

Connections that characterize callousness: Affective features of psychopathy are associated with personalized patterns of resting-state network connectivity

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ARTICLE INFO

Keywords:

Psychopathic traits
Default mode network
Person-specific
fMRI
GIMME
Community sample

ABSTRACT

Background: Psychopathic traits are hypothesized to be associated with dysfunction across three resting-state networks: the default mode (DMN), salience (SN), and central executive (CEN). Past work has not considered heterogeneity in the neural networks of individuals who display psychopathic traits, which is likely critical in understanding the etiology of psychopathy and could underlie different symptom presentations. Thus, this study maps person-specific resting state networks and links connectivity patterns to features of psychopathy.

Methods: We examined resting-state functional connectivity among eight regions of interest in the DMN, SN, and CEN using a person-specific, sparse network mapping approach (Group Iterative Multiple Model Estimation) in a community sample of 22-year-old men from low-income, urban families (N = 123). Associations were examined between a dimensional measure of psychopathic traits and network density (i.e., number of connections within and between networks).

Results: There was significant heterogeneity in neural networks of participants, which were characterized by person-specific connections and no common connections across the sample. Psychopathic traits, particularly affective traits, were associated with connection density between the DMN and CEN, such that greater density was associated with elevated psychopathic traits.

Discussion: Findings emphasize that neural networks underlying psychopathy are highly individualized. However, individuals with high levels of psychopathic traits had increased density in connections between the DMN and CEN, networks that have been linked with self-referential thinking and executive functioning. Taken together, the results highlight the utility of person-specific approaches in modeling neural networks underlying psychopathic traits, which could ultimately inform personalized prevention and intervention strategies.

1. Introduction

Psychopathic traits predict chronic criminal behavior and have been estimated to account for approximately \$460 billion of the annual cost of crime (Kiehl and Hoffman, 2011; Skeem et al., 2011). Unfortunately, there are no empirically-supported treatments for individuals with psychopathic traits (Reid et al., 2013), likely due, in part, to a lack of understanding of the etiology and individuality of psychopathic traits, which can be efficaciously examined using neural networks.

2. Neural networks and psychopathy

Etiological theories of psychopathy have centered either on emotional or attentional deficits. In the former, psychopathy is marked by deficient emotional processing via deficits in the paralimbic system (Blair, 2010; Kiehl, 2006). In the latter, psychopathy is marked by an attention bottleneck (i.e., failure to attend to contextual information when engaged in goal-directed behavior) due to impairments in top-down circuitry (e.g., prefrontal cortices; Larson et al., 2013; Newman

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<https://doi.org/10.1016/j.nicl.2020.102402>

Received 30 June 2020; Received in revised form 18 August 2020; Accepted 25 August 2020

Available online 28 August 2020

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and Baskin-Sommers, 2012). In general, neuroimaging investigations of these theories have focused on activation in specific regions (i.e., paralimbic and prefrontal). Importantly, recent empirical work and theory have suggested that abnormalities in connectivity within and across *multiple* networks could explain *both* emotional and attentional deficits associated with psychopathic traits (Contreras-Rodríguez et al., 2015; Espinoza et al., 2018; Ewbank et al., 2018; Geurts et al., 2016; Korponay et al., 2017; Leutgeb et al., 2016; Motzkin et al., 2011; Philippi et al., 2015; Tang et al., 2013; Waller et al., 2018; Yang et al., 2012; Yoder et al., 2014).

The Impaired Integration theory of psychopathy (II theory; Hamilton et al., 2015) suggests that connectivity within and between three resting-state networks underlies affective and cognitive processes involved in psychopathy, as they are implicated in perspective-taking, fear conditioning, and inhibitory control: (1) the default mode network (DMN), including the ventromedial prefrontal cortex (vmPFC) and posterior cingulate cortex (PCC); (2) the salience (or cingulo-opercular) network (SN), including the anterior insula and dorsal anterior cingulate cortex (ACC); and (3) the central executive (or frontoparietal) network (CEN), including the dorsolateral prefrontal cortex (dlPFC) and posterior parietal cortex (PPC) (Menon, 2011). The DMN is typically activated during resting-state and during tasks related to social cognition, autobiographical memory, theory of mind, and moral reasoning, while being de-activated during externally oriented, non-social thinking (Buckner et al., 2008; Reniers et al., 2012). In contrast, the CEN is de-activated at rest and activated during working memory and decision-making in goal-directed behavior, particularly when tasks are cognitively challenging (Menon and Uddin, 2010). The SN is de-activated at rest and activated during various neurocognitive functions, adjusting arousal and attention based on external cues and internal states to enable switching between other networks (Seeley, 2019; Sridharan et al., 2008). Despite being de-activated at rest, brain regions that constitute the SN and CEN still function synchronously during rest (Biswal et al., 2010; Greicius, 2008; Menon, 2011; Menon and Uddin, 2010), providing opportunities to investigate their interplay with each other and with the task-negative DMN. Such investigations reveal that the SN modulates the activity of both the CEN and DMN (Goulden et al., 2014). The II theory posits that psychopathy is characterized by abnormal functioning of the SN and DMN, but intact functioning of the CEN. Compromised SN and DMN functioning is thought to impact the integration of complex sensory information, which is key in emotional learning (Blair, 2017) and relies on attendance to and integration of external (e.g., emotional faces; social norms) and internal cues (e.g., emotions; desires) to inform decision-making and behavior. II theory also posits that the CEN functions normally when engaged, which explains why individuals with psychopathic traits do not consistently display cognitive control deficits (Hamilton et al., 2015).

Very few studies have explicitly examined connectivity within and between regions of the DMN, SN, and CEN, as outlined in the II theory. In fact, researchers have only recently begun to look at associations between resting-state (i.e., “task-free”) connectivity and psychopathic traits. Most studies have utilized seed-based approaches to examine the connectivity of regions and the choice of regions of interest has been guided by task-based studies of neural activation (e.g., the amygdala, which has been implicated in emotion processing paradigms; Contreras-Rodríguez et al., 2015; Korponay et al., 2017; Motzkin et al., 2011; Philippi et al., 2015). A few studies have used a region of interest approach to examine connectivity among *a priori*, albeit differential, nodes within the DMN, SN, and CEN. For instance, Contreras-Rodríguez et al. (2015) found increased positive connectivity between regions within the CEN in offenders with psychopathic traits compared to non-offender controls. Additionally, compared to offenders *without* psychopathic traits, Motzkin et al. (2011) found reduced connectivity between regions within the DMN in offenders with psychopathic traits, and Philippi et al. (2015) found reduced connectivity between the SN and CEN. Other studies have used a whole-brain approach. For example,

Espinoza et al. (2018) found psychopathic traits to be associated with impaired connectivity between the SN and DMN, with findings varying by brain region (i.e., ACC versus insula). Thus, the existing literature is limited, and findings do not clearly converge across studies.

One reason why findings do not converge is because most relevant work utilizes group averages, assuming all individuals display similar patterns of connectivity, despite evidence that neurobiological mechanisms of psychopathy differ across people (Baskin-Sommers et al., 2011; Efferson and Glenn, 2018; Gao and Raine, 2010) and features of psychopathy (Espinoza et al., 2018; Korponay et al., 2017; Philippi et al., 2015; Vermeij et al., 2018; Wolf et al., 2015). In fact, recent research has turned towards examining biological heterogeneity that could reveal underlying mechanisms of differing symptom presentations (Insel, 2014). By ignoring variation among individuals, average approaches may produce spurious connections in neural networks that do not accurately describe individuals (Gates and Molenaar, 2012; Smith et al., 2011). Researchers cannot be confident in their interpretation of results without first ensuring accurate modeling of neural networks. This point is particularly relevant to the study of psychopathy, given that individuals with high levels of psychopathic traits have been shown to vary in terms of behavioral phenotypes, clinical outcomes, and biological correlates (including neural mechanisms) (Latzman et al., 2019).

Another reason why findings from neuroimaging studies of psychopathy do not converge is that most have examined the *strength* of specific connections (Johanson et al., 2020) and failed to consider the way neural networks are arranged (i.e., “topology”; De Vico Fallani et al., 2014; Kaiser, 2011), which has been utilized in other neuroscience work. Network analytic approaches are well-suited for testing hypotheses of the II theory regarding overall network functioning and architecture by examining topological features, including the characteristics of relations between regions (i.e., nodes) within a network (e.g., number, length, direction of connections or ‘edges’). For instance, network “density” (i.e., number of connections in a sparse network) indicates the extent to which information travels between nodes within the same network or across different networks. Additionally, “node centrality” (i.e., number of connections into and/or out of a specific node) reflects the importance of a node within a network for facilitating communication between networks (De Vico Fallani et al., 2014; Kaiser, 2011). In this way, topology underlies information processing and has been shown to predict cognitive functioning (Cohen and D’Esposito, 2016).

Therefore, network topology approaches to resting-state fMRI have potential to reveal the neural architecture underlying “baselines” or “intrinsic” patterns of connectivity associated with psychopathic traits. However, only two studies have been conducted in adults using such an approach. In one study, psychopathic traits were associated with increased centrality of DMN and SN nodes (Lindner et al., 2018), whereas the other study did not find any significant associations with DMN or SN features (Tillem et al., 2019). Notably, these studies were limited by focus on either women from the community or incarcerated offenders. Regarding female populations, research suggests that there are gender differences in the expression of psychopathic traits, (Efferson and Glenn, 2018), highlighting the need to examine network topology in male samples. Regarding offender populations, psychopathic traits vary dimensionally in the community (Lilienfeld, 2018), and thus psychopathic traits may have a different etiology or presentation among individuals who have not been incarcerated for offenses (Gao and Raine, 2010). Indeed, with one exception (Lindner et al., 2018), all previous resting-state connectivity studies of psychopathy in adults have been conducted in offender populations (Contreras-Rodríguez et al., 2015; Espinoza et al., 2018; Korponay et al., 2017; Motzkin et al., 2011; Philippi et al., 2015; Tillem et al., 2019). Finally, neither study examined psychopathy at the facet-level. Previous studies have found that psychopathy consists of distinct symptom sets or ‘facets’: interpersonal (e.g., grandiosity, manipulation), affective (e.g., lack of remorse,

callousness), lifestyle (e.g., sensation-seeking, irresponsibility), and antisocial (e.g., violence, criminal versatility) (Dotterer et al., 2016; Mahmut et al., 2011; Neal and Sellbom, 2012; Neumann et al., 2012; Seara-Cardoso et al., 2012). Although highly correlated, these facets are characterized by unique behavioral deficits underpinned by distinct neural systems (Carré et al., 2013; Deming et al., 2018; Litzman et al., 2019; Vermeij et al., 2018). Thus, it is unclear whether the four facets are characterized by unique network features, as has been demonstrated using other resting-state approaches (e.g., Espinoza et al., 2018).

Novel network approaches now combine more traditional, group-level approaches with person-specific approaches, which assume that participants are heterogeneous and have data that should not be averaged. For example, group iterative multiple model estimation (GIMME; Gates and Molenaar, 2012) is a data-driven approach that creates person-specific networks by first mapping connections between nodes that are statistically meaningful at the group-level (i.e., found across the entire sample), and then adding connections that are statistically meaningful at an individual-level (i.e., are unique to a person) – all while providing connection estimates that are unique for individuals. Simulation studies show that GIMME outperforms other network approaches, including Granger causality and Bayes nets, particularly when data are heterogeneous (Gates and Molenaar, 2012). Empirical studies utilizing GIMME to understand neural mechanisms of psychopathology have demonstrated significant variability in neural network configurations across patients, highlighting biological heterogeneity within the same diagnosis (Beltz et al., 2018; Price et al., 2017). However, no studies have yet applied person-specific approaches to the mapping of neural networks underlying psychopathic traits.

3. Current study

The goal of this study was to delineate associations between psychopathy (including interpersonal, affective, lifestyle, and antisocial facets) and person-specific network connectivity within the DMN, CEN, and SN in an ethnically diverse, male community sample at heightened risk for antisocial behavior (Hyde et al., 2016). We used GIMME (Beltz and Gates, 2017; Gates and Molenaar, 2012) to generate person-specific connectivity maps for each participant, and we examined whether psychopathic traits were uniquely associated with network features (i.e., density; node centrality) across participants.

4. Methods and Materials

4.1. Participants

The final sample included 123 participants from the Pitt Mother & Child Project, a longitudinal study of 310 low-income, ethnically diverse boys and their families (Shaw et al., 2012). Families were recruited from Allegheny County Women, Infants, and Children Nutritional Supplement Clinics in 1991 and 1992 when the boys were 6 to 17 months of age (Shaw et al., 2003, 2012) and seen almost yearly from age 1.5–23 years. At the first assessment, mean per capita income of family members was \$2,892 per year, with a mean Hollingshead socioeconomic status score of 24.5, indicative of a working class-to-im-poverished sample. This sample is considered to be at heightened risk for antisocial behavior based on gender, familial socioeconomic status, and urbanicity, allowing us to examine hypotheses in a sample with a wide range of variability in psychopathic traits (Beck and Shaw, 2005; Gard et al., 2017; Hyde et al., 2016). All procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

At age 22 years, 255 participants from the original sample completed questionnaires, with a subsample of participants ($n = 180$) participating in magnetic resonance imaging (MRI) (Shaw et al., 2012).

The MRI component introduced some data loss (Table S1), resulting in 126 men with high quality resting-state fMRI data. Of the 126 participants, one was excluded because of a diagnosis of autism spectrum disorder, and two were missing data on psychopathic traits. Of the included participants ($n = 123$), most self-reported their race as European American ($n = 66$, 52.8%) or African American ($n = 45$, 36.6%; $n = 13$, 10.6% self-reported “other”). Participants reported a relatively low mean income ($M = \$13,770.30$, $SD = \$12,605.34$). The included 123 participants did not significantly differ from the original 310 participants in family income at recruitment ($t(309) = -1.58$, $p = .12$), mother’s education ($t(311) = -1.56$, $p = .12$), race ($\chi^2(3) = 1.18$, $p = .76$), or parent-reported externalizing behaviors ($t(277) = 0.204$, $p = .85$) (measured using the Child Behavior Checklist at age 2; Achenbach, 1991).

4.2. Measures

Self-reported psychopathic traits. We assessed psychopathic traits at age 22 using the 29-item Self-Report Psychopathy Short-Form (Neumann and Pardini, 2014; Paulhus et al., 2015). The items measured four dimensions of psychopathy: interpersonal manipulation (e.g., “I think I can beat a lie detector”), affective callousness (e.g., “I never feel guilty over hurting others”), erratic lifestyle (e.g., “I’ve often done dangerous things just for the thrill of it”), and criminal tendencies (e.g., “I have tried to hit someone with a vehicle”) (Neumann and Hare, 2008) (see Supplemental Materials).

Resting-state fMRI and preprocessing. Resting-state functional images were collected while participants were awake, passively viewing a fixation cross for 6 min. T2*-weighted images (TR = 2000; TE = 27; FOV = 24 cm; flip angle = 75°; 39 3.10 mm slices; 180 TRs) were acquired using a research-dedicated Siemens 3-T Tim Trio. We conducted standard preprocessing in FSL (<http://www.fmrib.ox.ac.uk/fsl>) (Jenkinson et al., 2012), including removal of the first four volumes, motion correction using MCFLIRT (Jenkinson et al., 2002), slice-timing correction, non-brain removal, co-registration to high resolution structural scans (MPRAGE), normalization to MNI 152 space using 12-dof linear registration in FLIRT, and spatial smoothing using a Gaussian kernel (6-mm) (see Supplemental Materials). ICA-AROMA was applied at the subject-level to remove motion-related artifacts (Pruim et al., 2015a, 2015b).

Eight *a priori* ROIs (network nodes) defined three networks (Goulden et al., 2014; Hamilton et al., 2015; Sridharan et al., 2008) (see Table S3 for coordinates): vmPFC and PCC for the DMN; right and left insula and the ACC for the SN; and right and left PPC and dlPFC for the CEN. These ROIs have been implicated in previous resting and task-based connectivity studies of the interplay among the DMN, SN, and CEN (Chiong et al., 2013; Goulden et al., 2014; Sridharan et al., 2008). Mean timeseries at each volume were extracted using subject-specific spherical ROI masks (see Supplemental Materials).

4.3. Analysis plan

We conducted analyses in two steps. First, we generated person-specific networks for each participant using GIMME. Second, we extracted network features from each participant’s map to examine associations with psychopathic traits across the sample. We conducted all analyses in MPlus version 8.2 (Muthén and Muthén, 2020) using robust maximum likelihood estimation which is robust to relaxed assumptions of the data (e.g., non-normality) (MLR; Yuan and Bentler, 2000) with the exception of GIMME, which relies on lavaan in RStudio (Lane and Gates, 2017).

GIMME. We submitted node timeseries for the 123 participants to GIMME, a sparse modeling approach that iteratively adds only statistically meaningful connections (i.e., improve fit of a null or less parameterized model) to a network. Most alternate approaches model all connections between regions (i.e., create saturated networks) and then

use forms of regularization to minimize weak connections, but this can be subjective and lead to networks that are denser than necessary to explain the raw data (Fornito, 2016). Sparse modeling approaches instead minimize spurious contemporaneous connections (Gates et al., 2010).

For each participant, GIMME generates a unified structural equation model (uSEMs; Gates et al., 2011), which includes both contemporaneous (i.e., one node predicts another in the same functional volume) and first order lagged (i.e., one node predicts itself or another at the next functional volume) connections. Each connection has a person-specific direction (i.e., positive or negative) and magnitude (reflected by beta weights). Thus, GIMME improves upon other network approaches that only model contemporaneous, zero-order correlations of activation between regions (Fornito, 2016; Gates and Molenaar, 2012). Moreover, GIMME includes a grouping algorithm to account for both homogeneity (in group-level connections) and person-specific heterogeneity (in individual-level connections) (Gates and Molenaar, 2012). By including a group-level structure (i.e., capitalizing on shared information across individuals) in addition to person-specific features, GIMME overcomes limitations of other approaches in which low signal-to-noise ratio induces unreliable estimates at the individual level (Gates and Molenaar, 2012; Smith et al., 2011).

GIMME begins by estimating 8 autoregressive terms (i.e., lagged prediction of each ROI by itself) in each participants' network (Beltz and Gates, 2017; Friston et al., 2000; Woolrich et al., 2001). Lagrange multiplier tests are then used to identify connections to estimate that are statistically meaningful at the group-level (i.e., significantly improve model fit for 75% of the sample). Next, Lagrange multiplier tests are again used to free connections that are statistically meaningful at the individual-level (i.e., significantly improve model fit for a given participant). Additionally, at several points during model fitting, non-significant connections are pruned if their influence changed with the addition of new connections (Gates and Molenaar, 2012). Model building ends when the network fits the data well, and final maps are evaluated with alternative fit indices, with two of four required to attain excellent fit (Brown, 2014): root mean squared error of approximation (RMSEA) ≤ 0.05 , standardized root mean residual (SRMR) ≤ 0.05 , comparative fit index (CFI) ≥ 0.95 , and non-normed fit index (NNFI) ≥ 0.95 .

Notably, GIMME produces models with first order lagged connections, meaning that the lagged connections represent estimates at one time point prior (i.e., one functional volume earlier), with the assumption that the model residuals are white noise (i.e., all temporal information is captured by the modeled connections). To verify this assumption, we submitted individual-level models to *a posteriori* validation (as described in Beltz and Molenaar, 2015; see Supplemental Materials). If the validation process indicates that the first order connections did not sufficiently capture all sequential dependencies in each participant's data (according to white noise tests), then higher order lagged connections (i.e., estimates at two or three functional volumes prior) were added to the model. Previous directed functional connectivity studies demonstrate that this is important for accurately modeling all connections in the network, especially in resting state data (Beltz and Molenaar, 2015).

Network features. We extracted several features from the final networks to characterize person-specific patterns of resting-state connectivity. To account for individual differences in total number of connections, we used proportions. We calculated separate indices of positive and negative features. Whereas positive connections are expected among brain regions within the same network, previous theory and empirical studies suggest that the DMN and task-positive networks such as the CEN are inherently anticorrelated, which leads to negative connections (Chai et al., 2012; Fox et al., 2005; Kelly et al., 2008).

Network density. For each participant, we calculated within-network density (i.e., number of connections between nodes within a network, regardless of whether they were contemporaneous or lagged) separately

for the DMN, SN, and CEN. We similarly calculated between-network density: DMN-SN, DMN-CEN, and SN-CEN.

Node centrality. As exploratory analyses, we calculated node centrality for each participant (i.e., number of connections involving the node, regardless of whether they were contemporaneous or lagged) to determine whether any regions were "hub-like" (i.e., high number of connections to and/or from this region), as in previous studies that have examined network organization (Lindner et al., 2018; Lu et al., 2017; Tillem et al., 2019, 2018; Yang et al., 2012).

Associations between network features and psychopathic traits. To examine associations between psychopathic traits and neural connectivity, we ran a multiple regression for each index of network density, including both positive and negative connections, which were correlated dependent variables. Total psychopathy score was the predictor. Participant self-reported race, substance use (mean score on Alcohol and Drug Consumption Questionnaire; Cahalan et al., 1969), monthly income, and framewise displacement (after motion correction) were covariates.

To examine associations between psychopathic traits and node centrality, we ran a multiple regression model separately for each network to determine if psychopathic traits were associated with node centrality, with positive and negative centrality of each node within a network included as correlated dependent variables. Total psychopathy score was the predictor, and the same covariates were included.

We used the Bonferroni method to correct for multiple comparisons across the five models we ran (i.e., within network density, between network density, SN node centrality, DMN node centrality, CEN node centrality; $p = .05/5 = 0.01$).

We followed up significant effects to determine whether associations with the total score were driven by the interpersonal, affective, lifestyle or antisocial facets. As these were exploratory analyses, we did not apply a Bonferroni correction. Finally, as noted previously, an advantage of GIMME is the inclusion of both contemporaneous and lagged connections, and so exploratory follow-up analyses included in the Supplemental Materials examined whether significant associations were driven by contemporaneous or lagged connections.

5. Results

5.1. Descriptives

Descriptives are presented in the Supplemental Materials. Of note, in general the means of the SRP-SF in the current sample were slightly higher than or comparable to those found in previous community samples (Gordts et al., 2017; Paulhus et al., 2015; Seara-Cardoso et al., 2019). However, the mean on the criminal tendencies facet was higher than previous community samples, indicating that this sample was indeed at somewhat higher risk than typical community samples (Gordts et al., 2017; Paulhus et al., 2015; Seara-Cardoso et al., 2019).

6. Person-specific network modeling

Final GIMME networks generally fit the data well (Average Fit: RMSEA = 0.04, SRMR = 0.05, CFI = 0.97, NNFI = 0.94) (see Supplemental Materials). There were no group-level connections, indicating substantial heterogeneity across participants. There were between 11 and 27 individual-level connections ($M = 17.85$, $SD = 3.76$). All models contained positive connections ($M = 14.35$, $SD = 2.70$) and most models (93%) contained negative connections ($M = 3.50$, $SD = 2.24$). Additionally, all models contained both contemporaneous and lagged connections ($M = 6.89$, $SD = 2.60$; $M = 9.82$, $SD = 1.54$, respectively). *A posteriori* model validation determined that a first order model fit the data well for 67 participants (54.5% of the sample), but that 16 participants (13%) required lag 2 connections and 40 participants (32.5%) required lag 3 connections. See details in Supplemental Materials.

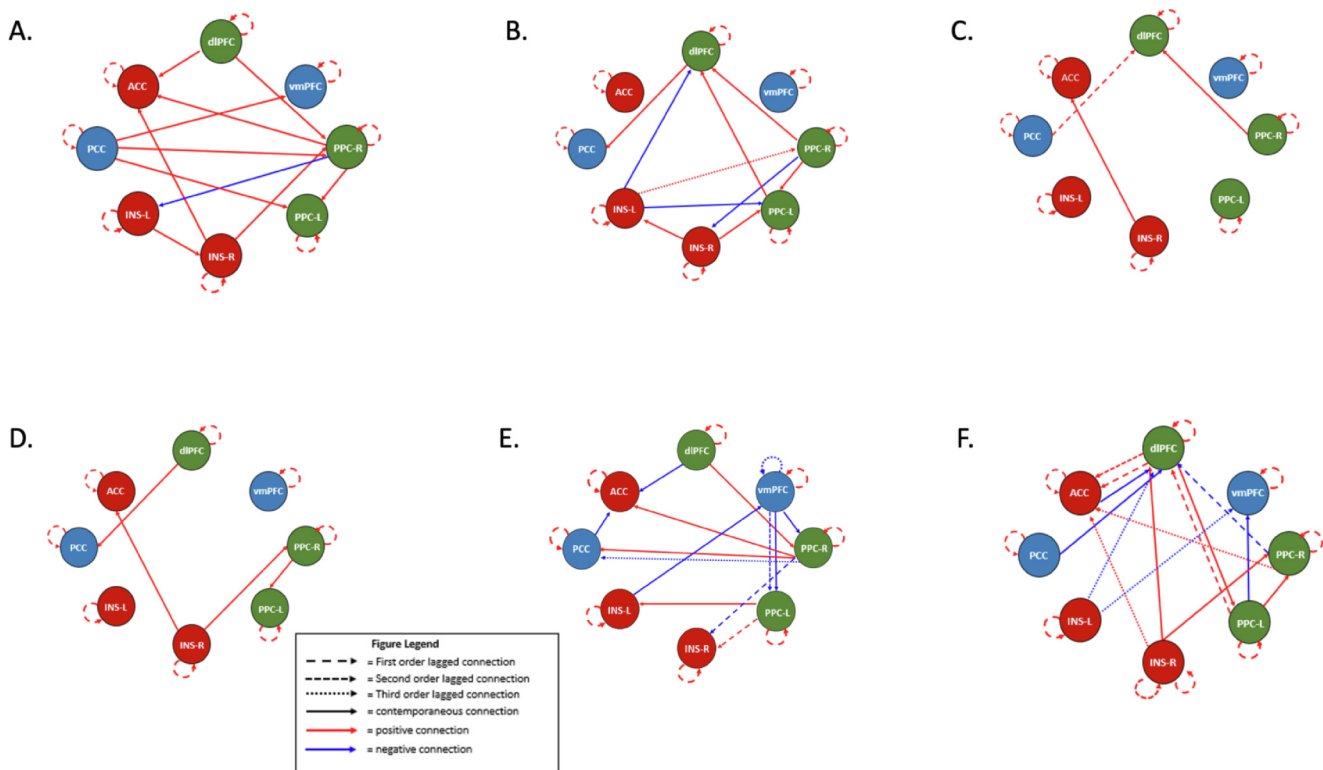


Fig. 1. Final GIMME networks for six illustrative participants. Solid lines depict contemporaneous connections and dashed lines depict lagged connections. Regions in red are within the salience network. Regions in blue are within the default mode network. Regions in green are within the central executive network. There was no group-level structure; thus, all lines depict individual-level connections (uniquely estimated for the participant) that also have associated β weights. Red lines depict connections with positive β weights and blue lines depict connections with negative β weights. 1A.) Participant with high levels of affective features (score = 23; Range in the sample 7 – 25); $\chi^2(73) = 106.06$, RMSEA = 0.05, SRMR = 0.05, CFI = 0.96, NNFI = 0.95. 1B.) Participant with high levels of lifestyle features (score = 23; Range in the sample 7 – 28); $\chi^2(75) = 105.77$, RMSEA = 0.05, SRMR = 0.05, CFI = 0.96, NNFI = 0.94. 1C.) Participant with first order model; $\chi^2(81) = 111.75$, RMSEA = 0.05, SRMR = 0.06, CFI = 0.95, NNFI = 0.93. 1D.) Participant with first order model; $\chi^2(80) = 102.50$, RMSEA = 0.04, SRMR = 0.06, CFI = 0.96, NNFI = 0.95. 1E.) Participant with second order model; $\chi^2(13) = 186.84$, RMSEA = 0.04, SRMR = 0.05, CFI = 0.96, NNFI = 0.91. 1F.) Participant with third order model; $\chi^2(19) = 259.25$, RMSEA = 0.04, SRMR = 0.04, CFI = 0.97, NNFI = 0.92. dIPFC = dorsolateral prefrontal cortex; vmPFC = ventromedial prefrontal cortex; PPC-R = right posterior parietal cortex; PPC-L = left posterior parietal cortex; INS-R = right insula; INS-L = left insula; PCC = posterior cingulate cortex; ACC = anterior cingulate cortex. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Fig. 1 depicts models from six illustrative participants to highlight the heterogeneity of networks across individuals. For instance, Participant A had primarily positive contemporaneous connections (one negative contemporaneous connection; no lagged connections beyond the 8 auto-regressives; **Fig. 1A**). Most were between the SN and CEN and between the DMN and CEN, as well as one connection between the DMN and SN. In contrast, Participant B had positive and negative contemporaneous connections, as well as one positive lagged connection (**Fig. 1B**). There were several (primarily negative) connections between the SN and CEN and one positive connection between the DMN and CEN, but no connections between the DMN and SN. Participants C and D had relatively sparse maps (i.e., a few connections were statistically meaningful to their networks) and appear structurally similar, revealing how homogeneity (if it exists) can be captured in these person-specific networks. Participants E and F have networks that required the inclusion of second- and third-order lags, respectively, based on *a posteriori* validation (i.e., activation in a region two or three functional volumes prior predicted current activation). Qualitatively, these six networks exemplify significant heterogeneity; in fact, the same connection was not present across all six networks. However, the networks also demonstrate some homogeneity with respect to specific connections (e.g., four participants had a positive connection between the right insula and ACC) and patterns of connectivity (e.g., there were more connections between the SN and CEN than between the DMN and SN).

7. Associations among resting-state networks and psychopathic traits

Total psychopathy. Total psychopathy was associated with increased positive network density (i.e., more positive connections) between the DMN and CEN ($B = 0.28$, $p = .003$) (**Fig. 2**; **Table S4**). Additionally, total psychopathy was associated with increased positive node centrality ($B = 0.21$, $p = .02$) and decreased negative node centrality ($B = -0.23$, $p = .02$) of the PCC (within the DMN); however, these associations did not withstand the Bonferroni correction (**Table S6**). There were no other significant associations between total psychopathic traits and network features. Full results are included in the **Supplemental Materials**.

Interpersonal, affective, lifestyle, and antisocial facets. Although all four facets of psychopathy were significantly associated with increased positive network density between the DMN and CEN in zero-order correlations (interpersonal: $r = 0.18$, $p = .049$; affective: $r = 0.29$, $p = .001$; lifestyle: $r = 0.23$, $p = .012$; antisocial: $r = 0.24$, $p = .008$), when accounting for their overlap in the regression model, only the association with affective traits was significant ($B = 0.28$, $p = .049$; **Table S9**) (**Fig. 2**). In fact, networks in **Fig. 1** demonstrate these differences, as Participant A was high in affective traits and had several positive connections between the DMN and CEN, whereas Participant B was high in lifestyle traits and only had one such connection.

Additionally, in the zero-order correlations, the affective facet was associated with increased positive PCC centrality ($r = 0.22$, $p = .013$),

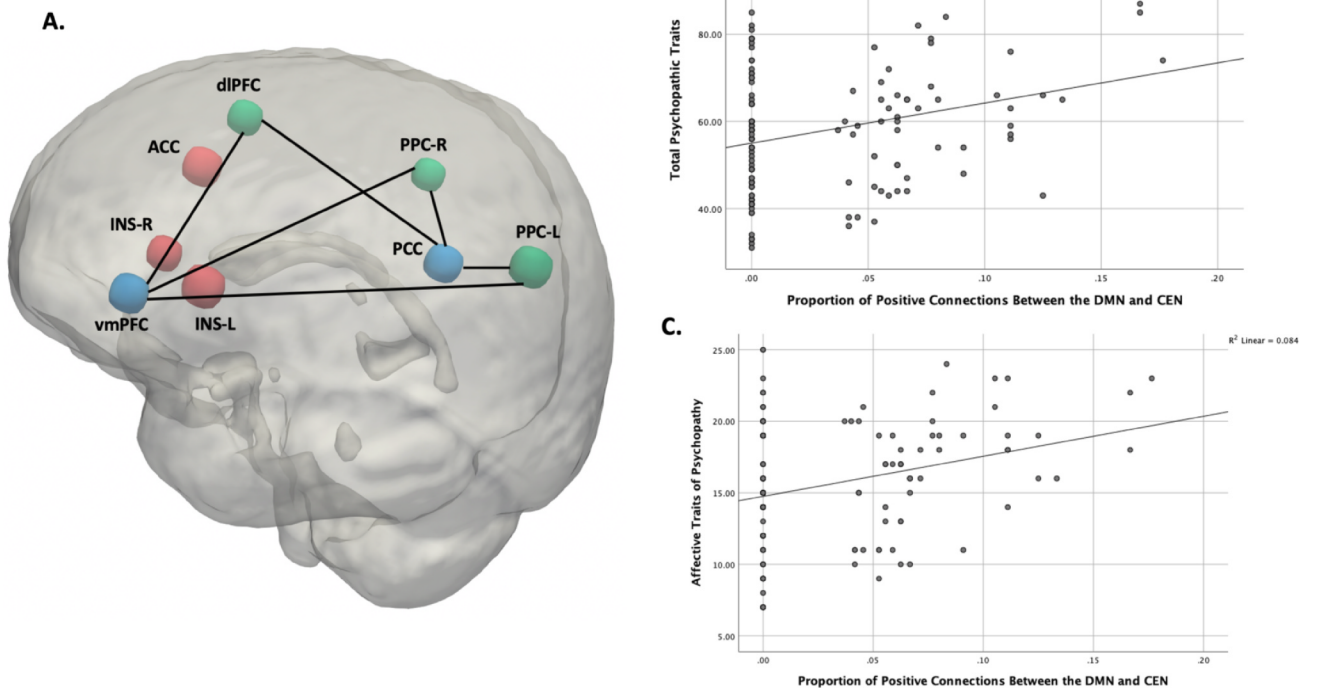


Fig. 2. Psychopathic traits were associated with increased positive density between the default mode network and central executive network across all participants. 2A.) Schematic depiction of possible connections between the default mode network and central executive network regions of interest. Regions in red are within the salience network. Regions in blue are within the default mode network. Regions in green are within the central executive network. 2B.) Scatter plot represents bivariate correlations between *total psychopathic traits* and positive density between the DMN and CEN for each participant. 2C.) Scatter plot represents bivariate correlations between *affective traits* of psychopathy and positive density between the DMN and CEN for each participant. DMN = default mode network. CEN = central executive network. dIPFC = dorsolateral prefrontal cortex; vmPFC = ventromedial prefrontal cortex; PPC-R = right posterior parietal cortex; PPC-L = left posterior parietal cortex; INS-R = right insula; INS-L = left insula; PCC = posterior cingulate cortex; ACC = anterior cingulate cortex. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

and the lifestyle facet was associated with reduced negative PCC centrality ($r = -0.21$, $p = .022$). However, none of the four facets were associated with either positive or negative PCC density in the regression model (accounting for overlap among facets; Table S10). Consistent with a discovery science approach, these findings should encourage future hypothesis-drive work.

8. Discussion

Core to major etiologic theories of psychopathy is the notion that disconnection between the DMN, SN, and CEN underlie emotion and attention deficits. Here we find that men from a low-income, ethnically diverse community sample had significant heterogeneity in resting state connectivity networks using a sparse, person-specific network approach. Yet, there was consistency in connectivity patterns, such that psychopathy was associated with increased positive density in connections between the DMN and CEN, somewhat fitting with the II theory. Exploratory analyses indicated that this association may be driven by affective features of psychopathy. There was also suggestion that psychopathy was associated with increased positive and decreased negative node density of the PCC, a node of the DMN.

Neural networks were person specific. In fact, there were *no* connections among ROIs that were common across participants. This may seem unsurprising because it is consistent with previous research that has applied GIMME to samples marked by significant heterogeneity (e.g., mixed gender sample with varying levels of psychiatric comorbidities; Dotterer et al., 2019). However, it emphasizes the dangers of relying on averaging approaches (i.e., relying on combining neural metrics across individuals) that dominate the extant literature (i.e., they

mask important individual differences in neural mechanisms). Moreover, as demonstrated by the illustrative networks in Fig. 1, all participant networks contained numerous contemporaneous *and* lagged connections at only the individual level, as there were no estimated group-level connections. Additionally, *a posteriori* validation revealed that a number of participants' network models required higher order lagged connections. These nuanced network features would not have been captured using traditional network approaches that only model contemporaneous connections at the group- or individual-level or that fail to conduct *a posteriori* validation. Thus, the network features utilized in the current study are likely robust, as they were derived from accurate, person-specific neural networks (supported by large-scale simulation studies of GIMME; Gates and Molenaar, 2012).

Findings were partially consistent with II theory. Psychopathic traits were only associated with positive DMN-CEN connectivity, but not connectivity within or between the DMN and SN. Further, it is unclear if positive connectivity is posited by the II theory, which merely suggests that there will be differences in connectivity among the three networks. Findings with our network-focused approach significantly extend past work that used seed-based connectivity approaches, in which psychopathic traits were associated with positive connectivity between *regions* within the CEN and DMN (Espinoza et al., 2018), by showing that psychopathy is linked to the organization and functioning of the DMN and CEN *broadly*. Importantly, the DMN and CEN are typically anticorrelated; the CEN is activated during effortful cognitive tasks, whereas the DMN is activated at rest and during self-referential thinking (Buckner et al., 2008). As such, increased communication (and less segregation) between the DMN and CEN reflected in positive DMN-CEN density may interfere with higher-order cognitive processes that

involve both networks, such as decision-making and theory of mind, which appear to be impaired in individuals with psychopathic traits (Hamilton et al., 2015). Moreover, hyperconnectivity between the DMN and other networks, including the CEN, has been observed in individuals with other disorders marked by social cognitive deficits (autism spectrum disorder; Ecker et al., 2015; e.g., schizophrenia; Hu et al., 2017). Thus, further work should examine DMN-CEN connectivity during tasks that require the integration of contextual information during decision-making or goal-directed behavior (e.g., Larson et al., 2013). Future studies could therefore determine whether increased positive DMN-CEN connectivity reflects hyper-focus on goal attainment and internal cues and inflexibility in behavior at the expense of attending to important environmental cues as proposed by II theory.

This study is also novel in showing that the association between psychopathy and DMN-CEN connectivity may be driven by affective features of psychopathy, when accounting for their overlap with the other facets (i.e., interpersonal, lifestyle, antisocial). In contrast to the lifestyle and antisocial features, affective and interpersonal features have been associated with unimpaired or even heightened basic attention abilities (i.e., better attentional control, better response inhibition, and increased error monitoring), but deficits in flexibly using contextual information to modulate attention (Baskin-Sommers et al., 2012; Hoppenbrouwers et al., 2015; Sadeh and Verona, 2008; Veit et al., 2013). As such, individuals who predominantly display affective features may uniquely benefit from intervention targeted towards attention to context (Baskin-Sommers et al., 2015). Although some studies using traditional connectivity methods have similarly identified unique associations between affective features and DMN-CEN connectivity (Espinoza et al., 2018), this pattern has not been consistently replicated (Contreras-Rodríguez et al., 2015; Philippi et al., 2015; Pujol et al., 2012), potentially because those studies varied in sample characteristics (i.e., community versus clinical, gender) and failed to account for person-specific heterogeneity. Notably, this finding did not withstand Bonferroni correction and thus requires replication in future studies. Taken together, further research is needed to clarify the extent to which psychopathic facets are characterized by unique patterns of resting-state connectivity.

Heightened psychopathic traits were also associated with increased positive and decreased negative PCC node centrality, although these associations may not be robust (i.e., did not withstand Bonferroni correction). The PCC, a key node of the DMN, typically deactivates on cognitive tasks but activates during self-referential processing, future thinking, and mentalizing (Brewer et al., 2013; Leech and Sharp, 2014; Pearson et al., 2011). Further, the PCC appears to be involved in attention modulation, with patterns of activation differing for internally (increased activation) versus externally (decreased activation) directed attention (Brewer et al., 2013; Leech and Sharp, 2014; Pearson et al., 2011). More positive connections and fewer negative connections involving the PCC suggest that, in individuals with psychopathic traits, PCC activation is more activated and less inhibited by regions in other networks (consistent with other work; Contreras-Rodríguez et al., 2015; Motzkin et al., 2011; Philippi et al., 2015; Pujol et al., 2012), potentially reflecting higher propensity for self-referential thoughts and internally-directed attention (i.e., heightened “self-focus”). However, further research with larger samples is needed to replicate this likely small association.

Psychopathic traits were not associated with resting-state connectivity within or between the SN and DMN, in contrast to II theory. Previous studies using traditional methods (Philippi et al., 2015) and network modeling (Lindner et al., 2018; Tillem et al., 2019) have also not consistently found these associations. Thus, emotion and attention impairments observed in psychopathy may not be driven by abnormal communication among regions within the SN or DMN themselves, but instead reflect an imbalance with other networks, such as the CEN. Importantly, the focus in this study was on accurate individualized resting-state networks, whereas II theory is based on findings derived

using traditional averaging approaches, which are known to create spurious results (Molenaar, 2004). Thus, these null findings may partially undermine the hypotheses of II theory. It is also possible that psychopathic traits are associated with network features involving nodes other than those included in the current study (Espinoza et al., 2018; Philippi et al., 2015), which were identified *a priori* based on previous work (Chiong et al., 2013; Goulden et al., 2014; Sridharan et al., 2008). For instance, psychopathic traits may be associated with connectivity among other regions in these networks, particularly within the DMN (e.g., dorsal medial prefrontal cortex, temporoparietal junction), which includes subsystems that appear to underlie distinct social cognitive processes (Andrews-Hanna et al., 2010; Li, Mai, and Liu, 2014). Thus, future research would benefit from the inclusion of additional nodes from the SN, DMN, and CEN to better understand connectivity within psychopathy at a systems-level.

9. Strengths and limitations

The current study had several strengths, including a low-income, racially diverse community sample at heightened risk for antisocial behavior, and a novel mapping approach that has been demonstrated to accurately model neural networks by capturing both homogeneity (if it exists) and heterogeneity (Gates and Molenaar, 2012). However, there are limitations. The sample size was reduced because of data loss, which diminished the power of the analyses. Additionally, results may not be generalizable to populations characterized by extreme levels of psychopathic traits, antisocial behavior, or other comorbid psychopathology, including offender or clinical populations. Moreover, as resting-state networks change across development, the current results cannot be generalized to younger (e.g., children) or older populations. Additionally, participants reported on their own psychopathic traits, potentially leading to biased responses, although research has not been able to substantiate associations between psychopathy and response style (Ray et al., 2013). Finally, caution should be used in interpreting negative connections. Previous resting-state studies of psychopathy focused on positive connections owing to debate surrounding negative connections (Lindner et al., 2018; Rubinov and Sporns, 2010). However, GIMME only models connections that are statistically meaningful in a network. Thus, negative connections are unlikely to be a statistical property of time-series data, and instead likely reflect inhibition.

10. Conclusions

In a low-income, racially diverse community sample of young men with a wide range of psychopathic traits, we found significant heterogeneity in neural network connectivity; in fact, there were *no connections* common across all participants. This finding emphasizes the limitations of traditional averaging approaches in understanding neural mechanisms underlying psychopathy. Although individuals had unique neural networks, there were key network features across the sample that were associated with psychopathic traits. Psychopathic traits were associated with patterns in person-specific networks, such as connectivity between the DMN and CEN and the integration of the PCC in the networks. Exploratory analyses suggest that affective features in particular may be characterized by DMN-CEN hyperconnectivity. Taken together, our findings demonstrate how person-specific approaches can be used to capture variability in biopsychosocial profiles, including neural mechanisms, that give rise to similar behaviors, which could ultimately inform individualized treatment efforts (Baskin-Sommers et al., 2015; Insel, 2014).

Funding

The research was supported by Grant R01 MH05090 from the National Institutes of Health to D.S.S., Grant R01 DA02622 to D.S.S. and E.E.F. H.L.D. was supported by a National Science Foundation

Graduate Research Fellowship. A.M.B. was supported by an Early Career Research Fellowship from the Jacobs Foundation.

CRedit authorship contribution statement

Hailey L. Dotterer: Conceptualization, Methodology, Software, Formal analysis, Data curation, Writing - original draft, Writing - review & editing, Visualization, Funding acquisition. **Luke W. Hyde:** Conceptualization, Investigation, Resources, Writing - review & editing, Supervision, Project administration. **Daniel S. Shaw:** Investigation, Data curation, Resources, Writing - review & editing, Project administration, Funding acquisition. **Emma L. Rodgers:** Software, Writing - review & editing, Visualization. **Erika E. Forbes:** Investigation, Data curation, Resources, Writing - review & editing, Project administration, Funding acquisition. **Adriene M. Beltz:** Software, Data curation, Resources, Writing - review & editing, Supervision, Funding acquisition.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.nicl.2020.102402>.

References

- Achenbach, T.M., 1991. Integrative Guide for the 1991 CBCL/4-18, YSR, and TRF Profiles. Department of Psychiatry, University of Vermont.
- Andrews-Hanna, J.R., Reidler, J.S., Sepulcre, J., Poulin, R., Buckner, R.L., 2010. Functional-anatomic fractionation of the brain's default network. *Neuron* 65 (4), 550–562.
- Baskin-Sommers, A.R., Curtin, J.J., Li, W., Newman, J.P., 2012. Psychopathy-related differences in selective attention are captured by an early event-related potential. *Personal. Disord. Theory Res. Treat.* 3 (4), 370.
- Baskin-Sommers, A.R., Curtin, J.J., Newman, J.P., 2015. Altering the cognitive-affective dysfunctions of psychopathic and externalizing offender subtypes with cognitive remediation. *Clin. Psychol. Sci.* 3 (1), 45–57.
- Baskin-Sommers, A.R., Newman, J.P., Sathasivam, N., Curtin, J.J., 2011. Evaluating the generalizability of a fear deficit in psychopathic African American offenders. *J. Abnorm. Psychol.* 120 (1), 71–78.
- Beck, J.E., Shaw, D.S., 2005. The influence of perinatal complications and environmental adversity on boys' antisocial behavior. *J. Child Psychol. Psychiatry* 46 (1), 35–46.
- Beltz, A.M., Gates, K.M., 2017. Network Mapping with GIMME. *Multivar. Behav. Res.* 52 (6), 789–804.
- Beltz, A.M., Molenaar, P.C., 2015. A posteriori model validation for the temporal order of directed functional connectivity maps. *Front. Neurosci.* 9.
- Beltz, A.M., Moser, J.S., Zhu, D.C., Burt, S.A., Klump, K.L., 2018. Using person-specific neural networks to characterize heterogeneity in eating disorders: Illustrative links between emotional eating and ovarian hormones. *Int. J. Eat. Disord.* 51 (7), 730–740.
- Biswal, B.B., Mennes, M., Zuo, X.-N., Gohel, S., Kelly, C., Smith, S.M., Colcombe, S., 2010. Toward discovery science of human brain function. *Proc. Natl. Acad. Sci.* 107 (10), 4734–4739.
- Blair, R.J.R., 2010. Neuroimaging of psychopathy and antisocial behavior: a targeted review. *Curr. Psychiatry Rep.* 12 (1), 76–82.
- Blair, R. J. R. (2017). Emotion-based learning systems and the development of morality. *Cognition*, epub ahead of print.
- Brewer, J., Garrison, K., Whitfield-Gabrieli, S., 2013. What about the "self" is processed in the posterior cingulate cortex? *Front. Hum. Neurosci.* 7, 647.
- Brown, T.A., 2014. *Confirmatory Factor Analysis For Applied Research*. Guilford Publications.
- Buckner, R.L., Andrews-Hanna, J.R., Schacter, D.L., 2008. The brain's default network. *Ann. N. Y. Acad. Sci.* 1124 (1), 1–38.
- Cahalan, D., Cisin, L.H., Crossley, H.M., 1969. American drinking practices: A national study of drinking behavior and attitudes. *Monogr. Rutgers Center Alcohol Stud.*
- Carré, J.M., Hyde, L.W., Neumann, C.S., Viding, E., Hariri, A.R., 2013. The neural signatures of distinct psychopathic traits. *Soc. Neurosci.* 8 (2), 122–135.
- Chai, X.J., Castañón, A.N., Öngür, D., Whitfield-Gabrieli, S., 2012. Anticorrelations in resting state networks without global signal regression. *NeuroImage* 59 (2), 1420–1428.
- Chiong, W., Wilson, S.M., D'Esposito, M., Kayser, A.S., Grossman, S.N., Poorzand, P., Rankin, K.P., 2013. The salience network causally influences default mode network activity during moral reasoning. *Brain* 1–13.
- Cohen, J.R., D'Esposito, M., 2016. The segregation and integration of distinct brain networks and their relationship to cognition. *J. Neurosci.* 36 (48), 12083–12094.
- Contreras-Rodríguez, O., Pujol, J., Batalla, I., Harrison, B.J., Soriano-Mas, C., Deus, J., Hernández-Ribas, R., 2015. Functional connectivity bias in the prefrontal cortex of psychopaths. *Biol. Psychiatry* 78 (9), 647–655.
- De Vico Fallani, F., Richiardi, J., Chavez, M., Achard, S., 2014. Graph analysis of functional brain networks: practical issues in translational neuroscience. *Phil. Trans. R. Soc. B* 369 (1653), 20130521.
- Deming, P., Philippi, C.L., Wolf, R.C., Dargis, M., Kiehl, K.A., Koenigs, M., 2018. Psychopathic traits linked to alterations in neural activity during personality judgments of self and others. *NeuroImage: Clinical* 18, 575–581.
- Dotterer, H.L., Beltz, A.M., Foster, K.T., Simms, L.J., Wright, A.G., 2019. Personalized models of personality disorders: Using a temporal network method to understand symptomatology and daily functioning in a clinical sample. *Psychol. Med.* 1–9.
- Dotterer, H.L., Waller, R., Neumann, C.S., Shaw, D.S., Forbes, E.E., Hariri, A.R., Hyde, L.W., 2016. Examining the factor structure of the Self-Report of Psychopathy Short-Form across four young adult samples. *Assessment* 1073191116640355.
- Ecker, C., Bookheimer, S.Y., Murphy, D.G., 2015. Neuroimaging in autism spectrum disorder: brain structure and function across the lifespan. *Lancet Neurol.* 14 (11), 1121–1134.
- Efferson, L.M., Glenn, A.L., 2018. Examining gender differences in the correlates of psychopathy: A systematic review of emotional, cognitive, and morality-related constructs. *Agg. Violent Behav.* 41, 48–61.
- Espinoza, F.A., Vergara, V.M., Reyes, D., Anderson, N.E., Harenski, C.L., Decety, J., Miller, R.L., 2018. Aberrant functional network connectivity in psychopathy from a large (N = 985) forensic sample. *Hum. Brain Mapp.* 39 (6), 2624–2634.
- Ewbank, M.P., Passamonti, L., Hagan, C.C., Goodyer, I.M., Calder, A.J., Fairchild, G., 2018. Psychopathic traits influence amygdala–anterior cingulate cortex connectivity during facial emotion processing. *Soc. Cogn. Affect. Neurosci.* 13 (5), 525–534.
- Fornito, A., 2016. Graph theoretic analysis of human brain networks. In: *fMRI Techniques and Protocols*. Springer, pp. 283–314.
- Fox, M.D., Snyder, A.Z., Vincent, J.L., Corbetta, M., Van Essen, D.C., Raichle, M.E., 2005. The human brain is intrinsically organized into dynamic, anticorrelated functional networks. *PNAS* 102 (27), 9673–9678.
- Friston, K., Josephs, O., Zarahn, E., Holmes, A., Rouquette, S., Poline, J.-B., 2000. To smooth or not to smooth?: Bias and efficiency in fMRI time-series analysis. *NeuroImage* 12 (2), 196–208.
- Gao, Y., Raine, A., 2010. Successful and unsuccessful psychopaths: A neurobiological model. *Behavioral Sciences & the Law* 28 (2), 194–210.
- Gard, A.M., Waller, R., Shaw, D.S., Forbes, E.E., Hariri, A.R., Hyde, L.W., 2017. The long reach of early adversity: Parenting, stress, and neural pathways to antisocial behavior in adulthood. *Biol. Psychiatry Cognitive Neurosci. Neuroimaging* 2 (7), 582–590.
- Gates, K.M., Molenaar, P.C., 2012. Group search algorithm recovers effective connectivity maps for individuals in homogeneous and heterogeneous samples. *NeuroImage* 63 (1), 310–319.
- Gates, K.M., Molenaar, P.C., Hillary, F.G., Ram, N., Rovine, M.J., 2010. Automatic search for fMRI connectivity mapping: an alternative to Granger causality testing using formal equivalences among SEM path modeling, VAR, and unified SEM. *NeuroImage* 50 (3), 1118–1125.
- Gates, K.M., Molenaar, P.C., Hillary, F.G., Slobounov, S., 2011. Extended unified SEM approach for modeling event-related fMRI data. *NeuroImage* 54 (2), 1151–1158.
- Geurts, D.E., Von Borries, K., Volman, I., Bulten, B.H., Cools, R., Verkes, R.-J., 2016. Neural connectivity during reward expectation dissociates psychopathic criminals from non-criminal individuals with high impulsive/antisocial psychopathic traits. *Social Cogn. Affect. Neurosci.* 11 (8), 1326–1334.
- Gordts, S., Uzieblo, K., Neumann, C., Van den Bussche, E., Rossi, G., 2017. Validity of the self-report psychopathy scales (SRP-III full and short versions) in a community sample. *Assessment* 24 (3), 308–325.
- Goulden, N., Khusnulina, A., Davis, N.J., Bracewell, R.M., Bokde, A.L., McNulty, J.P., Mullins, P.G., 2014. The salience network is responsible for switching between the default mode network and the central executive network: replication from DCM. *NeuroImage* 99, 180–190.
- Greicius, M., 2008. Resting-state functional connectivity in neuropsychiatric disorders. *Curr. Opin. Neurol.* 21 (4), 424–430.
- Hamilton, R.K., Hiatt Racer, K., Newman, J.P., 2015. Impaired integration in psychopathy: A unified theory of psychopathic dysfunction. *Psychol. Rev.* 122 (4), 770–791.
- Hoppenbrouwers, S.S., Van der Stigchel, S., Slotboom, J., Dalmaijer, E.S., Theeuwes, J., 2015. Disentangling attentional deficits in psychopathy using visual search: Failures in the use of contextual information. *Personality Individ. Differ.* 86, 132–138.
- Hu, M.-L., Zong, X.-F., Mann, J.J., Zheng, J.-J., Liao, Y.-H., Li, Z.-C., Tang, J.-S., 2017. A review of the functional and anatomical default mode network in schizophrenia. *Neurosci. Bull.* 33 (1), 73–84.
- Hyde, L.W., Shaw, D.S., Murray, L., Gard, A., Hariri, A.R., Forbes, E.E., 2016. Dissecting the role of amygdala reactivity in antisocial behavior in a sample of young, low-income, urban men. *Clin. Psychol. Sci.* 4 (3), 527–544.
- Insel, T.R., 2014. The NIMH research domain criteria (RDoC) project: precision medicine for psychiatry. *Am. J. Psychiatry* 171 (4), 395–397.
- Jenkinson, M., Bannister, P., Brady, M., Smith, S., 2002. Improved optimization for the robust and accurate linear registration and motion correction of brain images. *NeuroImage* 17 (2), 825–841.
- Jenkinson, M., Beckmann, C.F., Behrens, T.E., Woolrich, M.W., Smith, S.M., 2012. FSL. *NeuroImage* 62 (2), 782–790.
- Johanson, M., Vaurio, O., Tiihonen, J., Lähtenvuo, M., 2020. A systematic literature review of neuroimaging of psychopathic traits. *Front. Psychiatry* 10, 1027.
- Kaiser, M., 2011. A tutorial in connectome analysis: topological and spatial features of

- brain networks. *NeuroImage* 57 (3), 892–907.
- Kelly, A.C., Uddin, L.Q., Biswal, B.B., Castellanos, F.X., Milham, M.P., 2008. Competition between functional brain networks mediates behavioral variability. *NeuroImage* 39 (1), 527–537.
- Kiehl, K.A., 2006. A cognitive neuroscience perspective on psychopathy: evidence for paralimbic system dysfunction. *Psychiatry Res.* 142 (2), 107–128.
- Kiehl, K.A., Hoffman, M.B., 2011. The criminal psychopath: History, neuroscience, treatment, and economics. *Jurimetrics* 51, 355.
- Korponay, C., Pujara, M., Deming, P., Philippi, C., Decety, J., Kosson, D.S., Koenigs, M., 2017. Impulsive-antisocial dimension of psychopathy linked to enlargement and abnormal functional connectivity of the striatum. *Biol. Psychiatry Cognitive Neurosci. Neuroimaging* 2 (2), 149–157.
- Lane, S.T., Gates, K.M., 2017. Automated selection of robust individual-level structural equation models for time series data. *Struct. Eq. Model. Multidisciplin. J.* 24 (5), 768–782.
- Larson, C.L., Baskin-Sommers, A.R., Stout, D.M., Balderston, N.L., Curtin, J.J., Schultz, D.H., Newman, J.P., 2013. The interplay of attention and emotion: top-down attention modulates amygdala activation in psychopathy. *Cogn. Affect. Behav. Neurosci.* 13 (4), 757–770.
- Latzman, R.D., Patrick, C.J., Lilienfeld, S.O., 2019. Heterogeneity matters: implications for Poeschl et al.'s (2019) meta-analysis and future neuroimaging research on psychopathy. *Mol. Psychiatry* 1.
- Leech, R., Sharp, D.J., 2014. The role of the posterior cingulate cortex in cognition and disease. *Brain* 137 (1), 12–32.
- Leutgeb, V., Wabnegger, A., Leitner, M., Zussner, T., Scharnüller, W., Klug, D., Schienle, A., 2016. Altered cerebellar-amygdala connectivity in violent offenders: A resting-state fMRI study. *Neurosci. Lett.* 610, 160–164.
- Li, W., Mai, X., Liu, C., 2014. The default mode network and social understanding of others: what do brain connectivity studies tell us. *Front. Hum. Neurosci.* 8, 74.
- Lilienfeld, S.O., 2018. The multidimensional nature of psychopathy: Five recommendations for research. *J. Psychopathol. Behav. Assess.* 40 (1), 79–85.
- Lindner, P., Flodin, P., Budhiraja, M., Savic, I., Jokinen, J., Tiihonen, J., Hodgins, S., 2018. Associations of psychopathic traits with local and global brain network topology in young adult women. *Biol. Psychiatry Cogn. Neurosci. Neuroimaging* 3 (12), 1003–1012.
- Lu, F.-M., Zhou, J.-S., Zhang, J., Wang, X.-P., Yuan, Z., 2017. Disrupted small-world brain network topology in pure conduct disorder. *Oncotarget* 8 (39), 65506–65524.
- Mahmut, M.K., Menicatas, C., Stevenson, R.J., Homewood, J., 2011. Validating the factor structure of the Self-Report Psychopathy scale in a community sample. *Psychol. Assess.* 23 (3), 670.
- Menon, V., 2011. Large-scale brain networks and psychopathology: a unifying triple network model. *Trends Cogn. Sci.* 15 (10), 483–506.
- Menon, V., Uddin, L.Q., 2010. Saliency, switching, attention and control: a network model of insula function. *Brain Struct. Funct.* 214 (5–6), 655–667.
- Molenaar, P.C., 2004. A manifesto on psychology as idiographic science: Bringing the person back into scientific psychology, this time forever. *Measurement* 2 (4), 201–218.
- Motzkin, J.C., Newman, J.P., Kiehl, K.A., Koenigs, M., 2011. Reduced prefrontal connectivity in psychopathy. *J. Neurosci.* 31 (48), 17348–17357.
- Muthén, L.K., Muthén, B.O., 2020. *Mplus 8.4*. Muthén & Muthén, Los Angeles, CA.
- Neal, T.M.S., Sellbom, M., 2012. Examining the factor structure of the Hare Self-Report Psychopathy Scale. *J. Pers. Assess.* 94 (3), 244–253. <https://doi.org/10.1080/00223891.2011.648294>.
- Neumann, C.S., Hare, R.D., 2008. Psychopathic traits in a large community sample: Links to violence, alcohol use, and intelligence. *J. Consult. Clin. Psychol.* 76 (5), 893–899. <https://doi.org/10.1037/0022-006X.76.5.893>.
- Neumann, C.S., Pardini, D., 2014. Factor structure and construct validity of the Self-Report Psychopathy (SRP) Scale and the Youth Psychopathic Traits Inventory (YPI) in young men. *J. Pers. Disord.* 28 (3), 419–433.
- Neumann, C.S., Schmitt, D.S., Carter, R., Embley, I., Hare, R.D., 2012. Psychopathic traits in females and males across the globe. *Behav. Sci. Law* 30 (5), 557–574. <https://doi.org/10.1002/bsl.2038>.
- Newman, J.P., Baskin-Sommers, A.R., 2012. Early selective attention abnormalities in psychopathy: Implications for self-regulation. In: Posner, M.I., Posner, M.I. (Eds.), *Cognitive Neuroscience of Attention*, 2nd ed. Guilford Press, New York, NY, US, pp. 421–440.
- Paulhus, D., Neumann, C.S., Hare, R.D., 2015. *Self-Report Psychopathy Scale (SRP-4)*. Multi-Health Systems, Toronto, ON.
- Pearson, J.M., Heilbronner, S.R., Barack, D.L., Hayden, B.Y., Platt, M.L., 2011. Posterior cingulate cortex: adapting behavior to a changing world. *Trends Cogn. Sci.* 15 (4), 143–151.
- Philippi, C.L., Pujara, M.S., Motzkin, J.C., Newman, J., Kiehl, K.A., Koenigs, M., 2015. Altered resting-state functional connectivity in cortical networks in psychopathy. *J. Neurosci.* 35 (15), 6068–6078.
- Price, R.B., Lane, S., Gates, K., Kraynak, T.E., Horner, M.S., Thase, M.E., Siegle, G.J., 2017. Parsing heterogeneity in the brain connectivity of depressed and healthy adults during positive mood. *Biol. Psychiatry* 81 (4), 347–357.
- Pruim, R.H., Mennes, M., Buitelaar, J.K., Beckmann, C.F., 2015a. Evaluation of ICA-AROMA and alternative strategies for motion artifact removal in resting state fMRI. *NeuroImage* 112, 278–287.
- Pruim, R.H., Mennes, M., van Rooij, D., Llera, A., Buitelaar, J.K., Beckmann, C.F., 2015b. ICA-AROMA: A robust ICA-based strategy for removing motion artifacts from fMRI data. *NeuroImage* 112, 267–277.
- Pujol, J., Batalla, I., Contreras-Rodríguez, O., Harrison, B.J., Pera, V., Hernández-Ribas, R., Deus, J., 2012. Breakdown in the brain network subserving moral judgment in criminal psychopathy. *Soc. Cogn. Affect. Neurosci.* 7 (8), 917–923.
- Ray, J.V., Hall, J.R., Rivera-Hudson, N., Poythress, N.G., Lilienfeld, S.O., Morano, M., 2013. The relation between self-reported psychopathic traits and distorted response styles: A meta-analytic review. *Personal. Disord. Theory Res. Treat.* 4 (1), 1–14. <https://doi.org/10.1037/a0026482>.
- Reidy, D.E., Kearns, M.C., DeGue, S., 2013. Reducing psychopathic violence: A review of the treatment literature. *Aggress. Violent Behav.* 18 (5), 527–538.
- Reniers, R.L., Corcoran, R., Völlm, B.A., Mashru, A., Howard, R., Liddle, P.F., 2012. Moral decision-making, ToM, empathy and the default mode network. *Biol. Psychol.* 90 (3), 202–210.
- Rubinov, M., Sporns, O., 2010. Complex network measures of brain connectivity: uses and interpretations. *NeuroImage* 52 (3), 1059–1069.
- Sadeh, N., Verona, E., 2008. Psychopathic personality traits associated with abnormal selective attention and impaired cognitive control. *Neuropsychology* 22 (5), 669.
- Seara-Cardoso, A., Neumann, C., Roiser, J., McCrory, E., Viding, E., 2012. Investigating associations between empathy, morality and psychopathic personality traits in the general population. *Personality Individ. Differ.* 52 (1), 67–71. <https://doi.org/10.1016/j.paid.2011.08.029>.
- Seara-Cardoso, A., Queirós, A., Fernandes, E., Coutinho, J., Neumann, C., 2019. Psychometric properties and construct validity of the short version of the Self-Report Psychopathy Scale in a Southern European sample. *J. Pers. Assess.* 1–12.
- Seelye, W.W., 2019. The salience network: a neural system for perceiving and responding to homeostatic demands. *J. Neurosci.* 39 (50), 9878–9882.
- Shaw, D.S., Gilliom, M., Ingoldsby, E.M., Nagin, D.S., 2003. Trajectories leading to school-age conduct problems. *Dev. Psychol.* 39 (2), 189–200.
- Shaw, D.S., Hyde, L.W., Brennan, L.M., 2012. Early predictors of boys' antisocial trajectories. *Dev. Psychopathol.* 24 (3), 871–888.
- Skeem, J.L., Polaschek, D.L.L., Patrick, C.J., Lilienfeld, S.O., 2011. Psychopathic personality: Bridging the gap between scientific evidence and public policy. *Psychol. Sci. Public Interest* 12 (3), 95–162. <https://doi.org/10.1177/1529100611426706>.
- Smith, S.M., Miller, K.L., Salimi-Khorshidi, G., Webster, M., Beckmann, C.F., Nichols, T.E., Woolrich, M.W., 2011. Network modelling methods for FMRI. *NeuroImage* 54 (2), 875–891.
- Sridharan, D., Levitin, D.J., Menon, V., 2008. A critical role for the right fronto-insular cortex in switching between central-executive and default-mode networks. *Proc. Natl. Acad. Sci.* 105 (34), 12569–12574.
- Tang, Y., Jiang, W., Liao, J., Wang, W., Luo, A., 2013. Identifying individuals with antisocial personality disorder using resting-state FMRI. *PLoS One* 8 (4), e60652. <https://doi.org/10.1371/journal.pone.0060652>.
- Tillem, S., Harenski, K., Decety, J., Kosson, D., Kiehl, K.A., Baskin-Sommers, A., 2019. Psychopathy is associated with shifts in the organization of neural networks in a large incarcerated male sample. *NeuroImage: Clin.* 24, 102083.
- Tillem, S., van Dongen, J., Brazil, I.A., Baskin-Sommers, A., 2018. Psychopathic traits are differentially associated with efficiency of neural communication. *Psychophysiology* 55 (9), e13194.
- Veit, R., Konicar, L., Klinzing, J.G., Barth, B., Yilmaz, O., Birbaumer, N., 2013. Deficient fear conditioning in psychopathy as a function of interpersonal and affective disturbances. *Front. Hum. Neurosci.* 7, 706. <https://doi.org/10.3389/fnhum.2013.00706>.
- Vermeij, A., Kempes, M.M., Cima, M.J., Mars, R.B., Brazil, I.A., 2018. Affective traits of psychopathy are linked to white-matter abnormalities in impulsive male offenders. *Neuropsychology*.
- Waller, R., Gard, A.M., Shaw, D.S., Forbes, E.E., Neumann, C.S., Hyde, L.W., 2018. Weakened functional connectivity between the amygdala and the ventromedial prefrontal cortex is longitudinally related to psychopathic traits in low-income males during early adulthood. *Clin. Psychol. Sci.* 2167702618810231.
- Wolf, R.C., Pujara, M.S., Motzkin, J.C., Newman, J.P., Kiehl, K.A., Decety, J., Koenigs, M., 2015. Interpersonal traits of psychopathy linked to reduced integrity of the uncinate fasciculus. *Hum. Brain Mapp.* 36 (10), 4202–4209.
- Woolrich, M.W., Ripley, B.D., Brady, M., Smith, S.M., 2001. Temporal autocorrelation in univariate linear modeling of FMRI data. *NeuroImage* 14 (6), 1370–1386.
- Yang, Y., Raine, A., Joshi, A.A., Joshi, S., Chang, Y.-T., Schug, R.A., Narr, K.L., 2012. Frontal information flow and connectivity in psychopathy. *Brit. J. Psychiatry* 201 (5), 408–409.
- Yoder, K.J., Porges, E.C., Decety, J., 2014. Amygdala subnuclei connectivity in response to violence reveals unique influences of individual differences in psychopathic traits in a nonforensic sample. *Hum. Brain Mapp.*
- Yuan, K.-H., Bentler, P.M., 2000. 5. Three likelihood-based methods for mean and covariance structure analysis with nonnormal missing data. *Sociol. Methodol.* 30 (1), 165–200.