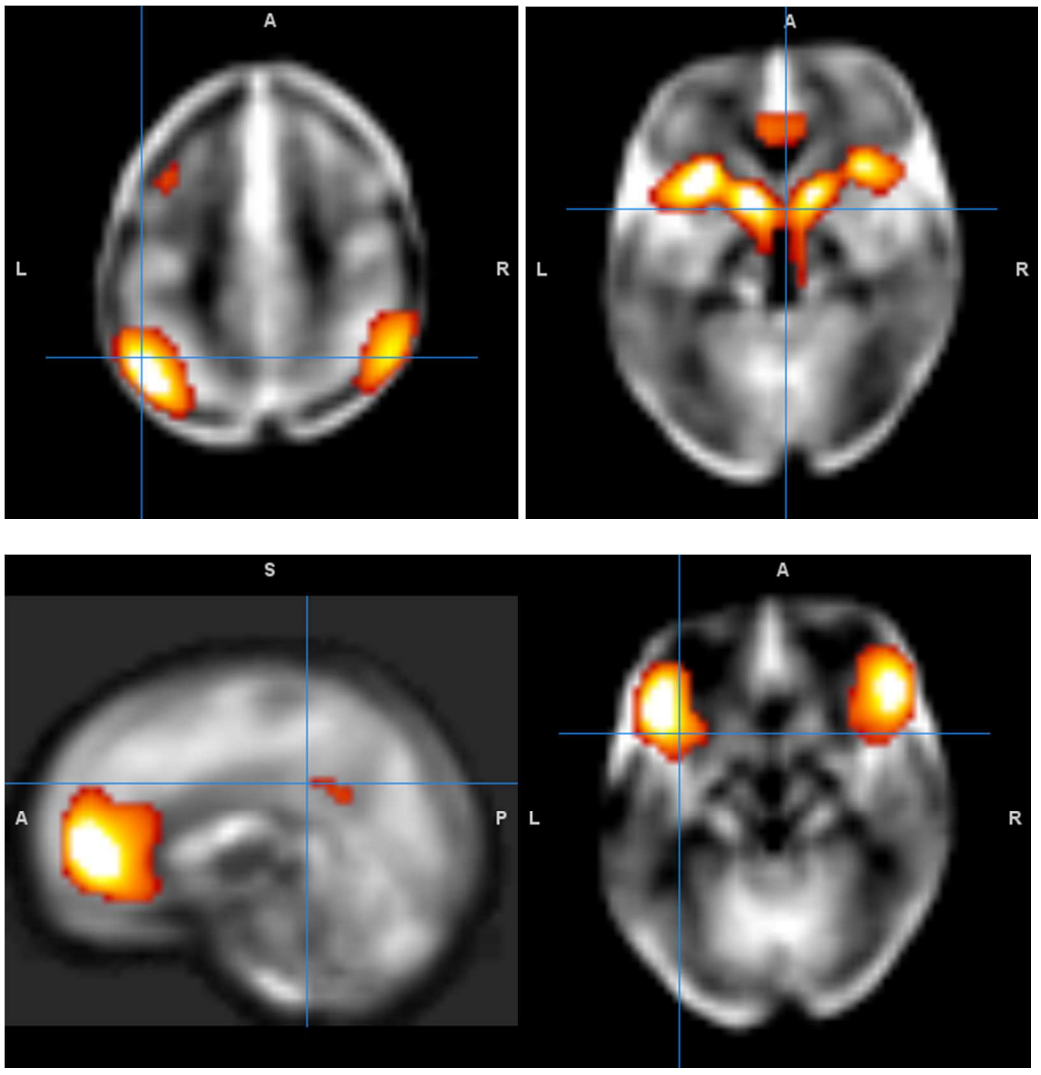

Figure 6. Location of ICNs. Clockwise from top left. 6.1 ICN 8, angular gyrus. 6.2 ICN 12, anterior cingulate, insula, and amygdala. 6.3 ICN 15, anterior cingulate and orbitofrontal cortex. 6.4 ICN 22, anterior and posterior cingulate.

First, bivariate correlations were calculated between the ICNs and Neuroticism, N-Volatility, and N-Withdrawal, after controlling for age, gender, WAIS scores, and a standard correction for movement. The movement correction is a summary statistic that reflects the average displacement across the six movement parameters (translational displacements along the X, Y, and Z axes, and rotational displacements of pitch, yaw, and row). Correlations between intelligence, creativity, the ICNs, and the ICN interconnectivity networks were also calculated; creativity was assessed through the CAQ, which was then log-transformed to decrease its non-normality.

Each network was regressed onto N-Volatility and N-Withdrawal, along with age, gender, WAIS, and the movement correction. Next, the inter-network connectivity values between the networks (e.g., between ICNs 8 and 12, ICNs 8 and 15, and ICN 8 and 22) were entered into a regression model with the same covariates. The regression was then repeated adding quadratic terms for N-Volatility and N-Withdrawal.

4.5 Results

ICN 22 did not have a significant correlation with Neuroticism, N-Withdrawal, or N-Volatility; the other three networks correlated significantly with N-Withdrawal, and ICN 8 correlated significantly with Neuroticism overall (Table 11). ICN 8 also correlated with log-transformed scores on the CAQ, but neither WAIS scores nor CAQ scores significantly correlated with any of the other ICNs or ICN interconnectivity (Table 12). The networks were also significantly correlated with each other (Table 13).

Network	Neuroticism	N-Withdrawal	N-Volatility
ICN 8	-.138*	-.187**	-.065
ICN 12	-.080	-.141*	-.008
ICN 15	-.115	-.155*	-.055
ICN 22	-.003	-.098	.084
ICNs 8 and 12	-.058	-.068	-.037
ICNs 8 and 15	-.142*	-.161*	-.096
ICNs 8 and 22	-.134*	-.157*	-.086
ICNs 12 and 15	-.140*	-.151*	-.101
ICNs 12 and 22	-.044	-.085	.003
ICNs 15 and 22	-.004	-.053	.041

Table 11. Bivariate Correlations Between ICNs and the Aspects of Neuroticism, Controlling for Intelligence, Age, Gender, and Movement.

Note. *Correlation is significant at $p < .05$. **Correlation is significant at $p < .01$.

Table 12. Bivariate Correlations Between ICNs and Measures of Intelligence and Creativity, Controlling for Movement.

Network	WAIS IQ	Overall CAQ
ICN 8	.051	.149*
ICN 12	.025	.104
ICN 15	-.027	.059
ICN 22	.049	.068
ICNs 8 and 12	.007	.080
ICNs 8 and 15	-.012	.013
ICNs 8 and 22	.014	.052
ICNs 12 and 15	-.013	.059
ICNs 12 and 22	.050	.045
ICNs 15 and 22	.012	.019

Note. *Correlation is significant at $p < .05$. **Correlation is significant at $p < .01$.

Table 13. Bivariate Correlations Between ICNs and Inter-ICN Connectivity, Controlling for Movement.

Network	1	2	3	4	5	6	7	8	9
1. ICN 8	-								
2. ICN 12	.438**	-							
3. ICN 15	.344**	.605**	-						
4. ICN 22	.227**	.469**	.582**	-					
5. ICNs 8 and 12	.240**	.280**	.123	.100	-				
6. ICNs 8 and 15	.178**	.184**	.184**	-.002	.439**	-			
7. ICNs 8 and 22	.208**	.168**	.102	.060	.328**	.501**	-		
8. ICNs 12 and 15	.094	.528**	.448**	.238**	.266**	.153*	.195**	-	
9. ICNs 12 and 22	.124	.513**	.407**	.297**	.255**	.090	.217**	.691**	-
10. ICNs 15 and 22	.078	.360**	.376**	.250**	.220**	.179**	.260**	.553**	.658**

Note. *Correlation is significant at $p < .05$. **Correlation is significant at $p < .01$.

Because multiple regressions are performed, it is necessary to have some correction for multiple tests. The most stringent correction for testing multiple networks is the Bonferonni correction, where the *p*-value of .05 divided by the number of tests. In this case, the *p*-value for within network connectivity would be .0125, and the *p*-value for between network connectivity would be .008. However, the Bonferonni correction assumes that tests are independent, which is not true for these analyses; the networks overlap and correlate. Further, Perneger (1998) points out that the use of this method means that the interpretation of findings is based on how many tests have been performed and that it can exponentially increase the probability of a type II error.

An alternative method is the Holm multistage procedure, which compares the test with the lowest p-value to .05 divided by the number of tests. If that test is significant, the next lowest p-value is compared to a value of .05 divided by the number of tests minus 1 (Shaffer, 1995). In the case of the within network regressions, the first p-value would be .0125, the second would be .017; the between network regressions would begin at .008, followed by .01, and so forth. Because of the relatively small number of comparisons, this method will be used.

Table 14. Regression Model for ICNs and the Aspects of Neuroticism.

ICN 8	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	Gender	**-0.124**	**0.031**	**15.820**	**1**	**<.001**
	Age	**-0.010**	**0.004**	**7.598**	**1**	**.006**
	WAIS IQ	0.001	0.001	2.182	1	.140
	Movement Correction	**0.405**	**0.127**	**10.198**	**1**	**.001**
	N-Volatility	0.022	0.022	0.961	1	.327
	N-Withdrawal	**-0.079**	**0.024**	**10.427**	**1**	**.001**[1]

ICN 12	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	Gender	**-0.087**	**0.031**	**7.965**	**1**	**.005**
	Age	-0.007	0.004	2.562	1	.109
	WAIS IQ	0.001	0.001	1.625	1	.202
	Movement Correction	**0.629**	**0.129**	**23.839**	**1**	**.001**
	N-Volatility	0.035	0.025	2.001	1	.157
	N-Withdrawal	**-0.070**	**0.030**	**5.481**	**1**	**.019**[3]
ICN 15						
	Gender	**-0.101**	**0.030**	**11.504**	**1**	**.001**
	Age	-0.006	0.004	2.633	1	.105
	WAIS IQ	0.000	0.001	0.146	1	.703
	Movement Correction	**0.720**	**0.110**	**42.779**	**1**	**<.001**
	N-Volatility	0.017	0.024	0.498	1	.480
	N-Withdrawal	**-0.061**	**0.027**	**5.215**	**1**	**.022**[4]
ICN 22						
	Gender	-0.040	0.036	1.270	1	.260
	Age	-0.004	0.005	0.641	1	.423
	WAIS IQ	0.002	0.001	3.377	1	.066
	Movement Correction	**0.727**	**0.120**	**36.551**	**1**	**<.001**
	N-Volatility	**0.078**	**0.027**	**8.346**	**1**	**.004**[1]
	N-Withdrawal	**-0.090**	**0.029**	**9.288**	**1**	**.002**[2]

Note. Bolded estimates are significant at a level of $p < 0.05$.
[1] significant at $p < .0125$; [2] significant at $p < .017$; [3] significant at $p < .025$; [4] significant at $p < .05$.

Using the Holm multistage procedure, all four networks had a significantly negative relationship with N-Withdrawal (Table 14). ICN 22 had a significantly positive relationship with N-Volatility ($p = .004$); connectivity in the other networks was not significantly predicted by N-Volatility. The quadratic terms were not significant for the within-network regression models, so this was not repeated for the inter-network connectivity. The first model was used to predict inter-network connectivity between the four networks. Connectivity between ICN 8 and ICN 15, as well as ICN 8 and ICN 22, had a significant negative relationship with N-Withdrawal at level of $p = .05$; however,

no effect was significant at the more stringent correction of .008 (Table 15). Connectivity between the other networks (8 and 12, 12 and 15, 12 and 22, and 15 and 22) was not significantly predicted by N-Withdrawal or N-Volatility at the level of $p = .05$. In all cases, N-Volatility was not significant, and N-Withdrawal trended in the negative direction.

Table 15. Regression Model for Connectivity between ICNs and the Aspects of Neuroticism.

ICNs 8 and 12	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	Gender	**-0.055**	**0.027**	**4.284**	**1**	**.038**
	Age	0.000	0.003	.002	1	.962
	WAIS IQ	0.000	0.001	.045	1	.832
	Movement Correction	0.058	0.095	.371	1	.543
	N-Volatility	0.001	0.019	.006	1	.937
	N-Withdrawal	-0.021	0.019	1.198	1	.274
ICNs 8 and 15						
	Gender	-0.009	0.027	0.106	1	.745
	Age	-0.002	0.003	0.245	1	.620
	WAIS IQ	0.000	0.001	0.064	1	.801
	Movement Correction	**-0.279**	**0.078**	**12.883**	**1**	**<.001**
	N-Volatility	0.000	0.020	0.000	1	.983
	N-Withdrawal	**-0.047**	**0.021**	**5.091**	**1**	**.024**
ICNs 8 and 22	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	Gender	0.016	0.031	0.270	1	.603
	Age	-0.005	0.004	1.718	1	.190
	WAIS IQ	0.000	0.001	0.225	1	.636
	Movement Correction	-0.031	0.078	0.156	1	.693
	N-Volatility	0.003	0.022	0.024	1	.876
	N-Withdrawal	**-0.055**	**0.027**	**4.087**	**1**	**.043**

ICNs 12 and 15	Parameter	B	Standard Error	Wald χ^2	df	*p*-value
	Gender	**0.624**	**0.151**	**5.740**	**1**	**.017**
	Age	-0.001	0.004	0.060	1	.806
	WAIS IQ	0.000	0.001	0.006	1	.938
	Movement Correction	0.194	0.124	2.443	1	.118
	N-Volatility	-.006	0.024	0.054	1	.816
	N-Withdrawal	-.048	0.025	3.623	1	.057
ICNs 12 and 22						
	Gender	-0.016	.032	0.237	1	.626
	Age	-0.002	.004	0.223	1	.637
	WAIS IQ	0.001	.001	2.271	1	.132
	Movement Correction	**0.394**	**.111**	**12.609**	**1**	**<.001**
	N-Volatility	0.026	.024	1.211	1	.271
	N-Withdrawal	-0.047	.025	3.629	1	.057
ICNs 15 and 22						
	Gender	-0.024	0.036	0.457	1	.499
	Age	-0.001	0.004	0.035	1	.852
	WAIS IQ	0.001	0.001	0.740	1	.390
	Movement Correction	**0.413**	**0.118**	**12.237**	**1**	**<.001**
	N-Volatility	0.039	0.028	2.043	1	.153
	N-Withdrawal	-0.046	0.030	2.366	1	.124

Note. Bolded estimates are significant at a level of $p < 0.05$.

4.6 Discussion

This study provides more specifics as to the neural basis of N-Withdrawal. Neuroticism as a whole was negatively correlated with connectivity in several areas related to emotional regulation and cognition; however, when separated into its aspects, N-Withdrawal was primarily driving this effect. The non-linear model was not a better fit for this relationship. N-Volatility had no significant effect on connectivity in three areas, but in one region that included that anterior and posterior cingulate, it actually had a significantly positive relationship. This further reinforces the importance of separating

the aspects; if Neuroticism was examined as a whole, N-Volatility and N-Withdrawal would effectively cancel each other out, making it more difficult to observe significant effects.

The consistently negative impact of N-Withdrawal on functional connectivity has implications for the neural basis of this trait. First, its effect on areas related to cognition (such as the angular gyrus and the orbitofrontal cortex) suggests a neural mechanism for the influence of Neuroticism on decision making and social cognition. Neuroticism correlates with increased sensitivity to punishment; this could be related to its effects in the OFC, which provides expectations for rewards and punishments of any given action (Schoenbaum et al., 2011). Its decreased functional connectivity in association with N-Withdrawal could relate to decreased reward sensitivity that is observed in Neuroticism and particularly in Internalizing disorders. It could also connect to increased punishment avoidance, as the decreased connectivity might tie to an inability to correctly assess the potential risks of a situation. The aversion of individuals who are high in Neuroticism to ambiguous stimuli could also relate to an inability to assess the ambiguous stimuli.

The angular gyrus is important for mental representations of others (Seghier, 2013), and Neuroticism correlates with making more negative attributions (e.g., Cheng & Furnham, 2001; Barlow et al., 2014). The decreased coherence in the angular gyrus might reflect this relationship. Further, the decreased network coherence in areas related to emotional regulation—particularly the cingulate cortex—may relate to an inability to prevent negative emotions from intruding on cognition, and therefore negatively influencing cognitive performance. This may also serve as part of the neural basis of

rumination; the areas of the brain that are supposed to regulate emotional activity have decreased connectivity, which would manifest as an inability to set aside recurring negative thoughts or ideas. It also suggests a possible explanation for previous fMRI studies that find Neuroticism associated with increased blood flow to these areas: the reduced connectivity would make communication less efficient, which would then require more activity to have the same effect.

The positive association of N-Volatility with functional connectivity in the anterior and posterior cingulate was an unexpected finding. It might relate to differences in emotional regulation techniques in N-Volatility compared to N-Withdrawal, and it further reiterates the possibility of the aspects having opposing effects. However, as this effect was not predicted *a priori*, the likelihood of it as a false positive should be considered until it is replicated in another sample.

5. General Discussion

The protective effect of intelligence from most forms of psychopathology has been repeatedly demonstrated; the mechanism behind this effect is less clear. The effect of psychopathology on creativity is less consistent, and still less is known as to the mechanism. In both cases, examining the common personality profile that is associated with psychopathology—in particular, the trait of Neuroticism—has elucidated these connections and the processes behind them. Separating Neuroticism into its aspects—which have different associations with psychopathology, as well as distinct underlying neural subsystems—has also clarified some of the observed contradictory effects observed in previous studies.

The preceding studies reinforce the importance of separating the aspects of Neuroticism—as they sometimes have opposing associations with other traits—and in focusing on Neuroticism and its aspects as common underlying factors across different types of psychopathology. Cumulatively, these studies suggest that the aspects affect cognitive performance and creative achievements in different ways, and differentially correlate with the functional connectivity of the brain. They also suggest that N-Volatility and N-Withdrawal can serve as useful endophenotypes in the attempt to determine the etiology of psychopathology.

As the results of studies one and two mirror the relationship between specific disorders and these traits—for example, the relationship between bipolar disorder (associated with N-Volatility), depression (associated with N-Withdrawal; Quilty et al., 2013), and creativity—they suggest that personality may be a mediating factor in these relationships. The effect of N-Volatility on cognitive performance suggests that the former explains some of the basis of the negative association between intelligence and Externalizing disorders. They can also account for some of the overlap and comorbidity in different types of psychopathology; for example, using N-Withdrawal as an endophenotype could help determine the neural substrate of shared variance in Internalizing disorders. The effect of N-Withdrawal on cognitive performance helps explain previous findings related to IQ and Internalizing disorders, specifically depression, and suggests a possible non-linear relationship. Finally, the third study suggests a neural basis for N-Withdrawal. N-Withdrawal correlates negatively with functional connectivity in areas related to emotional regulation, as well as cognitive

performance and decision making. Interestingly, these networks did not correlate with either creativity or intelligence, suggesting that they are uniquely related to N-Withdrawal in particular. Although they do not explain the connection between N-Withdrawal and cognitive performance, they do provide evidence for the neural substrate of N-Withdrawal.

6. Future Directions

These studies provide evidence for the connection between Neuroticism, creativity, and intelligence, as well as suggesting possible neural correlates of Neuroticism. However, particularly with regard to functional connectivity, research is just beginning. Future studies will clarify the meaning of functional connectivity differences, and will further elucidate the connection between neural processes and particular phenotypes, such as N-Withdrawal and N-Volatility. The current study only found one significant effect for N-Volatility—which was not predicted, and therefore may represent a false positive—and future studies can determine functional networks that are particularly related to this phenotype, as it has a separate neural substrate than N-Withdrawal.

At the moment, the translation of neural phenotypes such as functional connectivity to broader personality traits that encompass cognition, affect, and behavior, is relatively unclear. Within the larger context of neuroimaging, it is difficult to explicate the meaning of neural processes associated with traits. Increased activity in a particular region might mean that the region is performing *more* of a process, or it could mean that the brain is performing that process *less efficiently*—both have been proposed as

plausible explanations. Increased network coherence also does not translate to a straightforward explanation in terms of brain function. Future research will clarify how the neural processes reflect observable traits and behaviors.

Substantive creativity research related to psychopathology is also—relatively speaking—just beginning; significantly better assessment techniques for creativity have assisted this process. The CAQ is a highly useful tool for assessing real-world creative achievements; however, in the current study, one major limitation is the age of the subjects. Presumably, the best time to measure lifetime creative achievement would be after most achievement takes place, and the current samples include relatively younger individuals, with significant achievements still ahead of them. Repeating the current study in an older sample may capture more of this variance and therefore better explain the connection between N-Volatility, N-Withdrawal, and the different domains of creativity. Alternatively, designing a measure that reliably assesses creative achievement potential in some applicable way might also be useful for younger samples. Further, as more creativity measures can be assessed while undergoing neuroimaging, a clearer picture of the neural processes that underlie creativity will present, which may also elucidate the processes which connect creativity to psychopathology and Neuroticism more generally.

Finally, the non-linear effect of N-Withdrawal on cognitive performances is a good parallel to the effects of state neuroticism; this model should be examined in other, more varied measures of cognitive performance. N-Withdrawal (and Neuroticism overall) had no effect on cognitive performance in the all-male sample; however, this difference

did not appear to be due to the different gender makeup of the sample, as interactions with gender were not significant in the other samples. Thus, the variables that might moderate the existence of the curvilinear effect remain unknown. The linear negative effect of N-Volatility was expected, given the negative association between disorders characterized to some degree by N-Volatility (such as Externalizing disorders and bipolar disorder) and reinforces the need to separate the aspects. In the future, neuroimaging of cognitive tasks could reveal more about the underlying neural basis of these effects, particularly if networks associated with both intelligence and N-Withdrawal were examined.

The current studies also suggest that the "fitness cliff" model that pertains to schizophrenia (e.g., Nesse, 2004) could be useful in describing and explaining other traits. In an evolutionary context, some traits may be selected for as they are helpful up until a certain point; however, beyond a certain level of the trait in question (the edge of the "cliff"), it has an abruptly strong, negative effect on reproductive fitness. Applying this model to N-Withdrawal, the trait may be associated with improved cognition up until a certain threshold, after which it interferes with cognitive performance, possibly due to the development of psychopathology, and becomes detrimental. Its relationship with cognitive performance resembles the inverted u-shaped curve, similar to what has been observed with state neuroticism; however, the development of an Internalizing disorder (which correlates with higher N-Withdrawal) might more closely resemble the cliff function (i.e., a sharp drop in ability); further, the effects of N-Withdrawal on reproductive fitness might follow the cliff-shaped function. As with psychotic disorders,

where traits like apophenia encourage creativity until the disorder develops, which then interferes with creativity (and reproductive fitness), N-Withdrawal may enhance cognitive performance until a certain point, after which the disorder such as depression interferes with cognitive performance (and possibly reproductive fitness). Using the fitness cliff model to explain the effects of endophenotypes clarifies the effects on other traits, as well as suggests an evolutionary explanation for the endophenotypes.

7. Conclusions

These studies collectively tie together the effects of Neuroticism and its aspects on intelligence and creativity, and explore the underlying neural substrate of Neuroticism. They suggest that previous conflicting findings related to Neuroticism, creativity, and intelligence may be related to the opposing effects of the aspects of Neuroticism, as well as to non-linear relationships. N-Volatility has a positive relationship with creativity, suggesting that the trait is advantageous in certain circumstances, despite its association with psychopathology. N-Withdrawal may also be associated with better cognitive performance up to a certain threshold. Both cases suggest that personality plays a mediating role in the relationship between psychopathology, intelligence, and creativity; N-Withdrawal and N-Volatility are common underlying factors in etiologically related disorders, which manifest in different ways but share some genetic and neural substrates.

Intelligence and creativity are both important traits for positive life outcomes, and further understanding of their relationship with psychopathology could have a positive influence on treatment outcomes. Further, as N-Volatility and N-Withdrawal may provide the causal basis for this relationship, understanding their effects and neural substrates will

elucidate the mechanism for these relationships. Better insight into neural functioning will positively influence treatment of mental illness related to Neuroticism and its aspects, and may help encourage development of creativity and intelligence.

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