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RESEARCH ARTICLE

Pathological personality traits modulate neural interactions

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Abstract The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), includes an empirically supported dimensional model of personality pathology that is assessed via the Personality Inventory for the DSM-5 (PID-5). Here we used magnetoencephalography (MEG; 248 sensors) to evaluate resting-state neural network properties associated with the five primary DSM-5 maladaptive personality domains (negative affect, detachment, antagonism, disinhibition, and psychoticism) in 150 healthy veterans ("control" group) and 179 veterans with various psychiatric disorders ("psychopathology" group). Since a fundamental network property is the strength of functional connectivity among network elements, we used the absolute value of the pairwise correlation coefficient (aCC) between prewhitened MEG sensor time series as a measure of neural functional connectivity and assessed its relations to the quantitative PID-5 scores in a linear regression model, where the log-transformed aCC was the dependent variable and individual PID scores, age, and gender were the independent variables. The partial regression coefficient (pRC) for a specific PID-5 score in that model provided information concerning the direction (positive, negative) and size (absolute value) of the PID effect on the strength of neural correlations. We found that, overall, PID domains had a negative effect (i.e., negative pRC; decorrelation) on aCC in the control group, but a positive one (i.e., positive pRC; hyper-correlation) in the psychopathology group. This dissociation of PID effects on aCC was especially pronounced for disinhibition, psychoticism, and negative affect. These results document for the first time a fundamental difference in neural–PID relations between control and psychopathology groups.

Keywords Decorrelation · DSM-5 · Magnetoencephalography · Neuroimaging · Personality · PID-5

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Introduction

It is widely accepted that categorical classification systems reflect flawed assumptions about the nature of psychopathology (Widiger and Clark 2000; Widiger and Trull 2007), particularly in the case of personality pathology (Skodol et al. 2011; Widiger and Trull 2007). Consequently, the DSM-5 (American Psychiatric Association 2013) includes an empirically derived alternative model of personality pathology that permits dimensional assessment of pathological personality traits via the personality inventory for the DSM-5 (PID-5; Krueger et al. 2012). Akin to the structure of the Big Five (Costa and McCrae 1992), the PID-5 characterizes personality according to five higher-order personality domains, focusing on maladaptive expressions of those domains: negative affect, detachment, psychoticism,

antagonism, and disinhibition. Four of the PID-5 dimensions have clear empirical connections with maladaptive poles of the Big Five (psychoticism has a more complex relation with openness), and the model shares common ground with several other maladaptive personality models (Harkness et al. 1995; Widiger and Simonsen 2005), firmly grounding it within a robust empirical literature. Indeed, a number of studies support the validity of the pathological personality dimensions represented in the PID-5 (Anderson et al. 2013; Defruyt et al. 2013; Fossati et al. 2013; Hopwood et al. 2012; Krueger et al. 2012; Markon et al. 2013; Quilty et al. 2013; Suzuki et al. 2015; Thomas et al. 2013).

With support mounting for the construct validity of the DSM-5 trait domains, research aimed at elucidating the neural mechanisms underlying these personality dimensions will contribute to a better understanding of the biological basis of disordered personality traits. A substantial body of research in the burgeoning field of personality neuroscience has examined structural and functional neural correlates of normal range personality constructs (e.g., DeYoung et al. 2010; Ormel et al. 2013; Xu and Potenza 2012). However, across studies the findings have been inconsistent and have not uniformly converged on specific brain area involvement. For instance, structural neuroanatomical studies have found that neuroticism (which is similar to DSM-5 negative affect) correlates with several brain areas including the mid-cingulate cortex, caudate, middle temporal gyrus, cerebellum, dorsomedial prefrontal cortex, left medial temporal lobe, and precentral gyrus (DeYoung et al. 2010), orbitofrontal cortex (Wright et al. 2006), as well as total brain volume and intracranial volume (Bjørnebekk et al. 2013). Functional neuroimaging studies have implicated the middle frontal gyrus and precuneus (Kunisato et al. 2011), the bilateral superior temporal cortex and right posterior frontal lobe (Wei et al. 2014), the insular cortex (Deckersbach et al. 2006), superior parietal cortex (Sampaio et al. 2014), prefrontal regions including the medial prefrontal cortex (Kim et al. 2008), the hippocampus and midbrain in women (Sutin et al. 2010), and the middle temporal gyrus in men (Sutin et al. 2010). Neuroticism has also been found to predict resting-state functional connectivity between the precuneus and dorsomedial prefrontal cortex (Adelstein et al. 2011). Similar widespread brain involvement has been reported for other personality traits (e.g., DeYoung et al. 2010; Deckersbach et al. 2006; Kim et al. 2008; Kunisato et al. 2011; Wright et al. 2006). Although the findings differ across studies, they converge in supporting a biological basis of personality.

We take a different approach, as follows. First, to our knowledge, no prior research has examined neural correlates of the DSM-5 domains. The present study aims to fill that gap by evaluating neural mechanisms underlying the DSM-5 domains using magnetoencephalography (MEG). Second, we are unaware of any prior study to investigate personality traits with MEG. Hemodynamic neuroimaging techniques such as functional MRI (fMRI) and positron emission tomography (PET) are indirect indicators of brain function, given their reliance on blood flow and metabolism, and have comparatively poor temporal resolution. In contrast, electromagnetic neuroimaging techniques such as electroencephalography (EEG) and MEG have excellent temporal resolution and are direct measures of brain function, since they reflect integrated synaptic activity. In the case of EEG, however, electric currents become distorted as they pass through the skull and other tissues, in contrast to magnetic signals which pass undistorted and with minimal delay through soft tissues (Hämäläinen et al. 1993). The MEG signal reflects integrated synaptic activity with high fidelity and, therefore, is a highly accurate measure of brain activity, with excellent temporal resolution, in the millisecond range. Finally, given previous findings linking personality traits to widespread brain involvement (see above), the present study focuses on underlying neural mechanisms—specifically, synchronous cortical network communication properties—rather than focusing on involvement of specific brain areas.

Prior research using MEG has demonstrated that neural communication patterns are very similar and synchronous among healthy subjects (Langheim et al. 2006) and that deviations in synchronous neural interactions reflect brain disease and psychopathology (Engdahl et al. 2010; Georgopoulos et al. 2007, 2010). Moreover, dynamic properties of neural networks distinguish healthy from pathological brain function. Specifically, healthy brain function has been associated with decorrelated neural network activity (James et al. 2013). Decorrelation, or reduced strength of correlation of neural activity signals, is a key for enhancing efficiency of information processing (Kohn 2007). The importance of neural network decorrelation is well established but has primarily been studied in relation to the visual system (Atick 1992; Barlow and Földiák 1989; Ecker et al. 2010; Kohn 2007; Vanni and Rosentrom 2011; Vinje and Gallant 2000). We recently investigated decorrelation in relation to healthy versus pathological outcomes in veterans exposed to potentially traumatic events. Specifically, we demonstrated that decorrelated neural networks distinguished resilient veterans from those with posttraumatic stress disorder (PTSD) (who showed almost no evidence of decorrelation), and theorized that decorrelation underlies psychological adaptation (James et al. 2013).

In the present study, we sought to evaluate MEG-derived, resting-state neural communication patterns and neural network properties in relation to the DSM-5 personality domains in healthy control veterans and veterans diagnosed with psychological disorders. Resting-state studies permit characterization of underlying

neural processes that are neither specific to a given task nor related to task performance yet may vary across populations. These properties make task-free functional neuroimaging studies optimal for investigating correlates of personality traits. Based on our previous findings, we expected healthy functioning, but not psychopathology, to be characterized by decorrelated network activity. Given the relatively limited research on resting-state neural correlates of personality traits and the absence of prior research on neural correlates of the DSM-5 traits, specifically, we did not make predictions about trait-specific network activity.

Materials and methods

Participants

Study participants were 150 control veterans (141 males; mean group age 59.9) and 179 veterans with psychopathology (142 males; mean group age 52.6 years) taking part in a larger study evaluating neural functioning in veterans with and without PTSD and related disorders. Individuals with central nervous system disorders (e.g., Parkinson's disease, dementia, cerebral vascular accidents) or cardiac pacemakers or other imbedded ferrous metal (due to magnetic effects on MEG) were excluded. Individuals with documented substance dependence based on chart review were not recruited. Participants completed the Edinburgh Handedness Inventory (Oldfield 1971), the PID-5 (Krueger et al. 2012), diagnostic interviews, and underwent a MEG scan. The study protocol was approved by the Institutional Review Board at the Minneapolis VA Medical Center and was performed in accordance with the ethical standards outlined in the Declaration of Helsinki. All subjects provided written informed consent prior to participating in the study.

Personality assessment

The Personality Inventory for DSM-5 (PID-5; Krueger et al. 2012) is a 220-item questionnaire used to measure maladaptive personality traits as characterized in the DSM-5. Responses are selected from a four-point scale ranging from 0 ("very false or often false") to 3 ("very true or often true"). The items represent 25 empirically derived facets that load onto 5 higher-order personality domains: negative affect, detachment, antagonism, disinhibition, and psychoticism. The domain scales were calculated according to the most recent guidelines (see http://www.psychiatry.org/practice/dsm/dsm5/online-assessment-measures#Personality). Internal consistency of the five domains was excellent (median = .94; range = .89–.95).

Diagnostic assessment

Axis I disorders were evaluated using the Structured Clinical Interview for DSM-IV Axis I disorders (First et al. 2002) and Clinician-Administered PTSD Scale for DSM-IV (Weathers et al. 1999) using DSM-IV-TR criteria. Control participants did not meet criteria for any current Axis I disorder or for lifetime diagnosis of PTSD. Diagnostic characteristics of the psychopathology study participants were as follows: current PTSD (including subthreshold PTSD, 73 %), MDD or depressive disorder (39 %), other anxiety disorder (12 %), lifetime history of PTSD (8 %), traumatic brain injury (7 %), and other Axis I diagnosis (e.g., adjustment disorder, 3 %). Fifty-seven percent of psychiatric participants met criteria for one disorder, 35 % for two disorders, and 7 % for three or more disorders.

MEG data acquisition

MEG data were acquired using a 248-channel axial gradiometer system (Magnes 3600WH, 4-D Neuroimaging, San Diego, CA), band-filtered between .1 and 400 Hz, and sampled at 1017.25 Hz while subjects fixated on a spot 65 cm in front of them for 60 s. Data with artefacts (e.g., eye blinks, saturation) were eliminated from further analysis.

Data preprocessing and analysis

Single trial MEG data from all sensors underwent "prewhitening" using Matlab (version 2011b) by applying a (50, 1, 1) AutoRegressive Integrative Moving Average (ARIMA) model to remove trends and autocorrelations (Box and Jenkins 1976), yielding practically white noise innovations. All possible pairwise zero-lag crosscorrelations (N = 30,628, given 248 sensors) were computed between the prewhitened innovations (i.e., synchronous neural interactions). Since we were interested in the strength of the correlation (and not its sign), we took the absolute value of the correlation and log-transformed it to normalize its distribution (Fig. 1). To assess the associations of the PID dimensions to correlation strength, we analyzed the data in two stages. First, we performed five multiple linear regressions per sensor pair, where the log-transformed correlation was the dependent variable, and each PID domain score, age, and gender were the independent variables. This analysis yielded 30,628 partial regression coefficients (pRC) per PID dimension, one for each sensor pair. We then pooled all pRC and performed an analysis of variance (ANOVA) where the pRC was the dependent variable and the two groups (Control, Psychopathology) and five PIDs were the independent variables. (Age and gender had already been accounted for in the regressions above). We carried out four successive ANOVAs, as follows. In the first ANOVA,

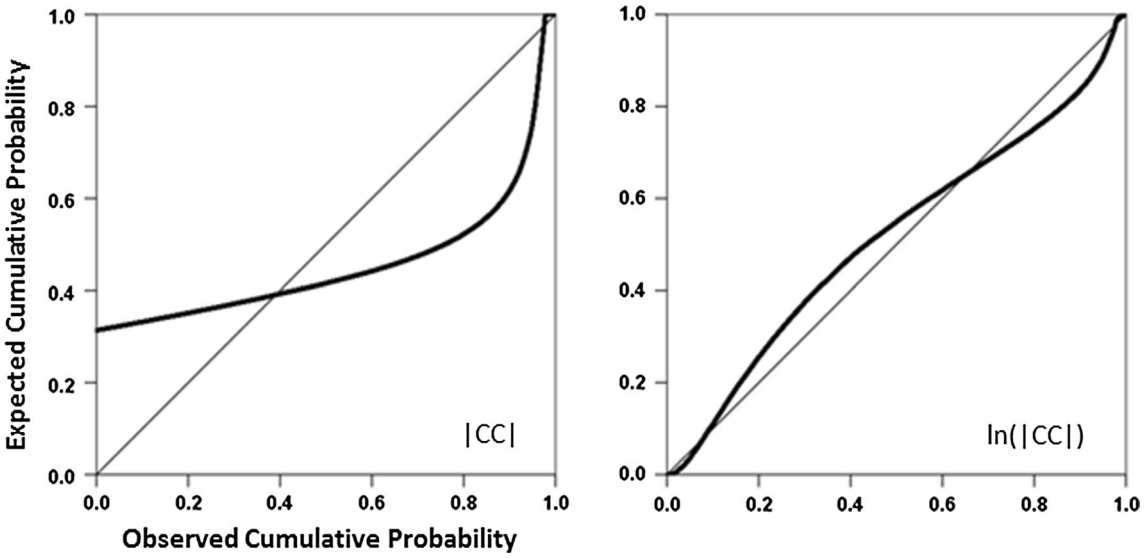

Fig. 1 Expected versus observed probability plots for |CC| (*left panel*) and its logarithmic transform (*right panel*) to illustrate the approximate normalization of the ln(|CC|) distribution; diagonal indicates perfect normality. $N = 30{,}628$ values from a single individual

Table 1 Descriptive statistics and correlations for PID-5 domains

Domain	Psychopathology		Controls						
	Mean (SD)	Range	Mean (SD)	Range	1	2	3	4	5
1 Negative affect$^{\pm}$	1.26 (.59)	.00–2.76	.60 (.43)	.00–2.04	–	.49*	.50*	.66*	.59*
2 Detachment$^{\pm}$	1.46 (.66)	.07–3.00	.59 (.43)	.00–2.16	.44*	–	.20	.50*	.48*
3 Antagonism	.64 (.44)	.00–1.93	.56 (.41)	.00–1.82	.26*	.10	–	.43*	.50*
4 Disinhibition$^{\pm}$	1.08 (.54)	.00–2.43	.51 (.43)	.00–2.04	.59*	.54*	.43*	–	.63*
5 Psychoticism$^{\pm}$	.93 (.59)	.04–2.56	.36 (.36)	.00–1.72	.58*	.56*	.43*	.73*	–

* Indicates $P < .001$

$^{\pm}$ Indicates mean difference significant at $P < .001$. Top portion of correlation matrix reflects control participants; bottom portion reflects psychopathology group

all pRCs were used, irrespective of their statistical significance; in the second and third ANOVA, pRCs were screened for their nominal significance, as a threshold, and ANOVAs were run on pRCs with probability levels $P < .05$ and $P < .0025$ to confirm that effects obtained with all pRCs were not due to low, nonsignificant pRC values. An additional ANOVA was then carried out with all pRCs as above but with a Hemisphere factor added (left, right, and interhemispheric) to evaluate possible differences between hemispheres. Finally, the distribution of pRC effects across different pairs of sensors was visualized in plots of pRCs with $P < .0025$ in sensor space.

Results

Descriptive statistics and correlations for the PID-5 domains are presented in Table 1. As expected, the psychopathology group endorsed significantly higher scores on the PID-5 domains (P's < .001), with the exception of antagonism ($P = .08$), which was low in both groups. A similar pattern of PID-5 domain score correlations was observed for both groups, with most domains moderately correlated.

To characterize the relations between the strength of functional neural interactions and PID-5 domains, we evaluated the pRC for each sensor pair and PID-5 domain score (accounted for age and gender). A positive pRC indicates positive covariation of the two measures, i.e., an increase in the strength of neural correlation as the PID-5 score increases. On the other hand, a negative pRC indicates negative covariation, i.e., a decrease in the strength of neural correlation as the PID-5 score increases, and vice versa. To assess various effects, we performed ANOVAs, as described in Methods. Both main effects of Group (Fig. 2, left panel) and PID (data not shown), as well as the

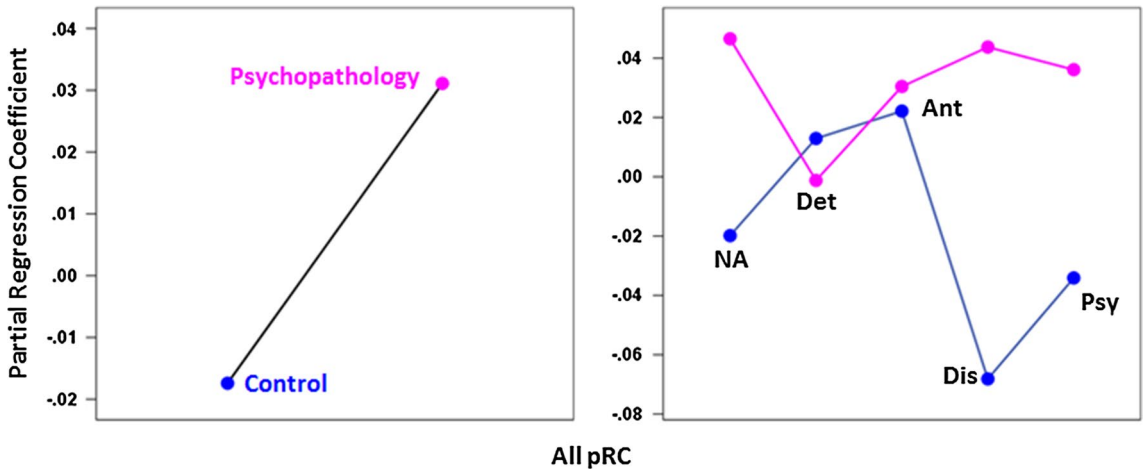

Fig. 2 Results of the ANOVA where all pRCs were used. *NA* negative affect, *Det* detachment, *Ant* antagonism, *Dis* disinhibition, *Psy* psychoticism. For the group plot (*left panel*): control pRC = −.015 ± .000452 (mean ± SEM), psychopathology pRC = .032 ± .000452; $N = 5$ PID × 30,628 = 153,140 per group. For the Group × PID plot (*right panel*), Control NA pRC = −.015 ± .001, Det pRC = .014 ± .001, Ant pRC = .024 ± .001, Dis pRC = −.068 ± .001, and Psy pRC = −.03 ± .001; Psychopathology NA pRC = .047 ± .001, Det pRC = .0 ± .001, Ant pRC = .031 ± .001, Dis pRC = .043 ± .001, and Psy pRC = .038 ± .001; $N = 2$ group × 30,628 = 61,256 per PID

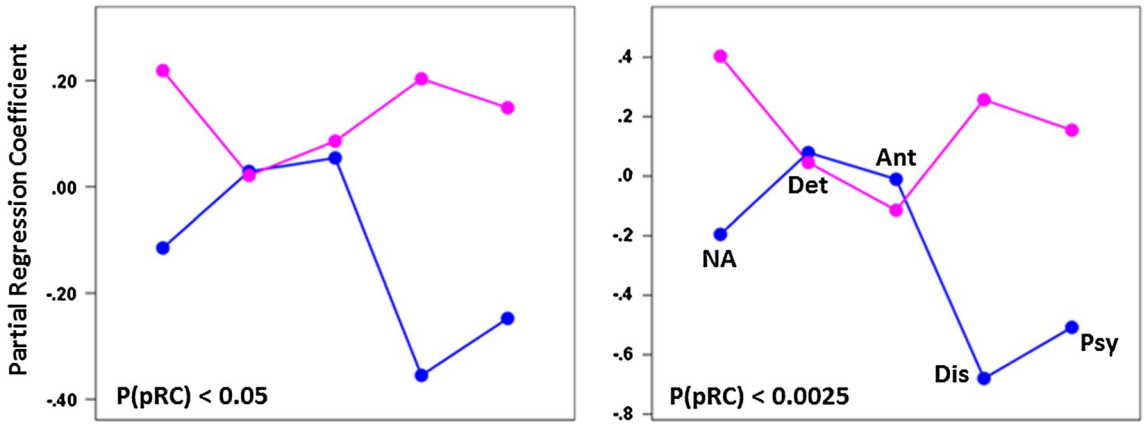

Fig. 3 Results of the ANOVA where pRCs at different significance thresholds were used, as indicated in the plot. For the $P < .05$ plot, Control NA pRC = −.115 ± .011, Det pRC = .029 ± .012, Ant pRC = .054 ± .013, Dis pRC = −.355 ± .009, and Psy pRC = −.248 ± .008; psychopathology NA pRC = .219 ± .009, Det pRC = .021 ± .011, Ant pRC = .086 ± .012, Dis pRC = .203 ± .009, and Psy pRC = .149 ± .007; $N = 2421$, 1903, 1621, 3071 and 4505 per PID above per group. For the $P < .0025$ plot, Control NA pRC = −.195 ± .075, Det pRC = .079 ± .097, Ant pRC = −.01 ± .1, Dis pRC = −.68 ± .047, and Psy pRC = −.508 ± .035; psychopathology NA pRC = .404 ± .06, Det pRC = .046 ± .069, Ant pRC = −.115 ± .042, Dis pRC = .257 ± .052, and Psy pRC = .155 ± .031; $N = 78$, 54, 116, 140, and 318 per PID above per group

Group × PID interactions (Fig. 2, right panel) were highly statistically significant (P's < .001). To assess the robustness of the effects, we carried out the same ANOVAs on statistically significant pRC values at different nominal thresholds ($P < .05$ and $P < .0025$, uncorrected). In both cases, main effects and interactions were highly significant (P's < .001) and the PID vs. Group patterns were also very similar (Fig. 3). Finally, Fig. 4 shows that similar results were obtained when the same ANOVA was performed separately for neural correlations within the left and right hemispheres, and for interhemispheric correlations; main effects and interactions were highly significant (P's < .001). The analyses above were done using all pRC values.

The sign and location in sensor space of the significantly modulated pRCs ($P < .0025$) are shown in Figs. 5, 6, 7 for negative affect, disinhibition, and psychoticism, respectively; these three PID dimensions showed the most consistent differences between the control and psychopathology groups, i.e., the most obvious Group × PID interactions (Figs. 2, 3, 4). As expected from the ANOVA

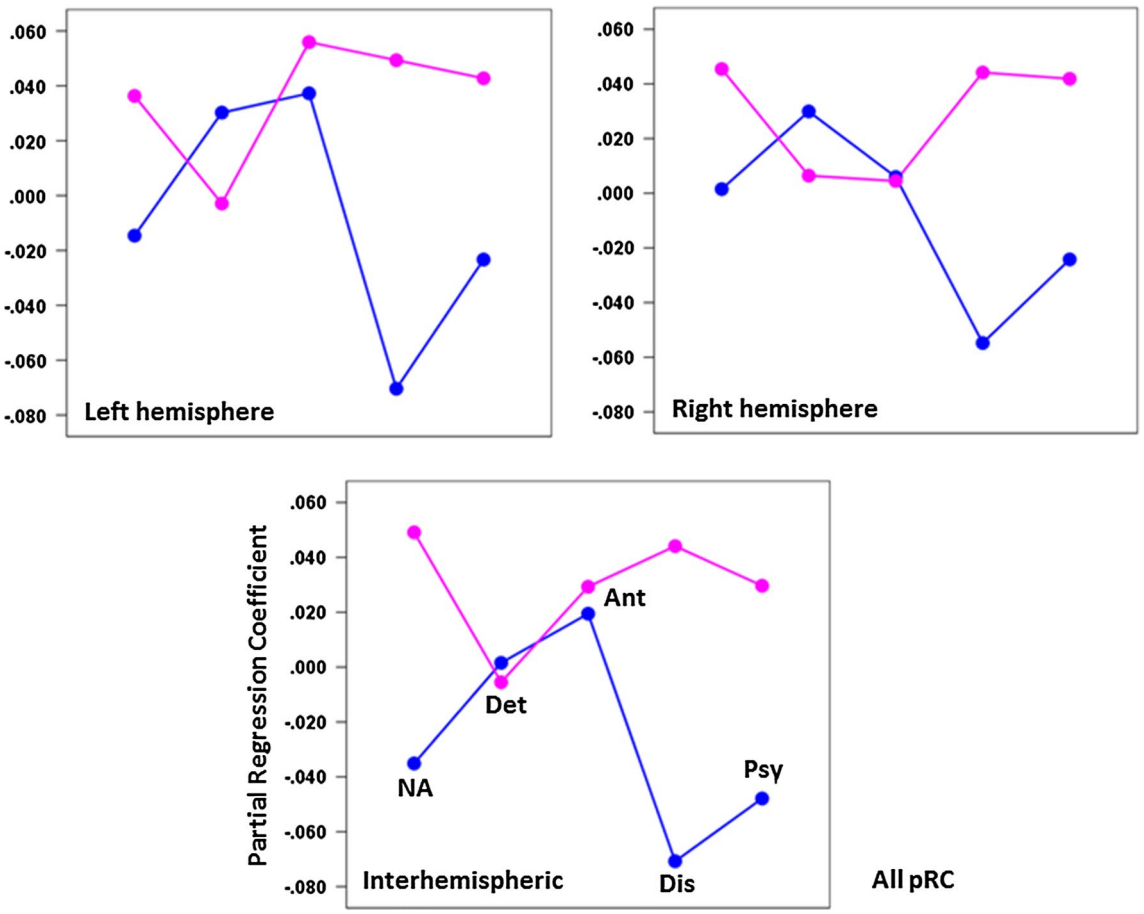

Fig. 4 Results of the ANOVA where all pRCs were used with Hemisphere as an added factor. It can be seen that the effects are very similar to those in Fig. 2 (*right panel*)

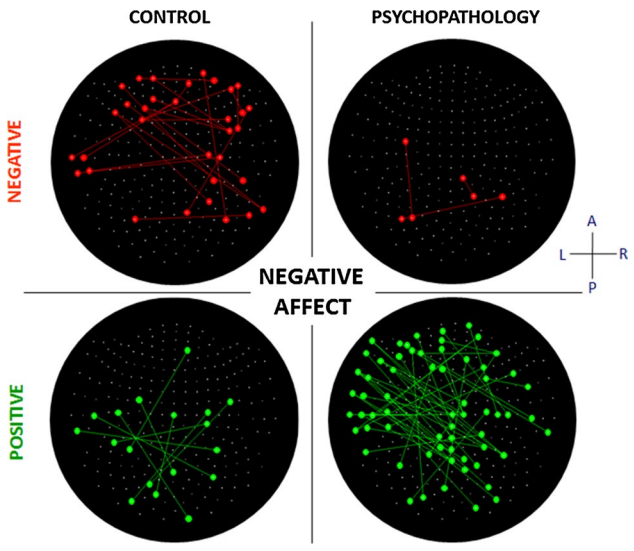

Fig. 5 Two-dimensional sensor-space plots depicting the negative (*red*) and positive (*green*) pRCs for negative affect in the control (*left*) and psychopathology (*right*) groups. *A* anterior. *P* posterior, *L* left, *R* right (colour figure online)

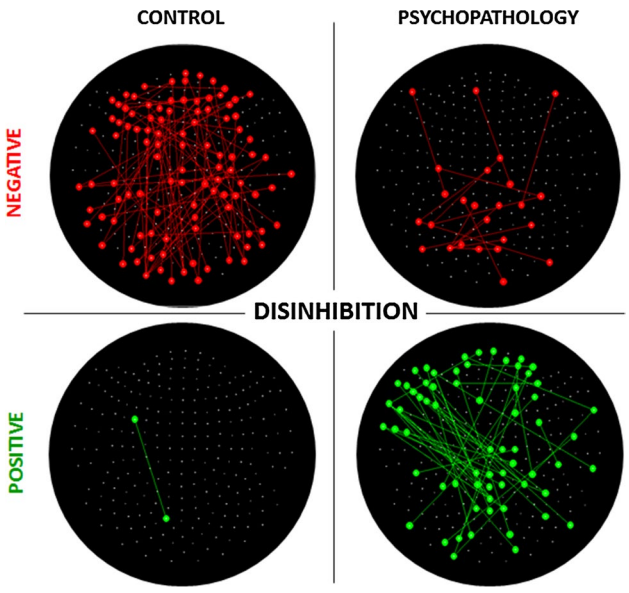

Fig. 6 Two-dimensional sensor-space plots depicting the negative (*red*) and positive (*green*) pRCs for disinhibition in the control (*left*) and psychopathology (*right*) groups (colour figure online)

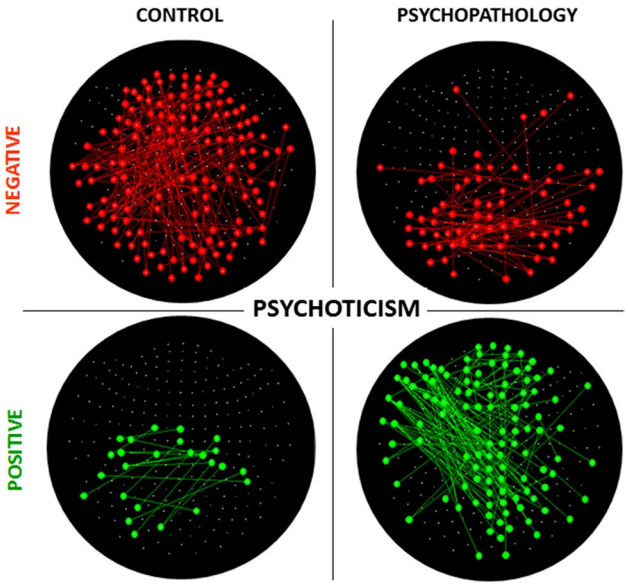

Fig. 7 Two-dimensional sensor-space plots depicting the negative (*red*) and positive (*green*) pRCs for Psychoticism in the control (*left*) and psychopathology (*right*) groups (colour figure online)

results above, negative pRCs dominated in the control group, whereas positive pRCs dominated in the psychopathology group for all three PIDs illustrated. Negative and positive pRCs were spatially restricted in distribution, depending on PID attribute, as follows. For negative affect (Fig. 5), negative pRCs in the control group largely involved interhemispheric connections, namely between frontal areas and between frontal and posterior areas (temporal on the left and parietal/parietooccipital on the right), whereas positive ones occupied mostly posterior areas. With respect to the psychopathology group, positive pRCs also involved predominantly interhemispheric connections, mainly between temporal/frontal areas on the left and midline/posterior parietal areas on the right. For disinhibition (Fig. 6), negative pRCs in the control group involved mostly interhemispheric connections between frontal and posterior areas bilaterally, broadly sparing the temporal lobe. With respect to the psychopathology group, positive pRCs involved long-range connections, mostly between temporal and frontal areas on the left and midline/posterior parietal areas on the right. Finally, for psychoticism (Fig. 7), negative, long-range pRCs were most predominant in the control group, again broadly sparing the temporal lobe. Regarding the psychopathology group, positive pRCs between left temporal and midline/posterior parietal were most prominent; negative pRCs were densely distributed bilaterally across posterior areas.

Finally, given the large number of study participants with trauma exposure, we performed all analyses controlling for lifetime trauma scores (King et al. 2006). Results were practically identical.

Discussion

The aim of the present study was to begin to elucidate the neural underpinnings of the DSM-5 alternative personality disorder model trait domains. Specifically, we sought to evaluate quantitatively possible covariation of neural interactions with DSM-5 personality domains. Overall, the findings demonstrate distinct neural network characteristics related to DSM-5 domains in healthy controls relative to those with psychiatric diagnoses.

With the exception of antagonism, all PID-5 trait scores significantly differed between groups; however, differences in the strength of neural functional connectivity were limited to disinhibition and psychoticism, and to a lesser extent, negative affect. This finding highlights the relative importance of those traits in terms of functional neural interactions and suggests that more substantial changes in functional neural connectivity may be involved in modulating (or, in the psychopathology group, expressing) them.

Regarding the nature of the neural connectivity differences observed, there were three major findings. First, negative pRCs dominated the relations between connectivity strength and PID in the controls, whereas positive pRCs were predominant in the psychopathology group. This reversal of the sign of the neural–PID relation, i.e., from decorrelation to hypercorrelation, indicates a fundamental difference in which PID characteristics are been processed by the brain in the two groups. Second, the most significantly affected neural modulations involved mostly long-range interactions, as can be appreciated by inspecting Figs. 5, 6, 7. And third, the spatial distribution of the brain–PID effects differed in the two groups, and among the PIDs. We discuss these points separately below.

We previously theorized that decorrelation, a neural phenomenon that has been implicated in information processing (Atick 1992; Barlow and Földiák 1989; Ecker et al. 2010; Kohn 2007; Vanni and Rosentrom 2011; Vinje and Gallant 2000), underlies psychological adaptation (James et al. 2013; Anders et al. 2015). The widely dispersed decorrelation observed in the control group, compared with the widespread hypercorrelation in the psychopathology group, further bolsters that theory. A substantial corpus of evidence (Eaton et al. 2011; Haslam et al. 2012) supports dimensional models of personality; consequently, it is not surprising that control participants endorsed some pathological personality responses. From our perspective, decorrelation may be viewed as a compensatory mechanism, permitting enough neural flexibility to reduce the manifestation of latent maladaptive personality characteristics, as

observed in control participants. In contrast, the hypercorrelation observed in the psychopathology group may serve to "lock in" maladaptive traits, thereby increasing the likelihood of future expression of maladaptation.

It is noteworthy that the direction of the effect of personality traits on neural functional connectivity reversed from decorrelation in the control group to hypercorrelation in the psychopathology group. A similar reversal in the direction of the strength of connectivity has been reported in studies comparing healthy controls to individuals with schizophrenia (Fletcher et al. 1999; Friston and Frith 1995). This highlights a fundamental difference in regard to functional connectivity between healthy and pathological functioning. That is not to say, however, that pathological personality traits necessarily reflect discrete phenomena. Rather, consistent with diathesis-stress models, it is likely that some healthy individuals may possess a vulnerability toward certain pathological personality traits, the expression of which is reduced by neural network decorrelation. Given certain circumstances (i.e., stressors), a range of biological changes to include changes in functional connectivity may occur, thereby altering the expression of the underlying traits. This suggests that within continua of personality traits, there may be biologically mediated cut-points that result in the expression of extreme psychopathology. The critical point at which neural networks switch from decorrelation to hypercorrelation and the mechanisms underlying that reversal remain to be elucidated.

Another notable finding concerns the spatial distribution of the neural networks involved. In the control group, PID-decorrelated neural networks were widely distributed across the cortex, particularly for disinhibition and psychoticism, while PID-hypercorrelation was sparse and primarily limited to posterior regions. In the psychopathology group, the reverse was evident—sparse PID-decorrelation, mostly in posterior regions, and widespread PID-hypercorrelation between left frontal and temporal regions with the midline and right hemispheric posterior parietal areas. In summary, these results indicate a broad mirroring in pRC sign, number and distribution between the control and psychopathology groups.

A common feature should also be noted, namely that pRCs of either sign and in both groups affected mostly long-range, interhemispheric connections, as evidenced also by the obvious lack of such modulation of interactions between nearby sensors. Similar to the asymmetric, interhemispheric findings observed here for the psychopathology group, prior research has demonstrated asymmetric activation of left (relative to right) frontal regions in relation to approach-related traits including anger (Harmon-Jones et al. 2010) and sensation-seeking (Santesso et al. 2008), and asymmetric right posterior parietal activation in relation to attentional deficits (Mevorach et al. 2006; Balle et al. 2013) and physiological arousal (Metzger et al. 2004). Indeed, aberrant personality reflects a complex interplay of cognitive, affective, physiological, and motivational processes that may be reflected in the left anterior-right posterior connectivity observed here.

The prominent group differences in MEG-derived neural functioning observed here are striking; however; the findings must be considered within the context of study limitations. First, individuals with personality disorders were not specifically recruited for this study; therefore, it is unclear whether the results obtained here would generalize to those diagnosed with primary personality disorders. However, because personality traits are continuous, we would expect differences between individuals with primary personality pathology and controls to be even more robust than those observed in the present study. Second, a large number of study participants met criteria for lifetime diagnoses of PTSD and other common posttraumatic mental health disorders (e.g., MDD), raising questions about the influence of trauma exposure on the pattern of findings. Our analyses, however, demonstrated that lifetime trauma exposure exerted virtually no influence on the findings. Furthermore, PTSD status does not appear to be driving our findings as our previous studies have demonstrated PTSD-related neural anomalies in the right temporal lobe (Engdahl et al. 2010; James et al. 2013), an area that was largely unaffected in the current study. On a related note, psychiatric disorders such as bipolar disorder, psychotic disorders, and substance use disorders were not represented in the current study; future studies may want to be more inclusive of various types of psychopathology to ensure adequate representation of personality pathology and generalizability of findings. Finally, the study participants were primarily middle-aged male veterans. We are currently collecting similar data on women veterans of various ages that will enable us to evaluate gender- and age-related differences in brain function associated with pathological personality traits. Additional studies evaluating the neural functioning associated with pathological personality in other non-veteran control and patient populations would be useful in establishing the stability of the findings, as would including additional covariates such as IQ and cognitive status.

The present study adds to the growing body of research highlighting the importance of task-free functional neuroimaging in the study of brain function and dysfunction. Task-free neuroimaging provides an unimpeded indication of inherent brain activity and abnormalities, which differ from those cued by external tasks (Broyd et al. 2009; Buckner et al. 2008). Because the MEG signal provides an accurate noninvasive direct measure of brain activity, the present study provides high fidelity assessment of brain function associated with DSM-5 personality traits.

Here we report results of the first study to examine the neural correlates of PID-5 domains using MEG. Overall, the findings suggest the following: (1) personality pathology is reflected in the strength of functional neural connectivity at the resting state; (2) healthy brain functioning is characterized by highly PID-decorrelated neural networks; (3) pathological functioning is characterized by PID-hypercorrelated networks; (4) these effects are most prominent for disinhibition, psychoticism, and negative affect; (5) the functional neural networks involved are widely dispersed with different brain areas involved in healthy versus pathological functioning. These findings begin to shed light on neural underpinning of the DSM-5 personality domains and provide additional evidence regarding the role of decorrelated brain networks in healthy function.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

Human subjects The study protocol was approved by the Institutional Review Board at the Minneapolis VA Medical Center and was performed in accordance with the ethical standards outlined in the Declaration of Helsinki.

Informed consent All subjects provided written informed consent prior to participating in the study.

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