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Impaired salience network switching in psychopathy

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Abstract

Growing evidence suggests that psychopathy is related to altered connectivity within and between three large-scale brain networks that support core cognitive functions, including allocation of attention. In healthy individuals, default mode network (DMN) is involved in internally-focused attention and cognition such as self-reference. Frontoparietal network (FPN) is anticorrelated with DMN and is involved in externally-focused attention to cognitively demanding tasks. A third network, salience network (SN), is involved in detecting salient cues and, crucially, appears to play a role in switching between the two anticorrelated networks, DMN and FPN, to efficiently allocate attentional resources. Psychopathy has been related to reduced anticorrelation between DMN and FPN, suggesting SN's role in switching between these two networks may be diminished in the

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disorder. To test this hypothesis, we used independent component analysis to derive DMN, FPN, and SN activity in resting-state fMRI data in a sample of incarcerated men (N = 148). We entered the activity of the three networks into dynamic causal modeling to test SN's switching role. The previously established switching effect of SN among young, healthy adults was replicated in a group of low psychopathy participants (posterior model probability = 0.38). As predicted, SN's switching role was significantly diminished in high psychopathy participants ($t(145) = 26.39$, $p < .001$). These findings corroborate a novel theory of brain function in psychopathy. Future studies may use this model to test whether disrupted SN switching is related to high psychopathy individuals' abnormal allocation of attention.

Keywords

psychopathy; MRI; neuroimaging; salience network; dynamic causal modeling

1. INTRODUCTION

People with psychopathy are notorious for their deceitful interpersonal style, callousness, impulsivity, and irresponsible lifestyle. Psychopathy is a significant risk factor for violent and non-violent criminal behavior [1–3], is overrepresented in prisons [4], and has been estimated to cost the United States $460 billion per year [5], making psychopathy one of the most costly mental health disorders. Clarifying the neurobiology of psychopathy could lead to applications for diagnosis, treatment, and criminal justice [6,7]. Yet, the neurobiology of the disorder remains poorly understood. In particular, the search for a reliable biomarker has fallen short[1], with neuroimaging studies producing evidence for altered activity in widespread brain regions [8] and inconsistent evidence for altered activity within focal brain regions or in specific contexts [9]. Another branch of research has taken a different approach to identifying core neural mechanisms associated with psychopathy, analyzing interactions among large-scale brain networks, with promising results [10–19].

Three large-scale brain networks in particular appear to serve core cognitive functions [20,21]: the default mode network (DMN), frontoparietal network[2] (FPN), and salience network (SN). Neuroimaging studies have consistently identified these networks both in the presence and absence of experimental tasks (i.e., during tasks and "resting state," respectively). In healthy populations, DMN and FPN are anticorrelated networks that respond in opposing patterns to externally-focused, cognitively demanding tasks. FPN increases activity during such tasks and plays a critical role in the allocation of selective attention [22,23]. In contrast, DMN tends to decrease activity during such tasks and appears to support internally-focused cognition such as self-reference or recollecting prior experiences [24]. SN plays a critical role in detecting novel and emotionally relevant cues and recruiting attentional networks (e.g., FPN) to engage with the salient task/stimuli [25,26]. Crucially, SN appears responsible for switching between the two anticorrelated networks [22,23,27,28]. Two key SN regions, the anterior cingulate cortex and anterior

[1]For a discussion of the methodological factors that may contribute to inconsistencies in the neuroimaging literature on psychopathy, see [8].
[2]Also referred to as "central executive network."

insula, have faster temporal dynamics than regions in DMN and FPN, flexibly change connectivity with other networks over time, and maintain high network centrality over time [29,30]. SN thus appears to be a critical hub for facilitating interactions between other networks.

The switching effect of SN on the two anticorrelated networks may be diminished in psychopathy, according to the Impaired Integration theory [31]. While this hypothesis remains to be tested, extant data corroborate the broader hypothesis that interactions among these three large-scale networks are altered. Psychopathy has been related to more positive functional connectivity (i.e., reduced anticorrelation) between DMN and FPN nodes in resting state [14,15,19], increased connectivity between SN and FPN nodes [13,15], and decreased connectivity between SN and DMN nodes [12,32]. Intriguingly, one study has extended these findings of altered *static* connectivity by observing altered *dynamic* changes in whole-brain connectivity states (i.e., including other large-scale brain networks). Espinoza and colleagues found that individuals with higher interpersonal/affective features of psychopathy (e.g., grandiosity, callousness) made less frequent switches between whole-brain connectivity states and spent more time in a state characterized by weaker functional connectivity overall [16]. In addition to altered functional connectivity, reduced white matter integrity has been observed in pathways between DMN nodes [33] as well as between the three networks [34]. Together, these findings suggest that psychopathy is associated with altered functional and structural connectivity between the three large-scale brain networks, notably increased competition between DMN and FPN, and reduced dynamic switching between connectivity states.

Diminished SN switching could be a neural correlate of cognitive deficits associated with psychopathy. In particular, people with psychopathy display impaired attention [35]. During cognitively demanding tasks, people with psychopathy display deficits in orienting to salient, goal-irrelevant information and recruiting attentional resources [31,36–39]. One prominent theory has posited that people with psychopathy often fail to attend, for example, to another person's emotional state or to the threat of punishment, and thus commit acts that cause others distress or result in punishment [35]. In addition, people with psychopathy display associative learning deficits and have difficulty using past experience to guide future behavior [31]. The Impaired Integration theory hypothesizes [31] that these cognitive deficits result from reduced coordination and flexible switching between a number of large-scale brain networks, including reduced SN switching.

The current study aimed to test the hypothesis that SN's role in switching between states predominated by DMN activity and states predominated by FPN activity is impaired in psychopathy. This is the first study to investigate this hypothesis directly. We analyzed resting-state fMRI data from a sample of incarcerated men and examined SN's switching role using methods that have previously been validated in a study of healthy adults. We hypothesized that 1) low psychopathy participants would show the expected switching effect of SN, and 2) this switching effect of SN would be diminished in high psychopathy participants. Importantly, our analyses of resting-state fMRI data will not allow for conclusions about SN's switching role in attention, or about SN's switching role during cognitively demanding tasks. We conducted additional preliminary analyses testing our

hypotheses within the context of a cognitively demanding task in a smaller sample of incarcerated men (see Supplemental Methods and Results, Tables S2–3, Figures S4–8).

2. MATERIALS AND METHODS

2.1. Participants

Incarcerated men in medium-security correctional facilities in the Midwest were recruited. A total of 351 participants completed resting-state fMRI scans. All participants were included in previous reports [15,16]. Included participants had no history of psychosis, bipolar disorder, PTSD, epilepsy, stroke, or head injury with loss of consciousness >30 min; were not currently using psychotropic medications; demonstrated >4th grade English reading level; had intact audition and vision; and had no MRI contraindications. Initially, analyses included participants 18-55 years old. However, because initial models that included low psychopathy participants older than 30 failed to replicate the SN switching effect that has previously been observed in participants 18-30 years old [22,23], primary analyses for this study were limited to $N = 148$ participants age 18-30 (see sample characteristics in Table 1). All participants provided written informed consent.

2.2. Assessments

Psychopathy was assessed using the Psychopathy Checklist-Revised (PCL-R) [4]. Twenty psychopathic traits were rated on a 0-2 scale based on information obtained during a 90-minute interview and file review. Groups of low (PCL-R ≤ 20; $n = 48$), intermediate (PCL-R > 20 and < 30; $n = 53$), and high psychopathy (PCL-R ≥ 30; $n = 47$) participants were formed according to recommended cut-offs of PCL-R Total scores [4]. IQ was estimated from the Wechsler Adult Intelligence Scale 3rd Ed. [40]. Lifetime substance use disorder diagnoses (for any substance) were determined using the Structured Clinical Interview for the DSM-IV [41]. Participants self-reported their number of lifetime head injuries that resulted in loss of consciousness, loss of memory, or symptoms such as headaches, dizziness, or nausea.

2.3. fMRI Acquisition and Preprocessing

All fMRI images were acquired on prison grounds in the Mind Research Network's Siemens 1.5T Avanto mobile scanner. Resting-state scans were acquired by a 12-element head coil, with the following parameters applied to an EPI gradient-echo pulse sequence: TR = 2000ms, TE = 39ms, flip angle = 90°, FOV = 24 x 24cm, 64 x 64 matrix, 3.4 x 3.4mm in-plane resolution, 4mm slice thickness, 1mm gap, 30 slices. During resting-state scans, participants were asked to lay still, eyes open, focusing on a fixation cross during the five-minute scan period.

All EPI volumes were despiked (ArtDespike in SPM), aligned to the first volume in the time series (Inrialign), registered to MNI EPI template space, and smoothed with 6mm FWHM kernel. The first 8 TRs (16000ms) were removed from each resting-state scan.

2.4. Independent Component Analysis

Independent component analysis (ICA) and non-linear dynamic causal modeling (DCM) methods replicated those implemented in a prior study of SN's modulatory effect on DMN-FPN connectivity [23]. ICA was carried out in the Group ICA of fMRI Toolbox (GIFT, http://www.trendscenter.org/software/gift/) in Matlab. The networks of interest were identified via constrained ICA, a semiblind ICA method that derives components that most closely match a specified anatomical template. For hypotheses about specific networks, this method holds advantages over blind ICA, resolving ambiguity regarding the number of components the model should define. Templates from a network atlas derived from resting-state whole-brain connectivity were used to specify DMN, FPN, and SN (Figure S1) [42]. Time courses were extracted from the ICA components and entered into DCM analyses.

2.5. Dynamic Causal Modeling

Effective connectivity of the three networks was modeled by stochastic, non-linear DCM in Statistical Parametric Mapping 12 (SPM12; http://www.fil.ion.ucl.ac.uk/spm/). Three fully connected models with no extrinsic input were compared to test the robustness of the switching effect of SN on DMN-FPN connectivity (Figure 1). DCM models tested 1) SN's modulatory effect on the bidirectional connections between DMN and FPN (the "SN modulation model"), 2) DMN's modulatory effect on the bidirectional connections between SN and FPN (the "DMN modulation model"), and 3) FPN's modulatory effect on the bidirectional connections between SN and DMN (the "FPN modulation model"). Random-effects Bayesian model selection (RFX-BMS) was performed to identify the model that best fit the data [43], with greater values of expected posterior model probability (i.e., the probability of a model generating the data of a randomly selected subject) and exceedance probability (i.e., the probability, given the group data, that a model is more likely than any other) indicating better model fit. RFX-BMS was performed separately for the low, intermediate, and high psychopathy groups, in line with a prior study comparing DCM model probabilities between two groups, cognitively impaired individuals and healthy individuals [28]. Modulation parameters (e.g., the modulatory effect of SN on DMN-to-FPN connectivity) were derived via Bayesian Model Averaging (BMA), which weights the estimated parameter by the posterior probability of each model [43,44].

2.6. SN Switching and Group Comparisons

To examine the replicability of a switching role of SN, we performed RFX-BMS in the low psychopathy group. We first performed RFX-BMS in the sample of $n = 118$ low psychopathy participants age 18-55 (sample characteristics in Table S1). However, upon failing to replicate the switching role of SN in this sample, we attempted to more closely replicate the methods of prior studies that had reported the SN switching effect in participants 18-30 years old [22,23]. We restricted RFX-BMS analyses to $n = 48$ low psychopathy participants age 18-30. All subsequent analyses included participants age 18-30.

Next, we made group comparisons to test for a reduced switching role of SN in high psychopathy participants. We treated psychopathy as a categorical rather than continuous

variable, as this is an established method for associating psychopathology with effective connectivity as measured by DCM [28,45]. One model with reference-coded variables compared the high psychopathy group (coded 0 in each reference variable) separately to each of the low and intermediate groups (coded 1 or 0) on posterior probabilities for the SN modulation model. A post-hoc *t*-test compared posterior probabilities between the low and intermediate psychopathy groups. Thus, we compared each psychopathy group separately to the other two. Finally, the reference-coded model and post-hoc *t*-test were repeated to examine the relationship between membership in the high psychopathy group and the SN modulation parameters. Tests were initially conducted without additional covariates, then repeated including the covariates of race, substance use disorder, and head injury (dichotomous variables coded white/non-white, present/absent, and present/absent, respectively), as well as age and IQ. One participant missing IQ scores and 18 participants missing head injury data were excluded from the covariate models.

3. RESULTS

The constrained ICA yielded three components as specified (Figure 2). To check that these components represented the hypothesized networks, the percentage of overlap between voxels in the template and significant voxels (uncorrected $p < .001$, $p_{FWE} < .05$, cluster extent threshold = 27 voxels) in the corresponding component was computed. The first component overlapped with the DMN template (100.0% of voxels in the DMN template were in the first component, 12.0% of significant voxels in the first component were in the DMN template), the second component overlapped with the FPN template (85.5% of voxels in the FPN template were in the second component, 13.7% of significant voxels in the second component were in the FPN template), and the third component overlapped with the SN template (100.0% of voxels in the SN template were in the third component, 8.3% of significant voxels in the third component were in the SN template). A similar method was used to ensure that the mean components did not differ between the low, intermediate, and high psychopathy groups (Supplemental Methods and Results, Figure S2).

When we performed RFX-BMS among low psychopathy participants age 18-55, we failed to replicate the SN switching effect (Figure S3). The SN modulation model did not show higher expected posterior model probability (0.262, 0.563, 0.175, respectively) or exceedance probability (0.202, 0.717, 0.081, respectively) than the DMN or FPN modulation model.

In the sample of participants younger than 30, we replicated the predicted SN switching effect in the low psychopathy group (Figure 3). The SN modulation model showed higher expected posterior model probability (0.383, 0.337, 0.280, respectively) and exceedance probability (0.415, 0.344, 0.241, respectively) than the DMN and FPN modulation models. The SN modulation model probabilities were similar to those reported in a prior study of healthy young adults (posterior model probability: sample 1 ≈ 0.440, sample 2 ≈ 0.520; exceedance probability: sample 1 = 0.514, sample 2 = 0.939) [23].

Next, and as predicted, posterior probabilities for the SN modulation model were significantly lower in the high psychopathy group compared to the low psychopathy group,

$t(145) = 26.39$, $p < .001$ (Figure 4A). These posterior probabilities did not differ between the intermediate and high psychopathy groups, $t(145) = 0.79$, $p = .43$. Posterior probabilities for the SN modulation model were also significantly lower in the intermediate compared to low psychopathy group, $t(99) = 27.98$, $p < .001$. Results were the same when covariates were included in the model.

Finally, analysis of modulation parameters revealed significantly reduced SN modulation of FPN-to-DMN connectivity in the high psychopathy group compared to the low psychopathy group, $t(145) = 2.28$, $p < .03$, but not the intermediate psychopathy group, $t(145) = 1.04$, $p = .30$ (Figure 4B). Post-hoc analysis revealed no significant difference between low and intermediate psychopathy groups for this modulation parameter, $t(99) = 1.27$, $p = .21$. There were no group differences for SN modulation of DMN-to-FPN connectivity: high compared to low psychopathy group, $t(145) = 0.27$, $p = .79$, high compared to intermediate psychopathy group, $t(145) = 0.06$, $p = .95$, and low compared to intermediate psychopathy group, $t(99) = 0.21$, $p = .83$. Results were the same with covariates included in the model.

For additional analyses of constituent clusters of psychopathic traits see Supplemental Methods and Results.

4. DISCUSSION

A growing body of research suggests that psychopathy may be related to altered connectivity between large-scale brain networks that support core functions. Following this line of research, we conducted novel analyses to test the hypothesis (first put forth by the Impaired Integration theory [31]) that the salience network's role in switching between states predominated by DMN activity and states predominated by FPN activity is impaired among individuals with psychopathy. First, we replicated the switching effect of SN during resting state in a subset of low psychopathy participants [22,23]. Second, as predicted, we observed that SN's switching role was significantly reduced in high psychopathy participants (as well as, unexpectedly, participants in the intermediate psychopathy group). As predicted, further analysis of modulation parameters revealed that SN modulation of FPN-to-DMN connectivity was uniquely impaired in the high psychopathy group. These findings may have implications for cognitive and affective deficits associated with psychopathy.

Among people with psychopathy, SN's role in switching between two networks (DMN and FPN) that are typically anticorrelated was impaired. This finding is best understood in light of ample research on healthy populations, which has identified a canonical pattern of activity in DMN and FPN during cognitively demanding tasks. FPN increases activity during such tasks and plays a critical role in the allocation of selective attention [22,23], while DMN tends to decrease activity [24]. Importantly, deviations from this canonical pattern are associated with impaired task performance. Healthy individuals make more errors (e.g., response inhibition errors in a stop signal task) when DMN remains active during a cognitively demanding task [46]. Similarly, healthy individuals display slower and more variable processing speed when DMN-FPN anticorrelation is reduced [47,48]. This canonical pattern of activity and connectivity appears to be altered in psychopathy. People with psychopathy have shown reduced deactivation of (medial) DMN regions during

cognitively demanding tasks [12,49,50] and reduced DMN-FPN anticorrelation [14,15]. Thus, competition between DMN and FPN could be related to cognitive abnormalities associated with psychopathy. We elaborate on this hypothesis in our discussion of the Impaired Integration theory below. The current study further suggests that SN switching may be a disrupted mechanism that contributes to competition between the two typically anticorrelated networks.

SN is a critical network for coordinating activity among other networks, possibly functioning at the top of a hierarchy of large-scale networks [51,52]. Among healthy individuals, two key SN nodes, the anterior insula and anterior cingulate cortex, appear to jointly coordinate the allocation of attentional resources through bottom-up attention switching and top-down biasing of sensory information [53]. Anterior insula receives input from multiple sensory modalities, suggesting the region likely plays the role of detecting salient sensory information [53]. Anterior insula also appears to send inhibitory signals to DMN and excitatory signals to FPN [22,51,54]. In contrast, anterior cingulate sends output to motor regions, suggesting the region likely plays the role of guiding action and maintaining control signals to other attention networks [53]. SN dysfunction in psychopathy has been documented in many studies. During cognitively demanding tasks, people with psychopathy have shown reduced activity when presented with salient sensory information in anterior insula [55–57] and anterior cingulate (based on meta-analyses [45]). Moreover, prior studies have found increased functional connectivity between SN and FPN nodes [13,15] and decreased connectivity between SN and DMN nodes [12]. However, connectivity within SN may be unaltered in psychopathy [17]. The extant evidence thus points to the following intriguing possibility: in psychopathy, SN's capacity to detect salient information may be diminished; consequently, SN might fail to exert inhibitory control of DMN and excitatory control of FPN, resulting in hyperactivity of medial DMN regions [12,49,50] and competition between DMN and FPN [14,15]. This hypothesis could have important implications for cognition and behavior, but requires further testing (for preliminary analyses, see Supplemental Methods and Results, Tables S2–3, Figures S4–8).

Impaired attention and associative learning have been frequently observed among people with psychopathy. Impaired SN switching may be a critical neural correlate of these cognitive deficits, as posited by the Impaired Integration theory [31]. Specifically, the theory predicts that impaired SN switching, along with other disruptions to communication between large-scale brain networks, results in inattention to perceptual features that are irrelevant to a current goal [31]. Indeed, ample research has associated psychopathy with deficits in orienting to salient, goal-irrelevant information (e.g., negative outcomes) and recruiting attentional resources during the performance of cognitively demanding tasks [31,35–39], two functions supported by SN [26]. It is possible that SN fails to orient to goal-irrelevant information and recruit attentional resources (e.g., FPN) during cognitively demanding tasks. Future studies are needed to test this hypothesis, as the current study examined SN switching during resting state, and our preliminary analysis of SN switching during a cognitively demanding task was likely underpowered (see Supplemental Methods and Results). Interestingly, prior studies have shown that people with psychopathy are capable of attending to goal-irrelevant information when it is presented prior to goal-relevant information [39,58]. This suggests SN may be capable of recruiting attentional

resources when psychopathic people are not already processing goal-relevant information. Additionally, the Impaired Integration theory posits that altered brain network interactions result in impaired binding of perceptual features into multimodal mental representations [31]. This hypothesis is supported by studies of perceptual processing [59,60] and associative learning [61–63]. However, another study failed to corroborate this hypothesis in an illusory visual paradigm [64]. In sum, diminished SN switching might contribute to impaired attention and associative learning, and further research is needed to test this hypothesis.

Impaired SN switching may also be related to psychopathic individuals' affective deficits. Psychopathic people are notoriously callous towards others and report shallow affective experience. In the broader emotion science literature, increasing evidence suggests that affect and emotion are not represented in specialized brain regions or circuits. Instead, large-scale networks including SN and DMN may interact to construct emotional experiences [65–67]. In addition to facilitating attention, SN plays a primary role processing signals from the body, which are thought to be essential components of affective experience [53,65,68,69]. Furthermore, DMN has been argued to map affect (i.e., pleasantness and arousal) onto discrete emotion categories such as fear, anger, and happiness [67]. Examining interactions among these large-scale networks while psychopathic people respond to emotionally evocative stimuli is a promising avenue for future research. In fact, one prior study has linked dysfunctional interactions between DMN and SN to psychopathic people's affective deficits. Decety and colleagues found reduced connectivity between anterior insula (of SN) and posterior cingulate cortex (of DMN) when psychopathic participants were taking the perspective of another person in pain [32].

To further characterize the nature of SN switching impairments in psychopathy, we analyzed the modulation parameters of the SN modulation model. Psychopathy was related specifically to SN modulation of FPN-to-DMN connectivity (but not DMN-to-FPN connectivity). However, the nature of the data make this finding difficult to interpret. In a typical DCM study, sensory input and task demands, along with knowledge of feedforward and feedback connections between regions, afford interpretations about the direction of information flow. A classic DCM study analyzed how attention and features of a visual stimulus affected connectivity between primary visual cortex (V1) and extrastriate visual cortex [70]. Given a wealth of literature on the flow of visual information through visual cortex, the study was able to conclude that attention modulates feedforward (V1-to-V5) but not feedback (V5-to-V1) connectivity. The current study analyzed communication between large-scale networks with varied anatomical connections (precluding interpretations about the *direction* of information flow between networks) during resting state (precluding interpretations about the *information* flowing between networks). Thus, SN modulation of FPN-to-DMN connectivity may be further examined in studies that employ a psychological task or that model connectivity between specific network nodes with known anatomical connections.

Dysfunction in these large-scale brain networks has been proposed to underlie a range of psychiatric disorders, not just psychopathy [71,72]. In fact, a diminished switching effect of SN on DMN-FPN connectivity has been observed in patients with schizophrenia [73]

and seniors with mild cognitive impairment [28]. Notably, diminished influence of SN on DMN activity has been observed in patients with behavioral variant frontotemporal dementia [27], whose behavioral profile resembles that of psychopathic individuals. Patients with this form of dementia undergo a “personality change” that often involves a lack of drive to engage in work and other personal obligations, irresponsiveness to the feelings of loved ones, and a disinclination for embarrassment (similar to irresponsibility, lack of empathy, and lack of remorse in psychopathy) [26]. However, psychiatric disorders may be distinguished by unique dysfunction within these core networks. For example, while mild cognitive impairment in advanced age has been uniquely negatively related to SN modulation of DMN-to-FPN connectivity [28], psychopathy was uniquely negatively related to SN modulation of FPN-to-DMN connectivity in the current study. Further inquiry is necessary to establish whether the observed abnormalities in large-scale brain network interactions are unique to psychopathy or shared among other disorders.

The current findings, though requiring replication, could potentially influence the development of treatments. Empirically validated treatments for psychopathy are lacking (although see [67]), perhaps in part because few treatments have targeted psychological or neurobiological mechanisms that are theoretically relevant to the disorder’s etiology [75]. Aberrant connectivity among large-scale brain networks could serve as a mechanism of change for treatments that use existing techniques. For example, real-time neurofeedback is a non-invasive, though resource-intensive, technique that can change momentary functional connectivity [76] and has yielded lasting treatment effects for other disorders such as ADHD [77]. Two studies of psychopathy have provided initial evidence that neurofeedback can alter SN function and change behavior [78,79]. Transcranial magnetic stimulation is another non-invasive technique for modulating cortical activity that has shown efficacy in treating other psychiatric disorders [80], but remains to be tested as an intervention for psychopathy. Alternatively, cognitive remediation targets cognitive processes such as attention, rather than neural activity, and has shown promising treatment efficacy for people with psychopathy [81]. Further study is needed to establish the efficacy of these techniques for treating psychopathy and to identify normalized interactions among large-scale brain networks as a key mechanism of change.

Several limitations of this study require consideration. First, the sample was limited to participants between 18 and 30 years old. Thus, the current findings may not generalize to older adults. We decided to limit the sample at a preliminary analysis stage in order to replicate prior studies that observed SN’s switching effect in a sample of participants age 18-30 [22], including a study by Goulden et al. [23]. However, Goulden et al. also observed evidence for SN’s switching effect in a second sample of participants older than 30. Another study also observed the effect in a sample of adults older than 30 [28]. To date, no other study has examined SN’s switching effect in incarcerated people of any age. More work is needed to understand large-scale network dynamics in incarcerated people older than 30. Furthermore, the current analyses of resting-state data did not address whether SN switching is diminished in response to cognitively demanding tasks. Our preliminary analysis of SN switching during such a task was likely underpowered (see Supplemental Methods and Results). The current study also fails to account for interactions between other networks (outside DMN, FPN, and SN), including those involved in executive function and attention

[82]. Dysfunctional interactions among these other networks, such as the dorsal attention network and visual network, could also be related to psychopathic individuals' attention deficits [31,83]. Lastly, the current study sought to replicate the switching effect of the salience network as a whole, but did not provide specificity about which SN regions drove the switching effect or which DMN or FPN regions were most causally affected. Analyzing the time series of specific nodes of interest rather than time series collapsed across each network, using Granger causality analyses or DCM, would help to address this issue.

5. CONCLUSIONS

In sum, mounting evidence has linked psychopathy to altered interactions between large-scale brain networks. This study provides novel evidence for a mechanistic explanation of these altered interactions, namely dysfunction of the salience network's switching role. Further characterizing these dynamic network interactions promises to advance neurobiological models of the disorder and influence the development of treatment.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Psychopathy is a significant risk factor for criminal behavior
- Prior evidence for altered interactions between large-scale brain networks
- Examined salience network's role in switching between two anticorrelated networks
- Switching role estimated via dynamic causal modeling
- Salience network's switching role was reduced for incarcerated men with psychopathy

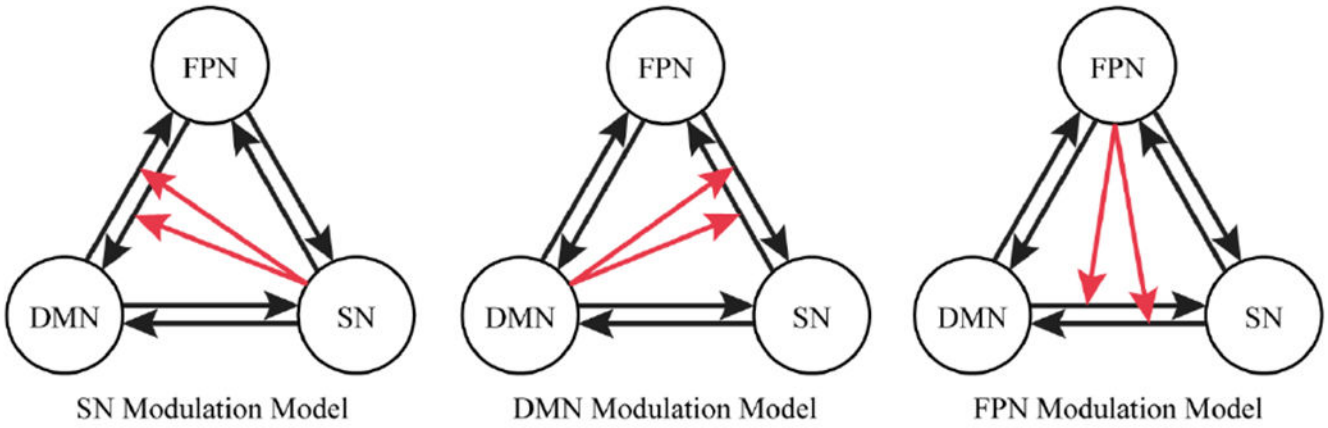

Figure 1.

Three fully connected DCM models specified for each participant and compared using random-effects Bayesian model selection. Sets of black arrows represent bidirectional connectivity between each network. Red arrows represent one network's modulatory effect on the connectivity between the other two networks. DMN = default mode network, FPN = frontoparietal network, SN = salience network

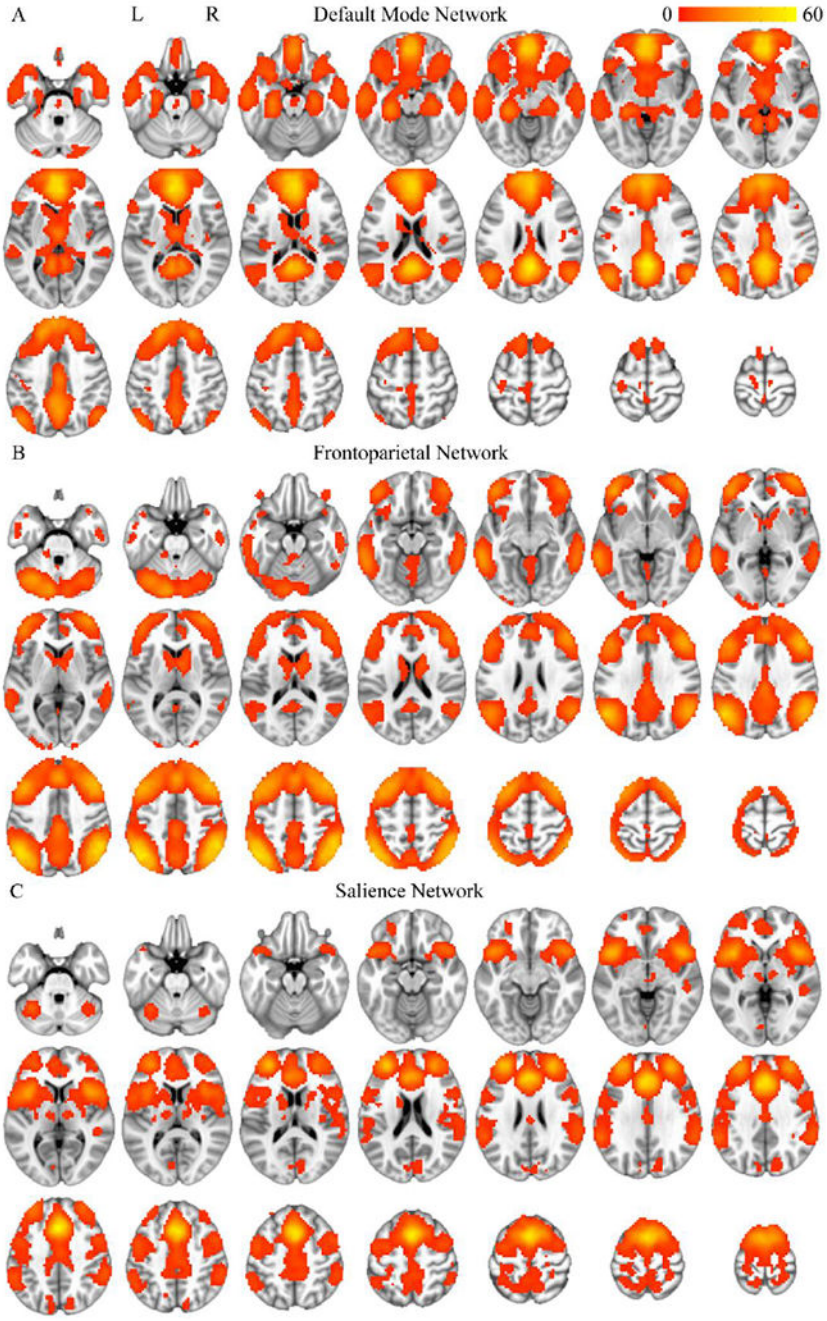

Figure 2.

Components derived from the constrained ICA, averaged across subjects. The first component corresponded to default mode network (DMN; A), the second component corresponded to frontoparietal network (FPN; B), and the third component corresponded to salience network (SN; C). The most inferior slice in each panel is z = −30, the most superior slice is z = 70, and each slice is separated by five mm. Voxels with significant (uncorrected $p < .001$, $p_{FWE} < .05$, cluster extent threshold = 27 voxels) *t*-values (displayed in the color bar) are shown.

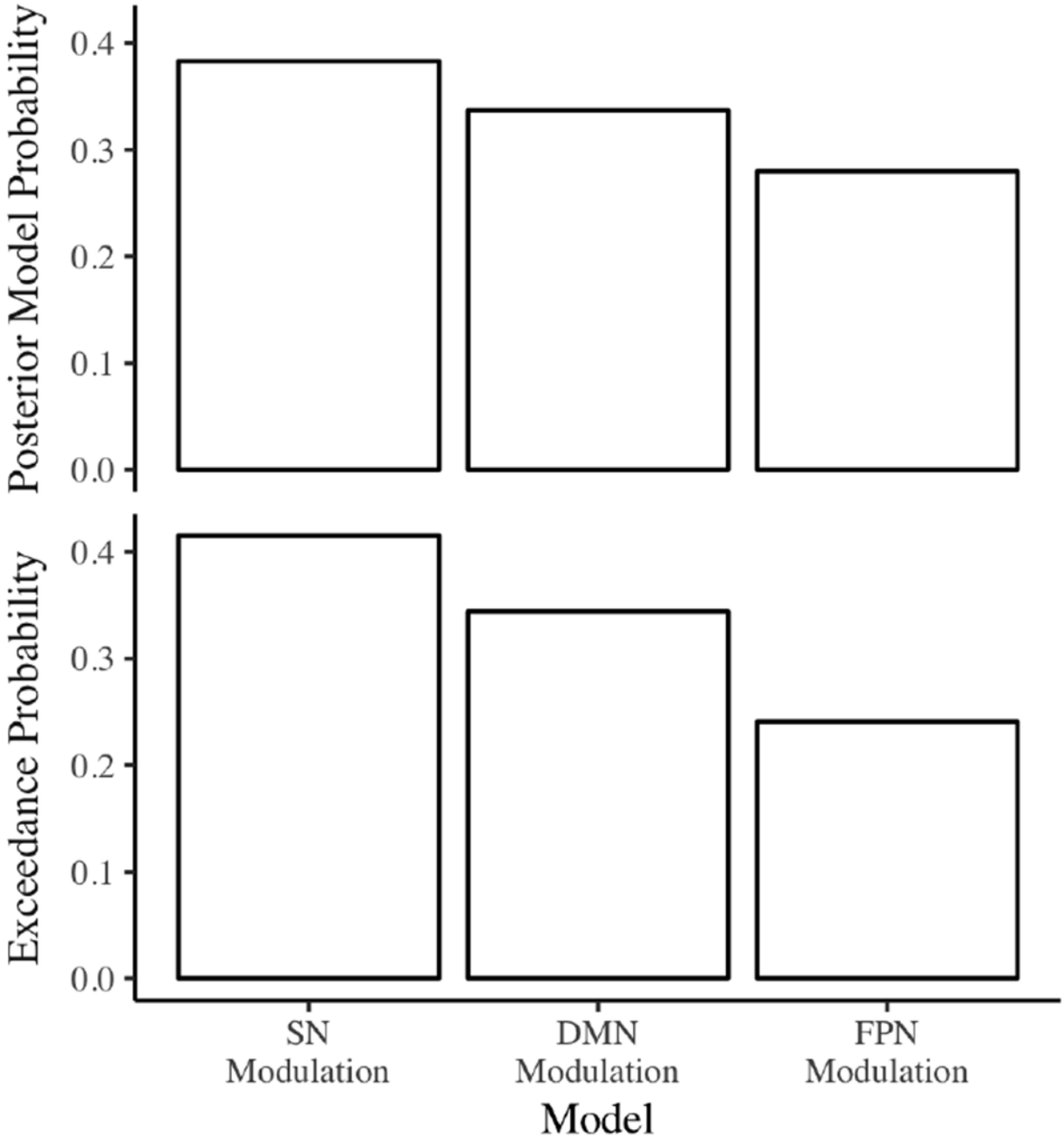

Figure 3.
Posterior model probabilities and exceedance probabilities for the low psychopathy group (PCL-R ≤ 20). DMN = default mode network, FPN = frontoparietal network, SN = salience network

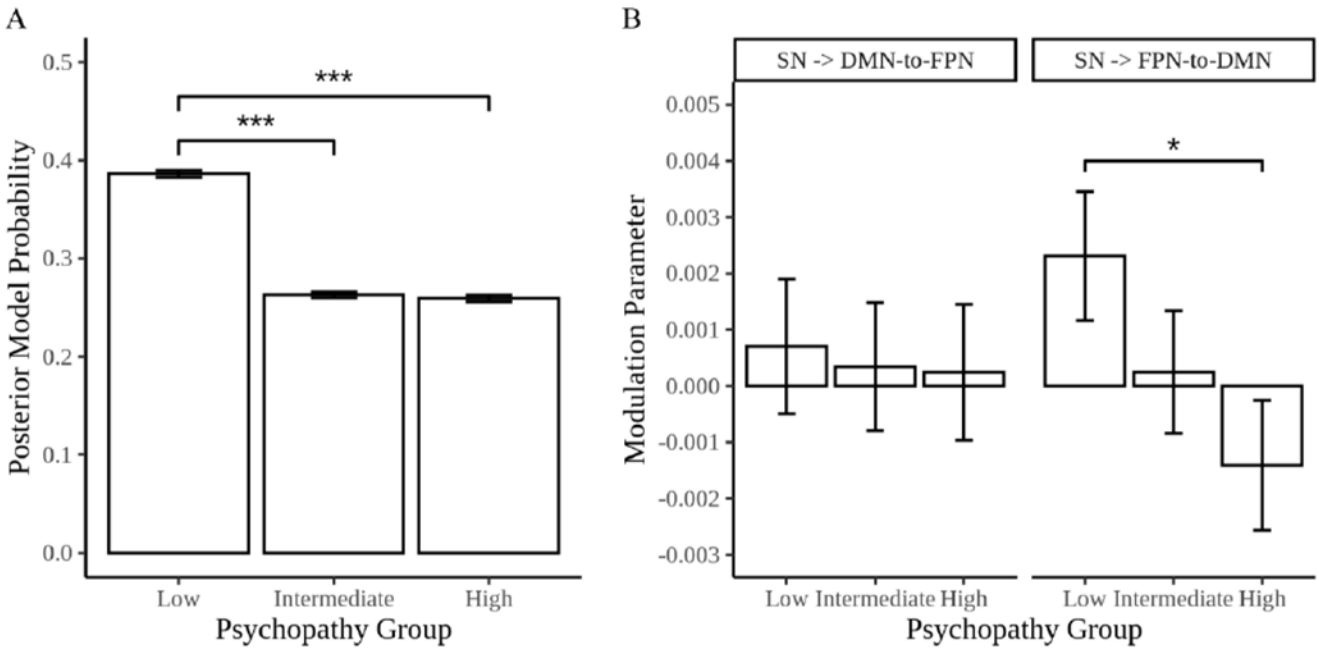

Figure 4.

A) Posterior model probabilities for the SN modulation model in the low (PCL-R ≤ 20), intermediate (PCL-R > 20 and < 30), and high (PCL-R ≥ 30) psychopathy groups. B) Estimates from the SN modulation model of the modulatory effect of SN on DMN-to-FPN connectivity (left) and FPN-to-DMN connectivity (right) in the low, intermediate, and high psychopathy groups. In both panels, error bars represent 1 standard error above and below the point estimate of the reference-coded model comparing the high psychopathy group to the low and intermediate psychopathy groups. DMN = default mode network, FPN = frontoparietal network, SN = salience network, * p < .05, *** p < .001

Table 1.

Sample characteristics (N = 148)

		Psychopathy Group		
Measure		**Low (*n* = 48)**	**Intermediate (*n* = 53)**	**High (*n* = 47)**
PCL-R Total	*M (SD)*	15.6 (3.3)	24.7 (2.3)	32.2 (2.2)
	Range	6.7-20.0	21.0-29.0	30.0-40.0
Age	*M (SD)*	25.0 (2.7)	25.9 (2.6)	25.9 (3.0)
	Range	19.4-30.0	20.9-30.0	19.4-29.9
IQ	*M (SD)*	96.8 (12.7)	96.5 (14.9)	98.1 (13.3)
	Range	74.0-123.0	70.9-126.0	72.0-134.0
Race/Ethnicity (White)	%	68.8%	54.7%	51.1%
Substance Use Disorder	%	81.2%	100.0%	93.6%
Head Injury	%	16.2%	32.7%	41.5%

Note: Psychopathy groups were formed according to recommended cut-offs (Hare, 2003): low (PCL-R ≤ 20), intermediate (PCL-R > 20 and < 30), and high (PCL-R ≥ 30).