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The role of executive function and the dorsolateral prefrontal cortex in the expression of neuroticism and conscientiousness

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The current study examined how specific neurological systems contribute to the expression of multiple personality dimensions. We used individuals with traumatic brain injuries to examine the contribution of the dorsolateral prefrontal cortex (DLPFC)—a region important for executive function and attention—to the expression of neuroticism and conscientiousness factors and facets. Results from Voxel-Based Lesion-Symptom Mapping analyses revealed that focal damage to the left DLPFC (Brodmann’s area 9) was associated with high neuroticism and low conscientious factor and facet scores (anxiety and self-discipline, respectively). Compared with lesioned and normal controls, veterans with damage in left DLPFC also reported higher neuroticism and lower conscientiousness facet scores, slower reaction times on the California Computerized Assessment Package assessment, and lower scores on the Delis–Kaplan executive function battery. Findings suggest that while neuroticism and conscientiousness remain psychometrically independent personality dimensions, their component facets may rely on a common neurocognitive infrastructure and executive function resources in general.

\textbf{Keywords:} Personality; DLPC; Executive function; Neuroticism; Conscientiousness; Self-control.

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Recent years have witnessed a shift from the development of purely descriptive psychometric models of personality (Big Five; John, Naumann, & Soto, 2008; McCrea & Costa, 2008; McCrea & John, 1992; Zuckerman, Kuhlman, Thornquist, & Kiers, 1991) toward development of causal, neurophysiological models of personality trait expression and individuality (DeYoung & Gray, 2009; DeYoung, Harris, Winkielman, & Pashler, 2010). Findings from modern personality neuroscience studies suggest that just as different personality dimensions are psychometrically independent, so too might be the neural systems that underlie their expression. Many of these studies reveal that morphological variation associated with the expression of various personality traits may indeed have dissociable neuroanatomical substrates that contribute to the expression of independent personality traits (Deckersbach, Miller, Klibanski, Fischman, & Dougherty, 2006; DeYoung, Harris, Winkielman, & Pashler, 2010; Knutson, Momenan, Rawlings, Fong, & Hommer, 2001; Omura, Constable, & Canli, 2005; Wright, Feczko, Dickerson, & Williams, 2007). Similarly, a number of functional neuroimaging studies find patterns of dissociable neural activity that selectively predict variation in specific personality traits (Eisenberger, Lieberman, & Satpute, 2005; Kumari et al., 2007; Omura et al., 2005).

While these approaches have advanced the understanding of the physiological basis of individuality, consistent with a psychological constructionist approach, i.e., the theoretical perspective which argues that neural regions and networks serve as basic psychological functions underlying a broad range of mental states (e.g., Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012; Feldman-Barrett, 2009), fewer studies have explored whether the expression of different, independent dimensions of personality, or facets thereof, might rely on a shared neuroanatomical infrastructure (c.f. Omura et al., 2005). In this study, we advance this line of questioning by examining whether facets of different traits—neuroticism and conscientiousness—are associated with the integrity of a common neuroanatomical region—the dorsolateral prefrontal cortex (DLPFC), in a large sample of patients with focal lesions in various brain regions. This approach provides a unique advantage over and above functional MRI (fMRI) studies, in that it provides a means to identify potential brain regions that are necessary to some extent for the expression of specific personality constructs, i.e., whether volume loss in a given brain region is associated with changes in the expression of specific personality traits.

Of the dimensions highlighted by prevalent five-factor (“Big Five”) models of personality (neuroticism, extraversion, openness, agreeableness, and conscientiousness; John et al., 2008; McCrea & Costa, 2008; McCrea & John, 1992), neuroticism has been most widely studied due to its relation to anxiety and depressive disorders (Kotov, Gamez, Schmidt, & Watson, 2010; Naragon-Gainey & Watson, 2011). Broadly, neuroticism identifies individuals who may be more likely to experience psychological distress (NEO Personality Inventory, Revised (NEO-PI-R); Costa & McCrea, 1992) and is measured by probing six core facets—anxiety, hostility, depression, self-consciousness, impulsiveness, and vulnerability to stress. In general, neuroticism is inversely related to brain volume in normal populations (Knutson et al., 2001) and is specifically associated with decreased volume (e.g., gray matter concentration and cortical thickness) in the superior and inferior frontal cortex (Brodmann’s areas (BAs) 6, 44), dorsomedial prefrontal cortex (PFC), orbitofrontal cortex, anterior cingulate cortex, and amygdala (Blankenstein, Chen, Mincic, McGrath, & Davis, 2009; DeYoung et al., 2010; Omura et al., 2005; Wright, Williams, Feczko, Feldman-Barrett, & Dickerson, 2006; 2007).

These findings suggest a prominent role for integrated cortical and subcortical planning, threat-detection, risk/reward, and learning systems in the expression of trait neuroticism. fMRI work and studies of regional cerebral glucose metabolism activity within these systems similarly show that they are critically involved in the expression of neuroticism (Deckersbach et al., 2006; Eisenberger et al., 2005). Furthermore, given that many facets of neuroticism likely evoke/involve negative affect (e.g., anxiety, hostility, and depression), we might also expect emotion regulation, or lack thereof, to play an important role in the expression of said traits. Indeed, past literature suggests that increases in neuroticism are associated with decreased tendencies to successfully regulate or reappraise emotions (Gross & John, 2003). Such regulatory processes directly recruit DLPFC (Banks, Eddy, Angstadt, Nathan, & Phan, 2007), suggesting that integrity within this region may be associated with neuroticism such that decreased DLPFC integrity is associated with increased levels of neuroticism.

In contrast to other personality dimensions, conscientiousness is the least understood from a cognitive neuroscience perspective. Conscientiousness describes the extent to which an individual is organized, persistent, controlled, and motivated in a goal-oriented manner (McCrea & John, 1992) and is comprised of six facets—competence, order/organization, dutifulness, achievement striving, self-discipline, and deliberation. Many previous volumetric investigations
of the neuroanatomical correlates of conscientiousness have reported null findings (Wright et al., 2006; 2007). However, there is evidence suggesting that both gross brain volume (Jackson, Balota, & Head, 2011) and cortical thickness within the DLPFC (middle frontal gyrus; DeYoung et al., 2010) are associated with conscientiousness scores. Collectively, this evidence suggests that conscientiousness is dependent, at least in part, on prefrontal regions that are integral for higher-order, complex cognition. As the DLPFC is essential for the execution of planned behaviors, integration of somatic and contextual information, and goal-directed behavior (Fuster, 1997; Miller & Cohen, 2001; Wood & Grafman, 2003), it may play a fundamental role in the expression of both neuroticism and conscientiousness.

To date, neuroanatomical studies of neuroticism and conscientiousness have focused on identifying dissociable neural regions involved in the expression of each of these traits. However, given the complex, multifaceted nature of personality traits, it is possible that the neural infrastructure supporting their expression is not comprised of the same specificity and independence that the traits’ psychometric properties possess. Like other complex psychological phenomena such as emotion or intelligence (e.g., Barbey, Colom, & Grafman, 2012; Barbey et al., 2012; Glascher et al., 2010; Murphy, Nimmo-Smith, & Lawrence, 2003; Wagner, Phan, Liberzon, & Taylor, 2003; Feldman-Barrett & Wagner, 2006), it is unlikely that any one neural system is both necessary and sufficient for the expression of any complex personality trait. Indeed, this could be one reason why traits such as neuroticism are associated with morphological differences in a wide variety of neural regions. Likewise, while the expression of a given personality trait may rely on unique neural regions, it is possible that the expression of numerous traits also depends on shared neural regions that generalize their processing roles to a wide variety of behavioral domains. In particular, one might expect that executive processing regions in the PFC (e.g., DLPFC), which operate in cognitive domains requiring effortful control over automated behavior, attention, planning, emotion regulation, and sequencing (Banks et al., 2007; Wagner, Maril, Bjork, & Schacter, 2001), to be involved in a number of personality traits that coincide with variation in these domains, such as neuroticism and conscientiousness.

Given that neuroticism is characterized by impulsivity (i.e., lack of controlled inhibitory processing), agitation, catastrophization, and may be associated with inefficacious emotion regulatory processes (anxiety; Costa & McCrea, 1992; Gross & John, 2003; John et al., 2008; McCrea & Costa, 2008; McCrea & John, 1992), we hypothesized that high levels of trait neuroticism would coincide with diminished executive processing and reduced DLPFC integrity (particularly BA 9). Conversely, given that conscientiousness is characterized by methodical planning and attention to detail, we hypothesized that high conscientiousness and component facets would coincide with efficacious executive functioning and DLPFC integrity. In this study, we examined these predictions as well as whether these two traits and their component facets may jointly be associated with DLPFC integrity, a crucial component of the executive processing system. To accomplish this, we first used a data-driven, exploratory analytic technique. Based on the results from these analyses, we then conducted hypothesis-driven analyses in a sample of combat veterans with focal traumatic brain injuries (TBIs) in the DLPFC.

**METHODS**

**Subjects**

Participants \((N = 249)\) were Vietnam Conflict Veterans drawn from Phase III of the W.F. Caveness Vietnam Head Injury Study registry (VHIS; see Krueger, Barbey, McCabe, Strenziok, & Zamboni, 2009; Krueger et al., 2011). This registry includes 199 patients (15 African American, 2 Asian, and 1 American Indian) with TBI resulting from combat-related penetrating head injuries (i.e., bullets and shrapnel), as well as 50 normal controls (six African American and one Asian) who also served in Vietnam in combat but had no history of neurological disorders. Patient and control groups were matched by age, level of education, handedness, episodic memory, and pre-injury intelligence (Table 1). Participants’ episodic memory was assessed with the delayed score of the logical memory subtest of the Wechsler Memory Scale, version III (WMS-III) (Wechsler, 1997), which assesses the amount of information from stories that a subject can recall after a 30-min delay. Pre-injury intelligence was assessed by computing percentile scores from the Armed Forces Qualification Test (AFQT-7A) (United States Department of Defense, 1960), an extensively standardized battery used by the US military that correlates highly with the Wechsler Adult Intelligence Scale intelligence quotient scores (Grafman, Jonas, Martin, Salazar, & Weingartner, 1988).

**Lesion data**

VHIS patient lesion data were taken from Computed Tomography (CT) scans. Lesion localization and
volume loss calculation were performed using the Analysis of Brain Lesions (ABLe) software implementation of MEDx v3.44 (Medical Numerics) (Makale et al., 2002; Soloman, Raymont, Braun, Butman, & Grafman, 2007). Lesions were manually traced in all relevant slices of CT images in native space. Tracings were completed by a trained psychiatrist with clinical experience in neuropsychological testing and reviewed by an investigator blind to the results of psychological testing (J.G.). Volume loss was calculated by summing traced areas and multiplying by slice thickness.

Subjects’ CT images were then spatially normalized to a CT template image in MNI space. This spatial transformation was then applied to the lesion image (Soloman et al., 2007). While it is always difficult to account for all possible damage incurred via penetrating TBI, this approach allowed for statistical comparison of imaging data and produced comprehensive calculations for both the percentage of volume loss across each subject’s whole brain as well as the percentage of loss within each BA using cytoarchitectural reference atlases (Lancaster, Woldorff, & Parsons, 2000; Maldjian, Laurienti, Burdette, & Kraft, 2003). A trained psychiatrist with experience in reading CT scans performed all lesion tracing.

The phase III VHIS battery

As part of Phase III data collection, both patients and controls completed a comprehensive battery of questionnaires measuring cognitive functioning and personality traits. As part of the cognitive functioning battery, VHIS participants completed the California Computerized Assessment Package (CaCAP; Miller, 1990), which assesses “basic executive functions involved in timed psychomotor skills requiring focused or sustained attention”. In addition to providing baseline measures of reaction time (RT) for both dominant and non-dominant hands, the CalCAP assesses RT on tasks requiring participants to respond as soon as they see particular digits (i.e., Choice RT). Participants also completed the Delis–Kaplan Executive Function System (D-KEFs; Delis, Kaplan, & Kramer, 2001), which incorporates standardized tests for examining high-level executive functions, such as procedural sequencing and reading comprehension, and produces a weighted, standardized overall achievement score where higher numbers represent aptitude in testing domains. Personality assessment was performed using the NEO-PI-R (Costa & McCrea, 1992). The NEO is a 240-item standardized assessment of personality based on the five-factor (Big 5) model of personality structure, which computes standardized t-scores for each of five higher-order personality traits from 30 facet scores (Costa & McCrea, 1992; McCrea & John, 1992).

Table 1

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Years education</th>
<th>Pre-injury IQ</th>
<th>Episodic memory</th>
</tr>
</thead>
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<tr>
<td>Injury</td>
<td>58.31 (3.09)</td>
<td>14.74 (2.54)</td>
<td>60.30 (25.43)</td>
<td>36.65 (9.61)</td>
</tr>
<tr>
<td>Control</td>
<td>59.00 (3.40)</td>
<td>15.19 (2.47)</td>
<td>65.40 (22.91)</td>
<td>39.89 (8.31)</td>
</tr>
</tbody>
</table>

Notes: Age represents the age of participants at the time of Phase III testing for the VHIS. Pre-injury intelligence was assessed by computing percentile scores from the Armed Forces Qualification Test (AFQT-7A). Episodic memory scores represent raw delayed scores on the logical memory subtest of the Wechsler Memory Scale, version III. IQ = Intelligence.

Analysis strategy

Analysis of participants’ data comprised two approaches. First, we analyzed lesion data using Voxel-Based Lesion Symptom Mapping (Bates et al., 2003). This exploratory approach utilizes the circumscribed lesion data in CT (or MRI) image volumes, transforms volumes into standardized space (i.e., MNI, Talairach), and performs voxel-by-voxel t-tests with respect to pre-defined behavioral scores entered for each subject. VLSM analyses produce image maps of voxels indicating areas of tissue loss in patients with behavioral scores that are significantly different from patients without tissue loss in those areas. Significance thresholds were specified prior to analysis at p < .005, 10 contiguous voxels. The VLSM analytical approach and thresholding used here are similar to general linear model implementations and have been identified as an acceptable threshold for the analysis of functional neuroimaging data (e.g., fMRI and PET) (Bates et al., 2003; Lieberman & Cunningham, 2009). It presents a more rigorous approach to identify the anatomical location of lesions that produce group-level differences between behavioral measures than standard region of interest approaches to lesion data (Bates et al., 2003). The
thresholding and analytical approach are also worthwhile and necessary in light of the fact that analyses were conducted on TBI patients with a wide distribution of injuries across the brain (as opposed to injuries specific to one region of the brain). A separate VLSM analysis was initially conducted for each of the Big 5 factors and their facets. Based on the outcome of these exploratory analyses, VLSM analyses proceeded with two separate regression analyses: one that regressed neuroticism composite scores on to conscientiousness composite scores and one that regressed conscientiousness composite scores on to neuroticism composite scores to isolate variance unique to each factor. We then entered the residualized NEO scores for the two factors and each facet in VLSM analyses, which included all Phase III VHIS lesion patients with valid scores on the NEO-PI-R (N = 190). Localization for significant clusters was performed using VOTL BA map implementations built into ABLe software (Lancaster et al., 2000; Maldjian et al., 2003; Solomon et al., 2007).

Once target brain regions were identified via exploratory VLSM analyses, we used hypothesis-driven one-way ANOVAs to examine whether DLPFC (BA 9) patients’ executive functioning scores (CalCap RT, D-KEFS achievement scores) and personality profiles were significantly different from TBI patients without DLPFC lesions and control-group participants. Although VLSM analyses reveal regions that are stochastically related to behavioral outcome scores, this analytical approach disambiguates whether VLSM results are generally confounded with TBI, as well as discern whether injury to a given region coincides with clinically relevant personality differences between lesioned patients and normal controls.

**RESULTS**

**Exploratory VLSM analyses**

All VHIS lesion patients with completed NEO-PI-R batteries were included in analyses (N = 190; see Figure 1 for lesion overlay map, including all patients entered into VLSM analyses). Initial VLSM analyses revealed that volume loss in left prefrontal structures, including DLPFC (BA 9), was related to high, but not low, neuroticism factor scores (BAs 9, 32, 44; see Table 2 and Figure 2(A)–(C)) and anxiety facet scores (BAs 9, 32, 45, 46; see Table 1 and Figure 2(D)–(F)). Conversely, volume loss in a comparable region of left prefrontal structures, including DLPFC (BA 9),

**Figure 1.** Lesion overlay map: VLSM analysis. Overlay map depicting lesion size in VHIS patients entered into VLSM analyses with NEO scores. Lesion coverage area depicts a minimum of four overlapping lesions per voxel.

*Note:* Warmer colors (yellow and red) indicate greater overlap in lesion location across subjects.

**Table 2**

<table>
<thead>
<tr>
<th>x</th>
<th>y</th>
<th>z</th>
<th>Cluster size</th>
<th>Z-score neuroticism trait</th>
<th>p-Value</th>
<th>Z-score anxiety facet</th>
<th>p-Value</th>
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<td>3.15</td>
<td>&lt;.001</td>
<td>3.78</td>
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</table>

*Note:* Neuroticism trait and anxiety facet Z-scores are normed against the population.
was related to low, but not high, conscientiousness trait scores. This volume loss was also related to low self-discipline facet scores (BAs 9, 46; see Table 3 and Figure 3(A)–(C)).

Effect sizes for the VLSM analyses (Cohen’s $d$), using a $z$-value of 2.57 and $p < .005$, ranged from .49 to 1.30; however, most significant voxels had $z$-values greater than 2.57. See Figure 4 for the lesion overlay map of all patients with left BA 9 damage. VLSM analyses were also conducted on the other personality factors and their facets (i.e., extraversion, openness, and agreeableness) and were not related to volume loss in the DLPFC.

**Hypothesis-driven comparisons between DLPFC patients, non-DLPFC patients, and normal controls**

VLSM analyses indicated that volume loss in left BA 9 was uniquely associated with neuroticism factor, anxiety facet, conscientiousness factor, and self-discipline facet scores. These analyses, however, do not provide insight in to whether VLSM results are confounded with TBI; whether injury to a given region coincides with clinically relevant personality differences between lesioned patients and normal controls; and whether there were unique relationships between volume loss in DLPFC, personality, and executive function. Thus, we next examined whether patients with large lesions in left BA 9 ($n = 19$) differed from patient controls with lesions in other anatomical locations ($n = 108$) and normal volunteers ($n = 51$) with respect to neuroticism and conscientiousness, as well as executive function (CalCAP and D-KEFS; see Table 4 for correlations among all variables of interest). Left BA 9 patients were selected if their volume loss was greater than the median volume loss among all left BA 9 patients.

Patient controls were selected if BA 9 was intact, bilaterally. All normal controls in the Phase III VHS registry were included in these analyses. Subsequently, one-way ANOVAs confirmed significant group differences on neuroticism anxiety facet scores ($F(176) = 4.42$, $p < .05$, $\eta^2 = .05$; see Figure 5(A)), conscientiousness self-discipline facet scores ($F(176) = 3.90$, $p < .05$, $\eta^2 = .04$; see Figure 5(B)), and both CalCAP Choice RT ($F(165) = 4.33$, $p < .05$, $\eta^2 = .05$; see Figure 5(C)) and D-KEFS overall weighted achievement scores ($F(171) = 8.43$, $p < .01$, $\eta^2 = .07$; see Figure 5(D)).

Post-hoc tests using the Bonferroni adjustment for multiple comparisons (18 comparisons total) showed that patients with large lesions in left BA 9 had higher neuroticism anxiety facet scores ($M = 60.06$, SD = 10.97) than either patient controls ($M = 51.57$, SD = 9.91; $\Delta = 8.49$, $p < .05$, $d = .81$) or normal controls ($M = 52.49$, SD = 12.92; $\Delta = 7.57$, $p < .05$, $d = .63$). There were no significant differences between patient controls’ and normal controls’ anxiety scores ($\Delta = -.92$, $p = .87$, $d = .08$). Left BA 9 patients reported lower self-discipline facet scores ($M = 39.41$, SD = 10.71) compared with patient controls ($M = 46.97$, SD = 10.07; $\Delta = -7.56$, $p < .05$, $d = .73$). A similar trend was observed between left BA 9 patients and healthy controls but did not reach significance ($M = 44.88$, SD = 11.87; $\Delta = -5.47$, $p = .20$, $d = .48$). There were no significant differences between patient controls’ and normal controls’ self-discipline scores ($\Delta = 2.09$, $p = .745$, $d = .19$) (see Figure 4(B)). Patients with large lesions in left BA 9 were also slower (i.e., had higher RTs) in CalCAP Choice tasks ($M = 475.24$, SD = 99.10) than with

\footnote{Volume loss was calculated based on estimations of the anatomical local of left BA 9 in patients’ native space and was not derived from VLSM maps in previous analyses. This limits the likelihood that any statistical relationships between volume loss and behavioral scores were spurious or artificially inflated (c.f. Vul, Harris, Winkielman, & Pashler, 2009). To determine whether selecting patients with larger BA 9 lesions biased analyses, we also probed for differences between patient controls, healthy controls, patients with larger BA 9 lesions, and patients with smaller BA 9 lesions on executive function and personality measures. The overall comparisons remained nearly identical. Differences between the conditions were found for D-KEFS scores ($p = .001$), CalCAP Choice RT ($p < .01$), anxiety ($p < .03$), and self-discipline scores ($p < .05$). Post-hoc Bonferroni comparisons indicated that patients with B9A lesions differed from patient and health controls on D-KEFS scores, CalCAP Choice RT, and anxiety scores (but only marginally compared with healthy controls on this variable). High BA 9 and low BA 9 groups only differed from one another on the anxiety facet score ($p = .05$). We also probed for differences between these conditions and other personality variables. There were no group differences between extraversion ($p = .53$), openness ($p = .27$), or agreeableness ($p = .58$).}
either patient controls ($M = 432.42$, $SD = 55.01$; $\Delta = 42.82$, $p = .05$, $d = .53$) or healthy controls ($M = 423.42$, $SD = 62.36$; $\Delta = 51.25$, $p < .05$, $d = .63$), while controls were not significantly different from one another ($\Delta = 8.44$, $p = 1.00$, $d = .15$).

Finally, left BA 9 patients showed lower D-KEFS overall weighted achievement scores ($M = 7.29$, $SD = 4.48$) than either patient controls ($M = 10.40$, $SD = 2.82$; $\Delta = −3.11$, $p < .001$, $d = .83$) or normal controls ($M = 10.66$, $SD = 2.99$; $\Delta = −3.37$, $p < .001$, $d = .88$). There were no significant differences between patient controls’ and normal controls’ D-KEFS achievement scores ($\Delta = −.26$, $p = .874$, $d = .09$).

Finally, to determine whether executive function mediated the relationship between BA 9 volume loss and personality factors and facets of interest, following the guidelines of Muller, Judd, and Yzerbyt (2005), a series of hierarchical regression analyses were conducted. Table 5 provides a complete summary of results. Overall, these analyses provide some evidence that executive function, and RTs on the CalCAP Choice tasks specifically, partially mediated the relationships between left BA 9 volume loss and neuroticism, anxiety, and self-discipline. Thus, while BA 9 appears to play some role in neuroticism, anxiety, and self-discipline via executive function processes, there is also likely a more complex, yet unknown pathway that qualifies these relationships.

**DISCUSSION**

In this study, we find support for the hypothesis that facets of two different personality dimensions—neuroticism and conscientiousness—depend at least
in part on a common neuroanatomical infrastructure, the DLPFC. Exploratory VLSM analyses revealed large clusters of lesion area within the left DLPFC that statistically differentiated high and low NEO scores on both neuroticism and conscientiousness factors and facets. In addition, we found that personality and executive function measures differentiated patients with lesions in the DLPFC from both patient and normal controls. Taken together, these findings are consistent with past research but are highly unique given the extent to which we can associate volume loss in a given brain region with the expression of a

**Figure 3.** Voxel-Based Lesion-Symptom Maps: low trait conscientiousness and self-discipline facet. (A–C) VLSM maps for residualized low conscientiousness factor scores (left BA 9). VLSM maps for low conscientiousness (self-discipline) facet scores (left BA 9). Highlighted pixels are significant at $p < .001$.
*Note:* Brighter colors indicate stronger statistical effects.

**Figure 4.** VHIS left BA 9 Lesion Overlay Map. (A, B) Overlay map depicting lesion size in VHIS patients with damage to the left BA 9, in (A) sagittal and (B) coronal planes. Lesion coverage area depicts a minimum of four overlapping lesions per voxel.
*Note:* Warmer colors (yellow and red) indicate greater overlap in lesion location across subjects.
## TABLE 4
Correlations between variables of interest and patients or controls

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</thead>
<tbody>
<tr>
<td>Healthy/patient controls</td>
<td>Delis–Kaplan</td>
<td>0.003</td>
<td>0.275**</td>
<td>0.262**</td>
<td>0.000</td>
<td>-0.161</td>
<td>-0.185</td>
<td>0.066</td>
<td>-0.094</td>
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<tr>
<td>CaCap</td>
<td>-0.081</td>
<td>0.558</td>
<td>-0.040</td>
<td>-0.026</td>
<td>0.008</td>
<td>-0.014</td>
<td>0.100</td>
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<tr>
<td>Neuroticism</td>
<td>-0.397**</td>
<td>-0.094</td>
<td>0.885**</td>
<td>0.280**</td>
<td>-0.261**</td>
<td>-0.266**</td>
<td>-0.056</td>
<td>-0.162</td>
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</tr>
<tr>
<td>Neuroticism: anxiety</td>
<td>-0.342*</td>
<td>-0.012</td>
<td>0.902**</td>
<td>0.219*</td>
<td>-0.245*</td>
<td>-0.178</td>
<td>-0.118</td>
<td>-0.138</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>0.255</td>
<td>0.057</td>
<td>-0.216</td>
<td>-0.274</td>
<td>0.146</td>
<td>-0.513**</td>
<td>0.782**</td>
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<tr>
<td>Conscientiousness: self-discipline</td>
<td>0.417**</td>
<td>-0.026</td>
<td>0.721**</td>
<td>0.231*</td>
<td>0.023</td>
<td>-0.020</td>
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<tr>
<td>Extraversion</td>
<td>0.082</td>
<td>-0.110</td>
<td>0.574**</td>
<td>-0.604**</td>
<td>0.100</td>
<td>0.244</td>
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<tr>
<td>Openness</td>
<td>1.138</td>
<td>-0.185</td>
<td>0.397**</td>
<td>0.442**</td>
<td>0.194</td>
<td>0.214</td>
<td>0.478**</td>
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<tr>
<td>Agreeableness</td>
<td>0.072</td>
<td>-0.197</td>
<td>-0.058</td>
<td>0.084</td>
<td>0.120</td>
<td>0.206</td>
<td>0.213</td>
<td>0.019</td>
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<tr>
<td>BA 9 damage</td>
<td>Delis–Kaplan</td>
<td>-0.615**</td>
<td>-0.245</td>
<td>-0.220</td>
<td>0.051</td>
<td>0.086</td>
<td>-0.357</td>
<td>-0.424</td>
<td>-0.341</td>
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</tr>
<tr>
<td>CaCap</td>
<td>-0.301</td>
<td>0.241</td>
<td>-0.560*</td>
<td>-0.552*</td>
<td>-0.221</td>
<td>0.390</td>
<td>0.550*</td>
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<tr>
<td>Neuroticism</td>
<td>0.908**</td>
<td>-0.295</td>
<td>-0.576*</td>
<td>-0.280</td>
<td>-0.066</td>
<td>-0.500*</td>
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<tr>
<td>Neuroticism: anxiety</td>
<td>-0.431</td>
<td>-0.607**</td>
<td>-0.186</td>
<td>-0.126</td>
<td>-0.528*</td>
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</tr>
<tr>
<td>Conscientiousness</td>
<td>0.859**</td>
<td>0.127</td>
<td>0.056</td>
<td>-0.008</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Conscientiousness: self-discipline</td>
<td>0.348</td>
<td>0.134</td>
<td>0.178</td>
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<td></td>
</tr>
<tr>
<td>Extraversion</td>
<td>0.272</td>
<td>0.484*</td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Openness</td>
<td>0.283</td>
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</table>

Notes: Correlations for healthy controls are presented above the diagonal space. Correlations for patients’ controls are presented below the diagonal space. All NEO scores represent t-scores. Numbers above empty space represent correlations between patients’ controls and the variables of interest. Numbers below represent correlations between healthy controls and the variables of interest. ***, Correlation is significant at the .01 level (two-tailed);*, correlation is significant at the .05 level (two-tailed); Delis–Kaplan, Total Weighted Achievement Score Item total scaled score; CaCap, Condition 3: Mean RT.
specific psychological construct in a manner that only lesion studies allow.

Our findings suggest that consistent with a psychological constructionist approach (Feldman-Barrett, 2009; Lindquist et al., 2012), the psychometrically distinct personality dimensions of neuroticism and conscientiousness may depend in part on a shared neuroanatomical infrastructure. According to the
psychological constructionists, even though a given brain region can perform more specialized operations, e.g., the occipital lobe’s role in vision, it is likely recruited by neural networks to subserve multiple psychological processes, including cognition, emotion, and executive function. This approach is intriguing from an evolutionary perspective given that the human brain both evolved in contexts very different from current environments and is defined by its ability to adapt to myriad novel situations. Such restraints would require different neural regions to contribute to many psychological processes, both predictable and novel.

To this end, our findings indicate that neuroticism, anxiety, conscientiousness, and self-discipline at least partly relied on the integrity, or lack thereof, of DLPFC, possibly because of this region’s central role in executive function. This stands to be an important contribution to the personality neuroscience literature, which has found more evidence for dissociable neural systems that uniquely contribute to independent personality dimensions to date (see DeYoung & Gray, 2009; DeYoung et al., 2010; but also see Omura et al., 2005). To be certain, a dissociation approach has been integral for identifying key neural structures involved in specific personality traits. However, it is also likely that some personality dimensions rely on goal-directed behavior as well as the inhibition of thoughts, goal states, or emotions that conflict with superordinate goals. Given this, certain traits will heavily rely on similar neurocognitive infrastructures such as lateral PFC, which serves an integral role itself in multiple executive functions, including task switching, working memory, inhibition, attention, and top-down control (e.g., Banich, 2009), and hierarchical computations (Badre, 2008). This study provides evidence for this conjecture and also impetus for the development of new models of personality that are more flexible and identify commonalities among psychometrically distinct personality traits. It is also worth noting that given the DLPFC is the slowest region to mature (Giedd & Rapoport, 2010), our findings suggest that neuroticism and conscientiousness might develop more slowly and are more susceptible to modification by experience than the other traits. Indeed, personality traits in general show much less consistency during formative years of development (ages 0–30 years) and typically steadily increase in consistency until individuals reach ages of 30 years and beyond (Roberts & DelVecchio, 2000). This would coincide with other findings linking prefrontal cortical development with more complex regulatory processes such as self and emotion regulation (Lewis & Todd, 2007).

This study also reveals how injury-related morphological differences in the brain were related to specific personality facets that comprise the conscientiousness and neuroticism trait dimensions. We found that damage to the DLPFC and the executive processing system is related to the expression of both neuroticism and conscientiousness, as well as certain facets of these traits. In the case of neuroticism, damage to the DLPFC was associated with the latent anxiety that coincides with neuroticism (see Knutson et al., 2001). Similarly, we found significant associations for certain facets of conscientiousness, but not others; self-discipline was related to DLPFC integrity, but facets like order were not. Conceptually, these are intuitive findings; facets reflecting integrity or impairment of attention and focus were most closely associated with lesions in the DLPFC. We found no evidence of association between DLPFC integrity and facets related to impulsivity and emotional reactivity, which are more likely served by threat-detection and reward system architecture (DeYoung et al., 2010; Omura et al., 2005; Wright et al., 2006, 2007).

With respect to the need for a flexible model of personality mentioned above, these findings suggest that exploring how specific neural systems contribute to nuanced facets of personality traits could be an important first step in identifying commonalities among different personality traits and in developing more predictive models of personality profiles.

Our findings are not without caveat. Like most research in personality neuroscience, we relied on self-report measures. We did not, furthermore, have pre-injury reports of personality profiles to compare with post-lesion reports, making it difficult to conclude with certainty which statistical relationships are unique to a disruption in normal neurological functioning through TBI. These findings could benefit from TMS studies, which could examine the impact of temporary lesions in the DLPFC on neurotic and conscientious behavior. Nevertheless, our findings are corroborated by past research which has demonstrated relationships among DLPFC and both conscientiousness (DeYoung et al., 2010) and neuroticism (Eisenberger et al., 2005).

In conclusion, our findings add to the field of personality neuroscience by demonstrating that the structural integrity of DLPFC, or lack thereof, is directly associated with the expression of neuroticism and conscientiousness. More important than highlighting a new neural substrate of these traits, we demonstrate that psychometrically independent personality dimensions are subserved by a similar neuroanatomical infrastructure. In addition, we expose the nature of the involvement of DLPFC in the expression of each
trait by isolating the specific facets that are most affected by TBI. Such findings can hopefully bring the field of personality neuroscience closer to identifying a comprehensive neural model for personality expression.

REFERENCES


